

# Comprehensive Clinical Psychology

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Comprehensive Clinical Psychology

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*Editors-in-Chief: Alan S. Bellack and Michel Hersen*

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## Introduction to *Comprehensive Clinical Psychology*

**Co-Editors-in-Chief**

**Alan S. Bellack and Michel Hersen**

### Background

Clinical psychology is a relatively new field. While its roots can be traced back to at least the late nineteenth century, its evolution as a distinct academic discipline and profession dates only to the Second World War. The first

20 years of this postwar period saw steady, albeit nonspectacular, growth. Based substantially in the United States and Europe during this period, the study of clinical psychology developed as an alternative to medical school and psychiatry for many students interested in clinical service careers or the scientific study of human behavior. Postgraduate training was conducted exclusively in large university psychology departments within a strict scientist-practitioner model. The total number of Ph.D. candidates admitted to graduate school programs each year was relatively small; there were fewer than 50 accredited programs in the United States during much of this period, each admitting only 5-10 students. The number of new Ph.D.'s produced each year was substantially less, as many students failed to complete the rigorous scientific requirements of these elite programs. Career opportunities were similarly delimited, due in no small part to restraints on clinical practice imposed by psychiatrists and other physicians. The dominant form of psychotherapy was psychoanalysis, and psychologists were either excluded from psychoanalytic institutes or trained only as lay analysts who were proscribed from clinical practice. Few jurisdictions awarded licenses for independent practice, and psychologists generally were not reimbursed for their activities unless they worked under the direction of a physician. A sizable minority of clinical psychologists followed their mentors into university positions, teaching and conducting research. The majority, who were more interested in clinical service, opted for work in large psychiatric or Veterans Administration hospitals, where the modal activity was psychological testing; verbal psychotherapy was provided at the discretion of medical supervisors. A gradually increasing number of psychologists elected to be in private practice, where there was a greater professional autonomy. Medical hegemony over services for psychiatric disorders was even greater in Europe and Latin America.

The last 30 years has witnessed a massive change in the profession, stimulated by a number of scientific, clinical, and economic factors. Psychoanalysis gradually fell out of favor due to a dearth of data on its effectiveness and a desire for shorter term treatments that were not the primary purview of psychiatrists. First, client-centered therapy and then behavior therapy emerged as brief, highly effective alternatives. The former was entirely a product of clinical psychology, and was the intellectual and technical forebear of the current mandate for empirical evaluation of psychotherapies.

Carl Rogers, his colleagues, and students were the first to demonstrate the feasibility of careful, objective evaluation of the therapy process as well as outcome. While behavior therapy owes much of its legacy to psychiatrists such as Joseph Wolpe, it was substantially a product of academic psychologists searching for an approach with a strong scientific underpinning (in this case learning theory) that could be subjected to rigorous scientific scrutiny. Early behavior therapy emerged simultaneously in the UK and the US: in the UK psychologists such as Hans Eysenck based their work on Pavlov and classical conditioning, while in the US researchers were following Skinner and operant conditioning theories. The two schools merged with cognitive therapy, developed largely by Beck and Ellis through the 1960s, when the limitations of behavior therapy in isolation became apparent, particularly with depressed patients, and cognitive-behavior therapy is now widely practised.

Behavior therapy and cognitive-behavior therapy have not only proven themselves to be effective with a broad array of disorders, they have since been shown to be very successful alternatives to pharmacotherapy as well. Notably, behavior therapy was able to produce significant changes in populations that had previously been warehoused as untreatable, including people with physical and developmental disabilities and schizophrenia. Many of the most important contributions to the behavior therapies came from the UK, The Netherlands, South Africa, Australia, and Scandinavia, providing a tremendous stimulus for the development of clinical psychology globally. The availability of cost-effective, scientifically sound nonmedical treatments has decreased the medical monopoly of psychiatric/mental health services around the world and fostered the evolution of clinical psychology as a legally sanctioned helping profession, as well as a prestigious scientific discipline.

Scientific advances in our understanding of the brain and the role of psychosocial factors in physical health and illness have led to the development of two other rapidly growing subspecialties of clinical psychology: neuropsychology and health psychology. Novel assessment and treatment technologies in these two areas have created professional opportunities for clinical psychologists in medical schools, general medical hospitals, and other nonpsychiatric settings. Clinical psychologists can now be found conducting research and providing services in departments of neurology and neurosurgery, medicine, cardiac surgery, pediatrics, anesthesiology, oncology, and other medical specialty areas, as well as in the traditional psychiatric settings. They increasingly serve as directors of governmental agencies and service facilities. They comprise a large percentage of research grant recipients in the US, Canada, and the UK, and sit on prestigious government and foundation review boards. In fact the field has earned sufficient public recognition that it now has the somewhat dubious distinction of having clinical psychologists as lead characters on television shows and in cinema.

Stimulated, in part, by these exciting developments in scientific progress and clinical creativity, the field has grown geometrically in the past two decades. Psychology is now the second leading undergraduate major in the US and is increasingly popular elsewhere in the world as well. There are now more than 175 doctoral programs in the United States, each admitting many more students per annum than the 5-10 that has been typical of traditional scientist-practitioner Ph.D. programs over the past 25 years. Some of these schools have entering classes as large as 200 per year. Moreover, along with the professional school movement, which began in the 1970s, a new degree, the Psy.D. (or Doctorate in Psychology), is regularly being offered as an alternative to the Ph.D. Basically a professional

rather than an academic degree, the Psy.D. is reflective of the local practitioner-scientist model rather than the scientist-practitioner.

Yet another trend in the field is the proliferation of master's level psychologists, specifically trained to carry out some of the more mundane functions formerly implemented by doctoral level psychologists. Indeed, each year in the United States alone 10 000 new master's level psychologists graduate from university programs. The financial and programmatic implication of such large numbers is obvious.

Statistics are not readily available about the size of the profession in all regions of the globe, but anecdotal evidence supports the hypothesis that the field is growing worldwide. As previously indicated, behavioral and cognitive-behavioral therapies owe a substantial debt to scientists and clinicians from Europe, Australia, and South Africa. There are now enough cognitive-behavior therapists to support national societies in most Western European countries, as well as Asia, Australia, and Latin America. Many of the most important developments in the psychosocial treatment of severe and persistent mental illness in the last decade have come from the United Kingdom, Australia, Switzerland, and Germany. Psychologists in Scandinavia, the United Kingdom, and the Netherlands have played a central role in the development of cognitive-behavioral treatments for anxiety and depression, and there have also been notable contributions from these regions to health psychology. As the hold of psychoanalytic therapies on psychiatric treatment in Europe continues its inevitable decline, there will be increasing opportunities for clinical psychologists to provide shorter term behavioral and cognitive-behavioral treatments. In addition, exciting developments are also emerging from Japan, China, and other countries in the Pacific rim. It seems likely that the global influence of regional approaches and thinking will lead to a more multicultural and universal psychology than has been the case in the past.

The scientific and clinical literatures have burgeoned along with the number of clinical psychologists in the world. This has been an era of rapid growth of knowledge and increasing specialization. General topics, such as psychological assessment, clinical child psychology, and psychotherapy, that used to merit only one or two graduate courses to establish expertise, have expanded and are subdivided to the extent that circumscribed specialty areas, such as neuropsychology, geropsychology, behavioral pediatrics, or cognitive-behavior therapy for depression can each require postdoctoral training. Consequently, hundreds of undergraduate, graduate, and professional level texts are published each year. Specialty journals abound. Where a few key generalist journals such as the *Journal of Consulting and Clinical Psychology* used to represent the entire field, each subdiscipline now has multiple journals, and there are both national journals (e.g., the *British Journal of Clinical Psychology*, the *British Journal of Health Psychology*, the *Australian Journal of Cognitive and Behavioral Therapy*) and journals representing specific populations or disorders (e.g., *Addictive Behaviors*, *Journal of Family Violence*, *Journal of Clinical Geropsychology*), or domains of practice (e.g., *Journal of Clinical Psychology in Medical Setting*). Specialization has made it difficult for professionals to keep abreast of developments within their immediate areas of expertise, and impossible for them to be conversant with the literature in other areas. Moreover, given the plethora of choices, it is also virtually impossible for either students or professionals to know where to find the most accurate, up-to-date information in most areas.

The combination of a large and increasing number of students and professionals, and rapidly growing scientific and clinical literature, makes this a particularly appropriate time for *Comprehensive Clinical Psychology*. This multivolume work encompasses the entire field, and represents a single source of information on the scientific status of clinical psychology and its subspecialties, on theory, and on clinical techniques. The work covers the history of the field, and current thinking about training, professional standards and practices, and sociocultural factors in mental health and illness.

### ***Genesis of Comprehensive Clinical Psychology***

Following preliminary conceptual discussions between Elsevier Science and Alan S. Bellack at several international conferences in 1994, Michel Hersen was asked to join as Co-Editor-in-Chief. The first official planning meeting for the project took place in June 1995. In addition to Elsevier Science staff, Alan S. Bellack and Michel Hersen invited Tom Ollendick, Nina Schooler, and Warren Tryon to serve as consultants. At that meeting, the philosophical and international scope of the project was agreed upon and established, with the scientific underpinnings of the field identified as the model. The objective here was to ensure that chapters reflect our core knowledge and that the material stand the test of time.

At that meeting, we also underscored that since clinical psychology was now an international discipline, the work should reflect contributions at the cross-cultural level, with chapters solicited from eminent psychologists worldwide. Although it was acknowledged that the United States was in the forefront of the field, the work could not simply represent the American perspective but to the extent possible would represent diversity at its best. Consistent with the international perspective, at the initial planning meeting, the importance of having an Honorary International Editorial Advisory Board comprised of international representatives was acknowledged, and the 10 specific volumes to comprise *Comprehensive Clinical Psychology* were identified. Preliminary outlines for each volume were developed and volumes editors were considered.

The international perspective was to be reflected at a tripartite level. First, diversity among editors and contributors for their respective volumes was selected as a goal. Second, chapters in each volume were designed to reflect diversity by providing the reader with worldwide examples, not simply the Anglo-Saxon view. Of course,

where basic facts and principles were the same, there was no need to present regional diversity. Third, and related to the first two parts, the Honorary International Editorial Advisory Board provided us with an international perspective on overall organization and specifics for the individual volumes.

Between June and October 1995, Alan S. Bellack and Michel Hersen, in consultation with Elsevier Science, invited the ten volume editors to assume their positions, and a meeting of the Editors-in-Chief, the ten volume editors (C. Eugene Walker, Arthur N. Wiens, Nina R. Schooler, Cecil R. Reynolds, Thomas Ollendick, Paul Salkovskis, Barry Edelstein, Marie Johnston and Derek W. Johnston, Nirbhay N. Singh, and Cynthia D. Belar), and Elsevier Science staff was convened in October of that year. At that meeting, each of the volume editors presented his or her conception of the relevant volume, and the nature of coverage and particular contributors was discussed at length. Most of all the philosophical underpinnings of the work were stressed so as to insure intervolum consistency.

Subsequent to the October 1995 meeting, the enormous work to bring this project to fruition began, with potential authors invited to contribute, manuscripts reviewed, and then edited. Were it not for the wonders of electronic communication, a project of this scope would not have been possible, especially given the international aspects involved. A lengthy series of checks and balances was instituted to guarantee the quality and excellence of each contribution. The volume editor first approved each contributor's chapter outline, followed by editing and approval of the text. This process frequently required several revisions. The Co-Editor-in-Chief then reviewed each chapter for scope, level, and overlap, but only after the volume editor had first verified the accuracy of references cited. After the Co-Editor-in-Chief's labors, the manuscript was reviewed by Elsevier staff for format, writing style, reference checking, and other technical issues.

### ***Aims and Scope***

The final organization and contents of the work evolved over a series of discussions between the Editors-in-Chief, the volume editors, and Elsevier Science. It was comparatively easy to select the primary domains that needed to be covered: history, treatment, assessment, research, training, and professional issues. It was also comparatively easy to identify the first two-thirds, or so, of specific topics that required chapter-length coverage: treatment of the primary DSM/ICD disorders, basic research strategies, standard assessment techniques, etc. However, organizing the vast set of requisite topics into coherent volumes, determining which topics warranted independent chapters, and assigning page limits to individual chapters proved to be daunting. Two broad organizational themes immediately suggested themselves: a focus on core themes or techniques across populations vs. integrated coverage of populations. For example, the former would have entailed volumes on treatment modalities, such as behavior therapy, as they are applied to children and adults, while the latter would call for separate volumes on children and adults that covered diverse approaches. To complicate matters, some topics, such as Research Methods and Professional Issues, do not lend themselves to breakdown by population, and others, such as Behavioral Medicine, do not lend themselves to a breakdown by themes or techniques. Volume length was also an important factor, making some content-based solutions less practical than others. For example, we determined that treatment should receive more attention than assessment; a strict population-based solution would have led to separate short volumes on assessment of adults and children. Ultimately, we opted for an organizational structure that balanced practical considerations with our collective prediction about how the individual volumes would be used. While it was different earlier in the development of the field, we believe that the current trend is for people to be more organized around populations than techniques. Hence, more people are likely to pick up and cross-reference a single volume on children or the elderly than a volume on Behavior Therapy. Our strategy for identifying chapter length topics and associated page limits is more difficult to explain. Once again, we relied on our collective judgement, honed by negotiation. In rough order, priority was given to topics that had established empirical literatures, that were deemed to be "important," that had broad interest, and that were likely to be at least as important in the next decade. Page limits were determined substantially by estimates of the first two criteria. We began with an overall target for the entire work and minimums and maximums for volumes, and then worked backwards to divide up the allotted pages among the chapters designated for each volume. Given that no scheme will please everyone, we are confident that the organization of the work adequately reflects the field now and in the foreseeable future.

Under the careful aegis of the outstanding group of experts comprising the Honorary International Editorial Advisory Board, 10 leading international scholars were selected to edit the 10 specific volumes.

*Volume 1 (Foundations)*, edited by C. Eugene Walker, provides a complete overview of the basic foundations of clinical psychology, with special emphasis on the relationship between clinical psychology and other fields of science. Beginning with a brief history of clinical psychology, as well as a look at its current scientific status, this informative volume covers such topics as the biological bases of clinical psychology, elucidating research in genetics, psychobiology, psychopharmacology, and the use of animal models in human mental health problems; clinical psychology in the behavioral sciences, including anthropology, epidemiology, sociology, and research psychology; and the major systems and theories that are used in clinical psychology. The volume also describes various techniques for library research and information retrieval in psychology.

*Volume 2 (Professional Issues)*, edited by Arthur W. Wiens, focuses on the professional, legal, and ethical issues that are relevant to clinical psychology. The volume addresses the various educational and training programs

available, such as doctoral study, internship training, and postdoctoral residency programs, and reviews the accreditation of these programs. Also highlighted are the various international government guidelines for registration, certification, and licensing, including a discussion of the advantages of specialty recognition and practice certificates. The volume concludes with a look at ethical and legal guidelines in the management of clinical psychology practices, national healthcare policies, and advocacy efforts for government support for practitioners.

*Volume 3 (Research and Methods)*, edited by Nina R. Schooler, explores the function of research in clinical psychology. The volume begins with an in-depth look at research approaches, including the use of descriptive studies, single case designs, observational methods, and other methods of analysis. The volume goes on to explore a broad range of topics that have been the focus of research, such as test development and validation, personality assessment, clinical interventions, and service evaluations and outcomes. Finally, various statistical techniques are reviewed, including descriptive and inferential statistics, factor analysis, and sampling and generalizability.

*Volume 4 (Assessment)*, edited by Cecil R. Reynolds, provides valuable information on the development and role of assessment in clinical practice, analyzing such topics as psychometrics; taxonomic, functional, and actuarial approaches to diagnosis; and specific instruments, techniques, and procedures. Chapters also review the range of assessment techniques and procedures used in clinical practice, with emphasis on intelligence, neuropsychological, personality, projective, computer-assisted, therapeutic, and forensic assessment. The volume concludes with a review of legal guidelines and regulations in the use of psychological testing.

*Volume 5 (Children & Adolescents: Clinical Formulation & Treatment)*, edited by Thomas Ollendick, draws on the experience and research of leading scientists and clinicians from Australia, Canada, Israel, the United Kingdom, and the United States to present state-of-the-art information on all aspects of child psychology and psychiatry, with special attention given to the psychopathology, assessment, treatment, and prevention of childhood behavioral disorders. The volume highlights the developmental-contextual framework used in the clinical formulation of these disorders, as well as process and outcome issues in treatment. Various theoretical perspectives are also reviewed, including applied behavior analysis, family systems therapy, play therapy, and pharmacologic therapy. In the final section, all of the major childhood disorders found in the DSM and ICD are described, with information on their prevalence, etiology, assessment, and treatment. This section also analyzes the empirical status of the various therapies used for treatment of childhood disorders.

*Volume 6 (Adults: Clinical Formulation & Treatment)*, edited by Paul Salkovskis, provides valuable insights into the basis of the psychological theories and interventions used for behavioral and emotional problems and reviews how to integrate clinical skills with these theories. Various treatment approaches are addressed, such as cognitive therapy, family therapy, and Humanistic/Rogerian/Gestalt approaches, as well as the issues related to treatments, including stress management, arousal reduction methods, suicidal behavior, and specific issues in working with groups. The final section details specific problem areas and disorders, ranging from such universally recognized problems as gambling and substance abuse to more specific disorders such as post-traumatic stress, depression, obsessive-compulsive, and the various phobias. Each chapter in the volume emphasizes approaches that have an empirical basis.

*Volume 7 (Clinical Geropsychology)*, edited by Barry Edelstein, addresses the emerging field of clinical psychology in the aging population. The volume begins with a review of this area of research, presenting important epidemiological information. The volume then offers a detailed look at issues that range from analyzing physiological and cognitive aspects to cognitive changes and specific neurological disorders common among older adults. Specific topics covered include sexuality, bereavement, anxiety, substance abuse, and schizophrenia. Each chapter presents a summary of clinical research and its practical application. Voids in the knowledge base are also noted, along with recommendations for the direction of future investigations. The volume also addresses management problems, such as incontinence, wandering, and aggressive behavior, and reviews the various mental healthcare systems available in different countries.

*Volume 8 (Health Psychology)*, edited by Derek W. Johnston and Marie Johnston, provides a comprehensive overview of the development and application of clinical health psychology. Beginning with a discussion of training, assessment, and measurement issues, this volume analyzes the key behaviors that either affect or are related to health. Topics covered include stress and disease, the experience of illness, and behavior that can affect the neuroendocrine, cardiovascular, and immune systems. The volume also provides a detailed analysis of specific clinical problems and their psychological aspects and interventions. These include cancer, diabetes, epilepsy, disfigurement, and smoking.

*Volume 9 (Applications in Diverse Populations)*, edited by Nirbhay N. Singh, covers the broad spectrum of diverse issues that clinical psychologists typically face in their work. Four sections outline the various psychological aspects found in different populations, as well as methods for assessment, diagnostic information, and interventions useful with these different groups. Section I focuses on select child, adolescent, and adult populations, including those with developmental disorders, learning disabilities, and mental retardation. Section II is devoted to various types of families and their issues, including families of individuals with HIV or AIDS, families of alcoholics, and families of children with serious emotional disturbances. Section III covers victims of violence and abuse, including child sexual abuse. Section IV examines perpetrators of violence and abuse, including sex offenders and issues of domestic violence.

*Volume 10 (Sociocultural and Individual Differences)*, edited by Cynthia D. Belar, covers cross-cultural psychopathology and interventions. Chapters examine such select topics as gender, sexual orientation, socioeconomic status, religions, and training for clinical psychologists. The volume also provides valuable insights into the use of clinical psychology in different parts of the world, as well as personality assessment across international settings.

Given the scope and detail of *Comprehensive Clinical Psychology*, Volume 11 is devoted to: (i) a Name Index, (ii) a Subject Index, (iii) a List of Contributors, and (iv) a list of the Contents of All Volumes. The Name Index is an accumulation of all the authors who are cited in text in the reference sections throughout the entire work. The Subject Index, consisting of more than 40 000 entries, is a consolidation of all the individual volume subject indexes. It is presented in word-by-word alphabetical sequence with a maximum of three levels of heading. Terminology in the index is based on standard internationally recognized sources. Cross-references are provided to assist the user to locate preferred terms and terms of related interest.

### **Acknowledgments**

To produce a tome of this magnitude requires an enormous number of individuals with unique talents working in concert. To begin with, we applaud the herculean efforts of our driving force and friend at Elsevier Science, Barbara Barrett. We also gratefully acknowledge the efforts of two other publishing editors at Elsevier Science, Susan Hanscom and David Hoole, who provided guidance and encouragement along the way. We are particularly thankful for the exceptionally hard work of Angela Greenwell and her staff in Oxford, who made sure that all tasks were implemented reasonably on time and who orchestrated the day-to-day management of this huge undertaking. Next, we thank our eminent volume editors, who had the difficult job of soliciting, tracking, and editing manuscripts for their respective volumes. Similarly, we thank the Honorary International Editorial Advisory Board for their excellent input in developing the outline for the work and suggestions as to potential international contributors. Of course, we owe a great deal to the individual contributors who agreed to share their expertise with us in a timely fashion. Finally, we are most appreciative of our own editorial assistants, Sonia McQuarters and Burt G. Bolton, who repeatedly have provided us with the kind of support that makes all of this a possibility.

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# Subject Index

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Every effort has been made to index as comprehensively as possible, and to standardize the terms used in the index in line with the following standards:

- *Thesaurus of Psychological Index Terms*, APA, Eighth Edition, for the selection of psychological terms.
- *Thesaurus of ERIC Descriptors*, ERIC, Twelfth Edition, for the selection of education terms not covered by the above.

- EMTREE Thesaurus for the selection of medical terms not covered by the above.
- IUPAC Recommendations for the nomenclature of chemical terms, with trivial names being employed where normal usage dictates.

In general, the index follows the recommendations laid down in BS ISO 999:1996.

In view of the diverse nature of the terminology employed by the different authors, the reader is advised to search for related entries under the appropriate headings.

The index entries are presented in word-by-word alphabetical sequence. Chemical terms are filed under substituent prefixes, where appropriate, rather than under the parent compound name; this is in line with the presentation given in the *Thesaurus of Psychological Index Terms*.

The index is arranged in set-out style, with a maximum of three levels of heading. Location references refer to volume number (in bold) and section number (separated by a colon).

*See* cross-references direct the user to the preferred term; for example, character *see* personality.

*See also* cross-references provide the user with guideposts to terms of related interest, from the broader term to the narrower term, and appear at the end of the main heading to which they refer; for example  
credentialing

*see also* professional certification; professional licensing; recredentialing

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# Comprehensive Clinical Psychology. Volume 1

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*Editors-in-Chief: Alan S. Bellack and Michel Hersen*

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## Preface Volume 1

Psychology is the science of human behavior. As scientists, psychologists attempt to understand, predict, and control human behavior. Clinical psychology is the specialty area of psychology that deals with emotional disturbance and mental health. The field of clinical psychology draws on the basic research data from psychology and several related areas of science in order to promote health and effective coping with life events.

Clinical psychology is the most popular area of specialization within psychology. In 1995 nearly 44% of the members of the American Psychological Association identified their major area of interest as clinical or child clinical psychology. An additional 10% identified their major interest as the closely related area, counseling psychology. This strong interest in clinical psychology no doubt stems from the fact that training in this area provides one with a broad and generic background that may lead to a wide variety of career options.

The majority of clinical psychologists find employment providing mental health care. This involves diagnostic assessment, psychotherapy, and related tasks in a wide variety of settings including private practice, outpatient clinics, hospitals, schools, and industry, to name a few. However, clinical psychologists may be found in a wide selection of other career options.

A significant number of clinical psychologists devote virtually all of their time to research. These clinical psychologists may work in a research center or in a university. Their research may be funded from federal government agencies, private foundations, or industry.

Many clinical psychologists are employed as teachers. They serve as faculty in major research universities as well as in teaching universities and medical schools. They teach basic psychology courses as well as service courses for other departments and professional courses for students preparing for a career in clinical psychology. Clinical psychologists are generally employed in departments of psychology, however, they are often found in other departments such as business, education, and biology, to name but a few.

Because of their skills in understanding and working with people, clinical psychologists are often selected for positions of management and leadership in many organizations. This is true whether or not the organization's mission is related to psychology *per se*. Thus, many clinical psychologists are administrators, consultants, liaisons, representatives, and program developers in a wide variety of settings.

Since training in clinical psychology provides the necessary background for making contributions in so many ways, most clinical psychologists combine more than one role as they pursue their career. For example, a typical clinical psychologist might have his or her primary employment providing mental health care but might also be involved in research and be a member of the adjunct faculty of a nearby university. Likewise, a clinical psychologist whose primary employment is in an academic setting, involving teaching and research, might also have a small private practice providing mental health care and might serve as a consultant to various other agencies and organizations.

By virtue of their training, clinical psychologists are able to make several unique contributions in the field of mental health that other professionals in this area are unable to provide. First, is their scientific approach and research skills. Traditionally, clinical psychologists have been trained in academic departments of psychology and have received a Ph.D. degree, which is the standard degree for scientists and scholars. They have also generally been trained in what is known as the "scientist-professional" model. This model developed very early in the history of the field and is based on the concept that a clinical psychologist should be a well-trained scientist who applies scientific knowledge as a professional. As a result, clinical psychologists are given extensive training in the philosophy of science, scientific method, statistics, and research design. Even though recent years have seen the development of free-standing professional schools of clinical psychology and professional degrees such as the Psy.D. (doctor of psychology), most clinical psychology training programs, including the programs in professional schools and the Psy.D. programs, continue the tradition of scientific orientation. In most clinical settings, clinical psychologists are distinguished by their scientific skepticism about clinical practices which are not based on research data and by their interest in conducting careful clinical research on issues related to their professional practice.

A second unique contribution of the clinical psychologist is expertise in psychological testing and assessment. The interest of psychologists in research naturally fostered the development of procedures for measuring human behavior since quantification is a basic necessity for scientific investigation. When psychologists began to apply their knowledge in the clinical arena, their initial

point of entry was their ability to provide reliable and valid information about patients based on psychological tests. Appropriate use and interpretation of psychological testing results requires sophistication in measurement theory and statistical prediction. Clinical psychologists received this as part of their academic and research training.

A third unique contribution of the clinical psychologist has to do with the extensive body of research into human behavior, personality, and the process of behavior change which has been developed in psychological research laboratories for over 100 years. The clinical psychologist's familiarity with this literature enables him or her to approach clinical problems with a high level of sophistication and with a wide range of options for effective intervention.

The heavy emphasis on a scientific basis for clinical practice is appropriately the theme of Volume 1 in this series. In this volume we will survey the foundations of clinical psychology including basic data from psychological science, biological science, social and behavioral science, and related areas.

The problem of emotional disturbance and mental illness has been with humans from the beginning. In the first chapter of this volume, Professor Donald Routh, of the University of Miami in the United States, traces the history of mental problems and the professions that have tried to alleviate these problems. He also presents a brief history of research in psychopathology, citing major advances from the earliest times to the present. This excellent chapter provides a broad perspective for understanding all of the material presented in this volume as well as all of the other volumes in this series.

Dr. Frank Collins of the Oklahoma State University in the United States has prepared an excellent summary of the scientific status of clinical psychology. He begins with a brief introduction and history of clinical psychology in America since the major events leading to the recent developments in the field occurred on this continent. He then discusses the basis of clinical psychology in science and contrasts this with other approaches such as philosophy and art. The current scientific status of clinical psychology in the major areas of assessment and treatment is reviewed and summarized. This nicely leads to a discussion of training in clinical psychology and the role of clinical psychology in health care. Dr. Collins concludes his chapter by emphasizing the scientific basis of clinical psychology. He foresees much more growth in the future, but believes that the current foundation provides excellent support for such growth and development.

In any field of scholarship and inquiry, accessing information efficiently is a matter of considerable importance. Since psychology as a field, has been concerned with human behavior including learning, cognition, communication, and related areas, psychologists have naturally been in the forefront of information processing and access. Dr. Kristi Alexander of the United States International University and Dr. Michael Roberts of the University of Kansas in the United States have prepared a comprehensive guide for the student, professional, or interested layperson who wishes to locate information available on the wide range of topics included in clinical psychology. Drs. Alexander and Roberts discuss access to journals and books including on-line journals which are newly appearing through Internet services. They then describe the major abstracting and indexing resources available, including those that are computerized. Their chapter concludes with an excellent consumer's guide to electronic media and the Internet for those interested in pursuing topics relevant

to clinical psychology. Computer and Internet accesses to psychological literature are a particularly exciting development because they make the same information instantaneously available to students and professionals as well as to the educated layperson anywhere in the world.

Human behavior results from the interaction between heredity and environment. Over the years, students of human behavior have shifted back and forth in ascribing more weight to one side or the other of this interactive equation. Recently, careful research has greatly added to our understanding of the role of genetics in many areas of personality and psychopathology. Drs. Thomas G. O'Connor and Kirby Deater-Deckard of the Institute of Psychiatry at the University of London and Robert Plomin of PaSU, Philadelphia, United States, in their chapter on Contributions of Behavioral Genetics Research to Clinical Psychology, provide the reader with an excellent survey of work in this important field. They skillfully blend information about the history of the area with explanations of key research strategies and methods as they summarize the current state of knowledge in major areas of interest to clinical psychologists such as anxiety, depression, alcoholism, schizophrenia, attention deficit disorder as well as several others. Much work needs yet to be done to further our understanding of the relative contributions of genetics and environment. Thus, the debate continues. O'Connor and his co-authors have provided us with an excellent summary of the current status in this area and with the tools to evaluate new developments as they occur.

The mind-body problem has been pondered by scientists and scholars for centuries. While the average person tends to regard these as separate realms, most scientists reject this notion in favor of a monistic view. Dr. Niels Birbaumer, University of Tübingen, Germany, and his colleague Professor Herta Flor, Humboldt-University, Berlin, reject separation of mind and body. Instead, they find it more useful to describe human behavior in terms of three levels: verbal reports, physiological responses, and overt or motor responses. Their chapter deals with psychobiology or biological psychology. These two synonymous terms likewise emphasize the unity rather than separation of mind and body. Much research in this field has contributed to our understanding of psychopathology and therapeutic intervention. Birbaumer and Flor present necessary background scientific information and then demonstrate the usefulness of this research information for understanding human behavior in numerous areas of concern to the clinical psychologist. The importance of understanding human behavior in totality including all three levels, as described in this chapter, cannot be overestimated. Clear thinking regarding assessment and effective intervention for human problems requires a complete view of the nature of humans.

One of the most dramatic developments in human society in the last 50-100 years has been the major effect that drugs play in our lives. Certainly, abuse of numerous substances by large numbers of people throughout the world has resulted in harm and misery. On the other hand, the rapid growth of scientific research in pharmacology has resulted in thousands of drugs that ameliorate illness and promote human welfare. Dr. Frank Holloway of the University of Oklahoma Health Sciences Center and Dr. Jessica Peirce of Brookline, Massachusetts, systematically review the major categories of psychotropic drugs which are of considerable importance to the practice of clinical psychology. They present basic scientific data and use that to discuss therapeutic benefits as well as undesirable side effects from drugs currently available. Since there is a huge literature on the basic chemistry of these drugs and an equally large literature on their clinical application, Holloway and Peirce can only survey this information in their present chapter. However, they cite numerous references for the

reader who wishes to pursue these topics in more detail. The material contained in this chapter is very timely given the movement among clinical psychologists to obtain legal authority to write prescriptions for medication for their patients. There is an increasing appreciation for the idea that effective care for patients requires intervention on all three levels described in the Birbaumer and Flor chapter. Thus, selective use of psychotropic drugs is an important part of providing total care.

The use of animals for basic research in the field of medicine is well known. There are numerous advantages to this. Often, a less complex organism than a human being can be employed, making experimentation easier. More rigorous experimental designs can be used than are possible with humans. Results can often be obtained immediately and more clearly. Possible harm to humans is avoided. What is not as well known is that animals are also often very useful in behavioral research that ultimately may be generalized to humans. Over the years, some of the most creative ground-breaking research on psychopathology has been based originally on animal experiments. Professor Paul Willner of the University of Wales in the United Kingdom discusses the rationale for animal models of psychopathology. He then describes research methods employing this approach and reviews the major findings in the areas of depression, anxiety, schizophrenia, and substance abuse. Professor Willner has done an excellent job of presenting the information in this area in a very readable and exciting way.

From the preceding paragraphs it is clear that biological sciences are an important part of the foundation of clinical psychology. However, many other areas of science also contribute significantly to making clinical psychology a science-based profession. Professor Jan Br  r of the Norwegian University of Science and Technology, Trondheim, Norway, has contributed an excellent chapter on the contributions of anthropology to clinical psychology. He discusses the concepts of health and illness, particularly what causes illness, within a cultural context. He draws information and examples from a wide range including ancient and contemporaneous Western, Eastern, and African cultures. He also discusses the development of the role of healer historically in different cultures and discusses the development of clinical psychology in this context. Professor Br  r's chapter helps us see clinical psychology within the "big picture."

Individuals have different abilities, tasks to perform, and needs at different times in their lives. All of these are important to the clinical psychologist. Drs. Gisela Labouvie-Vief, Wayne State University, and Manfred Diehl, Colorado State University, both in the United States, have prepared a thorough review of the theories of life-span development along with discussion of research methods and findings in this area. The information contained in this chapter is crucial to the clinical psychologist interested in understanding and providing care for patients.

Mental illness is sometimes described in terms of one's inability to function effectively and cope in interpersonal relations and psychotherapy has been described as the art of persuasion. While there is more to it than that, it is impossible to understand psychopathology or to offer effective treatment without taking into account the social context in which the person functions. Professor John Harvey and Ms. Julia Omarzu of the University of Iowa in the United States have prepared an excellent chapter reviewing areas of social psychology useful to the clinical psychologist. They deal with social perception, attribution, influence and persuasion, interpersonal attraction, relationships, altruism, and helping behavior.

The field of cognition in psychology deals broadly with the process by which we acquire information, process it, respond to it, and how it interacts with and affects feeling and behavior. Much in this area is crucial to understanding normal as well as abnormal behavior. Drs. Richard Hoffman and Mustafa al'Absi, University of Minnesota, Duluth School of Medicine, in the United States, have prepared an excellent and very readable account of some of the major research in this area. They also indicate how this information relates to psychotherapeutic intervention, notably in cognitive therapy.

A scientific discipline unique to health professions is the field of epidemiology. As Drs. James Anthony and Michelle Van Etten of the Johns Hopkins University in the United States point out in their chapter, the epidemiologist attempts to answer five questions, namely for a given illness how many are affected, where are the affected cases to be found, why are some people affected while others are not, what influences who becomes affected, and what can be done to intervene? Answers to these questions are very useful to the health professional. The answers often lead to clues regarding the etiology of an illness. They also alert health professionals to serious health problems and suggest effective interventions. Epidemiologists are sometimes referred to as "medical detectives" because their work often involves solving mysteries about the occurrence of outbreaks of illness.

Psychological theory provides the basis for decision making in clinical psychology. While there are hundreds of psychological theories that may be employed, there are four major theoretical approaches that provide the major rationale for most clinical psychologists. Volume 1 of *Comprehensive Clinical Psychology* concludes with four excellent chapters in which these approaches are reviewed by Professor Graham Davey, University of Sussex, United Kingdom (Learning Theory); Dr. Peter Fonagy, University College London, United Kingdom (Psychodynamic Theory); Dr. Constance Fischer, Duquesne University, United States (Phenomenological, Existential, and Humanistic Foundations for Psychology as a Human Science); and Drs. Jay Lebow, Chicago Center for Family Health, United States and Alan Gurman, University of Wisconsin Medical School, United States (Family Systems and Family Psychology). Each of these authors provides a description of the basic concepts of the theory under consideration along with a history of the development of the theory followed by a review of the applications of the theoretical approach to the work of the clinical psychologist.

The material presented in this volume, the first of 11, is intended to provide the historical, theoretical, and scientific foundation for the material that follows in the succeeding volumes. Taken together, the chapters in this volume provide a survey of the major ideas and approaches that serve as a basis for the field of clinical psychology. As the wide range of topics covered will attest, clinical psychology is a very broad field which draws support from many other fields. It is hoped that the present volume will provide information and perspective that will illuminate the topics covered in the following 10.

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# 1.01

# Hippocrates Meets Democritus: A History of Psychiatry and Clinical Psychology

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### 1.01.1 INTRODUCTION

The story goes that Hippocrates (460–377 BC), the famous physician, was once summoned to the Greek city of Abdera by its citizens in order to investigate the seemingly irrational behavior of the philosopher Democritus. Democritus (460–370 BC) is best known today for his view that everything consists of tiny particles called atoms. On this occasion, Democritus was seated under a plane tree surrounded by the carcasses of dogs and cats. He was experiencing an episode of melancholy and was dissecting these animals in order to discover the source of black bile, considered to be responsible for such mental disturbances (Burton, 1621/1971). The term “melancholy” literally means black bile in Greek, and Hippocrates’ theory of melancholy, like that of Democritus, was that it was caused by an imbalance of the “humors” of the body (e.g., Jackson, 1978). Hippocrates thus considered Democritus to be rational, not insane.

Hippocrates was not a psychiatrist, nor was Democritus a clinical psychologist. It would be

many centuries before either of these specialized fields was to emerge. Nevertheless, the principal theme of this chapter is that even from early historic times, mental disorders were the concern not only of physicians but also of philosophers and others who did not practice medicine. This fact can serve as a sort of historic precedent for the existence of clinical psychology and related nonmedical fields concerned with mental disorders. As Democritus once said in a letter to Hippocrates:

Wisdom is the sister of medicine: the one rescues the soul from passions, the other alleviates the disease of the body. The mind benefits from health, whereas ill health dampens the desire for virtue, and illness binds the soul. (Temkin, 1991, p. 70)

This history of “clinical psychology” is thus at the same time a history of aspects of medicine, psychiatry, and other related fields. It is concerned with a broad spectrum of pathophysiology, psychopathology, assessment, and various types of interventions for mental disorders.

The author's earlier inquiries into the history of clinical psychology concerned the organizations of clinical psychologists that developed in the USA beginning in 1917 and some of the practitioner pioneers in the field including Lightner Witmer, J. E. W. Wallin, and Leta Stetter Hollingworth (Routh, 1994, 1996, 1997). These writings gave relatively little recognition to events before the 1890s, to the contributions of adjacent disciplines such as psychiatry, or to the work of colleagues in other countries. The request from C. Eugene Walker to write the present chapter was interpreted as an invitation to explore not just the organizational history of clinical psychology but its substance as well, not just in recent times but from its origins. In its first 100 years, clinical psychology has become so intertwined with psychiatry (especially in the domain of research) that it made more sense to consider them jointly rather than separately. It soon became evident that writing such a history was nothing short of a lifetime project. However, the present chapter was prepared over only a two-year period and thus required the use of many secondary sources and even some tertiary sources in order for it to be completed at all. The main justification for trying to carry out such a task is that most clinical psychologists are remarkably ignorant about the history of their field.

The chapter begins with a brief history of ideas concerning mental illness in ancient, medieval, and early modern times. It then discusses the development of the specialty of psychiatry in the eighteenth century and after and of clinical psychology about 100 years later. Finally, it describes the further development of the two fields, considered as aspects of a larger whole, during the twentieth century, with a focus on developments in the areas of psychopathology, assessment, and treatment. Many histories of psychiatry and psychology have been written, beginning in the early nineteenth century (e.g., Marx, 1994). So far as the author is aware, however, this admittedly modest effort is the first attempt to write a combined history of psychiatry and clinical psychology.

### 1.01.2 THE ANCIENT GREEKS AND ROMANS

The Hippocratic writings of the fifth and fourth centuries BC and later (which have diverse authorship) consider mental disturbances to be illnesses like any other. Besides melancholy, the Hippocratic list of mental disorders included phrenitis, mania, hysteria, and paranoia. Plato (427–347 BC) added the category of dementia as well. Phrenitis (which literally

means inflammation of the mind) referred to the situation in which a person with fever begins to talk nonsense; this was considered a temporary condition which would clear up as soon as the fever was over. Mania referred to a furious, acute mental disturbance in the absence of fever. Hysteria consisted of bodily complaints made by women, interpreted as being due to the wandering of the uterus. Paranoia described a condition in which the person suffered from abnormal thinking (in the absence of fever). Dementia referred to a deteriorating mental condition often seen in the elderly. Plato, in the *Republic*, describes in detail individual differences in learning ability. He was also aware of the existence of persons with congenital mental incompetence (Plato, 1933). The terminology of mental disorders often changes over time, as do concepts of psychopathology, although some of the phenomena these terms and concepts refer to may be timeless. It would thus be a mistake to identify the above concepts of the Greeks with those we use today, even when they are called by the same names.

Hippocratic medicine emphasized the importance of proper diet, exercise, and moderation in all things. It considered most illnesses to result from an imbalance of the “four humors”: blood, black bile, yellow bile, and phlegm. Given this theory, one reasonable type of treatment tried to reduce the excessive amount of a particular humor, by bleeding the patient, using emetics or purgatives, or inducing sweating.

Different schools of thought resembling some present controversies in the philosophy of science were already present among Hippocratic physicians, as the following quotation makes clear:

Two main sects had formed among his followers. For the so-called Dogmatists, or Logicians, Hippocrates was the founder of a scientific medicine based on natural philosophy, anatomy, and physiology—in short, on hidden causes, as their opponents, the Empiricists contended. In the opinion of the latter, nature could not be fathomed; hence speculation about these hidden causes, which included elements, humors, and pneuma, was useless. They held that medicine had to make do with a knowledge of obvious causes, such as hunger, cold, and sleep. The Empiricists thought of Hippocrates as himself an Empiricist, their great clinical teacher of the past. (Temkin, 1991, p. 6)

Galen (AD 130–200), the most famous successor of Hippocrates, was born in Greece but taught and practiced in Alexandria and Rome. He was an early “scientist–practitioner” in that he was an expert in anatomy as well as medicine. He carried out some of the first physiological

experiments. For example, he demonstrated that one could eliminate a pig's squeal by sectioning a particular nerve (Sarton, 1954; Temkin, 1973). Some of the mistakes in Galen's writings on anatomy resulted from the dissection of apes rather than human cadavers (human dissection not being allowed).

### 1.01.3 CHINA AND INDIA

The study of psychopathology did not originate only in Greece but has other roots throughout the world. A well known example from two centuries BC in China is the *Yellow Emperor's book of internal medicine* (Veith, 1955). This book interprets disease (according to Veith, this included the kind we would consider to be mental illness) as resulting from an improper balance of the powers of "yin" and "yang." These are considered to be caused by moral transgressions of Tao, "the way." Traditional Chinese medicine as described in this book includes an elaborate description of procedures such as acupuncture, still in use in China and elsewhere today. According to these precepts, the person who lives properly has every reason to expect to reach age 100, in vigorous good health. It is proverbial that mental illness is rarely diagnosed in China, perhaps in part because it would involve public loss of "face." Also, children feel obliged to honor their parents and to care for them even if senile dementia has made them burdensome and foolish. The identified prevalence of mental illness is also low among the overseas Chinese. Veith (1963) noted that while yin and yang are implicated in the scholarly account of mental illness in China, the popular view is different. Uneducated people interpret disturbed behavior as being the result of supernatural causes such as spirits entering into the body of the sufferer. Exorcism is thus a common treatment.

An important nonmedical source of the assessment component of present-day clinical psychology and psychiatry is the tradition of formal mental tests established in China. Public examinations served for almost 1000 years as the principal way of recruiting and selecting "Mandarin" civil servants. In China, this system was abolished by the empress dowager only in 1905 (Hanson, 1993), which oddly enough, was just when psychology's involvement in psychometrics was beginning.

In India, at least according to Sigerist's (1945) admittedly dated account, many persons receive medical care, whether for physical or mental illness, only from indigenous practitioners. Some of these doctors follow the principles of the *Ayurveda*, the "science of longevity," as described by Brahmin texts dating back 3500

years. Others follow the Unani, or Greek school, as filtered through Arabian and Persian cultures.

### 1.01.4 THE NEAR EAST

Judaism, Christianity, and Islam have been competing religious and cultural forces for millenia in the Near East and elsewhere, as they continue to be. All of them have their own methods for treating illness, be it physical or mental. Consider, for example, Jesus' restoration of the sight of a man with a missing eye and raising Lazarus from the dead. As Temkin (1991) points out, secular medicine did not pretend to perform such feats. In Greece, Christianity eventually superseded the Asclepian religious approach to healing. This involved visiting the temples of Asclepias, consulting the priests, and perhaps sleeping there overnight. However, these religions each managed to co-exist relatively peacefully with Hippocratic medicine and its descendants right up to the conquest of Alexandria (the center of Hellenistic medicine) in AD 642. After that event, Islamic physicians assimilated the medical teachings of the Greeks and made their own independent contributions to the field.

The most famous of the Islamic physicians was Avicenna (AD 980–1037), whose Arabic name was Ali al-Husayn ibn Sina. Magner (1992) cites some of the work of Avicenna as an early example of psychotherapy:

[One] challenging case involved a young man who suffered from melancholy and the delusion that he was a cow. The man mooed loudly, refused to eat, and begged to be killed and made into stew. The patient cheered up immediately when Avicenna sent word that the butcher would come to slaughter him. Finally, Avicenna came to the sickroom with a butcher's knife and asked for the cow. Mooing happily, the young man was bound hand and foot, but after a thorough examination, Avicenna declared that the cow was too thin to be butchered. The patient ate so eagerly that he soon recovered his strength and was cured of his delusion. (p. 143)

According to Dols (1987) and Magner (1992), the Muslims also built hospitals including provisions for the care of the mentally ill. The largest of these was established in Cairo in 1286. Formerly a palace, this hospital had a capacity of 8000 patients. Other such hospitals were located in Baghdad and Damascus and existed as early as the ninth century AD. The treatments for mental patients included baths, massage, bloodletting, leeches, cautery, medication with purgatives, emetics, sedatives, and opium. There was also concern for the patient's diet, opportunities to participate in pleasant conversation,

and diversions such as music, dance, and drama. If necessary, Dervishes performed exorcisms. Some violent patients were in chains, and whips were used on occasion.

The condition we know as mental retardation was also familiar. As Dols (1987) says:

The village idiot, the court jester, and the wise fool were tolerated forms of possible derangement. The archetypal wise fool in Islamic literature is Buhlul, the entertaining and harmless critic of social conditions and mores. He typically lurks undisturbed in the cemetery, is molested in the streets by children, or is fettered and put in chains. Having abandoned everyday cares, he trusts in God's aid and men's charity. His intense sensitivity plays on the ambiguity between the unholy and the holy fool. (p. 13)

### 1.01.5 MEDIEVAL EUROPE

Medieval European times were not noted for scientific advances of any kind, certainly not with respect to the understanding and treatment of psychopathology. The medieval era does have its positive side in terms of the Christian emphasis on the virtue of caring for the unfortunate, including those with mental disorders. Thus, in the sixth century AD, Benedict of Nursia proclaimed the ideal of service toward those living in monasteries who were ill (Jackson, 1972). However, such care did not extend very far to those in the community, as Rosen (1964) notes:

During the medieval period, public authorities took only limited responsibility for the mentally deranged. Mentally or emotionally disturbed members of a community were left at liberty as long as they caused no public disturbance. Custody of the mentally ill generally rested with their relatives and friends; only those considered too dangerous to keep at home, or who had no one to care for them, or who were socially disturbing, were dealt with by communal authorities. (p. 377)

The institutions for the mentally ill that developed in that era provided at most food, lodging, and basic physical care. They included general hospitals such as the Hotel-Dieu in Paris, the hospital of St. Mary of Bethlem in London (built in 1247, later notorious as "Bedlam"), and the madhouses that were part of the hospitals at Elbing (built in 1326) and Hamburg (built in 1375). Priests offered exorcism to persons considered to be possessed by devils, and both the secular and the ecclesiastical authorities sometimes subsidized pilgrimages by the mentally disordered to religious shrines such as Lourdes.

In the twelfth century a church was built at Gheel, a village north of Brussels, Belgium, to

honor St. Nymphna. The village developed the pattern of boarding mentally disturbed patients in the homes of the residents, and this Shrine of Gheel became an enduring legend in community care. The famous psychiatrist Esquirol visited it in 1822.

One high point in medieval and renaissance thought concerning mental disturbance is the philosophical writings of the Portuguese King Duarte (1391–1438) known as *Leal Conselheiro* ("loyal counselor"). Duarte experienced mental depression and undertook to write about it as a form of self-therapy. What he had to say is well worth quoting:

And it seems to me . . . that sorrow generally has six causes: first and chiefly, fear of death, dishonor, pain, or spiritual and bodily suffering. Second, anger . . . Third, strong desire unfulfilled or delayed. Fourth, unhappiness which we acquire because of dishonor, death, losses, imprisonment, illnesses, retention, and longing. Fifth, disordered compression, which truly is called an illness of the melancholy humor. Sixth, talk, conversation of sad persons . . . By each of these means, more or less, we become sad, according to the feelings and passions which are dominant in each of us. (Roberts & Sacks, 1954, p. 22)

As it turned out, Duarte's melancholy abated after his mother became ill, and he turned his thoughts away from himself to care for her.

An acknowledged horror story of the middle ages and renaissance (from the standpoint of the understanding and treatment of psychopathology) is the publication of *Malleus Malificarum* ("hammer of the witches") by two German Dominican monks, Heinrich Kraemer and Johann Sprenger (1486/1971). This was a manual for the identification, torture, and trial of witches. It was endorsed by the Pope and went into 19 editions over the next 300 years (Leigh, 1957). According to this book, witches, though they might appear to be merely lunatics, were actually in league with the devil and caused all manner of mischief, including hailstorms, crop failure, and sexual dysfunctions such as impotence in other persons. Most of the "witches" who experienced the regimen prescribed by these two doctors of the church were aging women.

### 1.01.6 FROM THE RENAISSANCE TO THE SEVENTEENTH CENTURY

The answer to the "witches' hammer" was Johann Weyer's book, *De Praestigiis Daemonum* ("the slight of hand of demons"), published in 1583 (Mora, 1991). Weyer, an experienced physician, was employed by a wealthy duke and seemed to be unafraid of the enemies he might make by his writings. He had considerable

personal experience in examining various individuals who were accused of witchcraft and usually found that they were poor, frail, mentally unstable women who could not possibly have done the deeds of which they were accused. He thus disputed Kraemer and Sprenger's *Malleus Malificarum* very thoroughly. Weyer's book was put on the Papal index of forbidden writings.

Another important medical figure of the renaissance was Paracelsus (1493–1541), the name adopted by a flamboyant Swiss man named Philippe Aureole Theophrastus Bombastus von Hohenheim. Paracelsus was of modest background rather than being a member of the nobility (as his name might imply) and was not as well educated as many of his medical peers, whom he often scandalized by communicating in the vernacular rather than in Latin, the common language of scholars of that time. His father had worked in the mines, and Paracelsus knew a lot about minerals. An alchemist as well as a physician, he introduced chemical treatments. Temkin (1946) estimates that Paracelsus was as important to the history of medicine as Luther was to church history. The followers of Galen had always recommended herbal remedies, and now Paracelsus came along with chemical ones—the founder of a new school of thought known as “iatrochemistry.” Paracelsus was also responsible for a very modern-sounding insight into the possible role of minerals in the drinking water in the etiology of goiter and Cretinism (a form of what we could now call mental retardation) that was so prevalent in Switzerland.

Robert Burton (1577–1640), the author of *The anatomy of melancholy* (Burton, 1621/1971), lived in this era. Burton was not a physician but a clergyman and an Oxford don. He was in a state of melancholy himself when he wrote this huge universal compendium of ancient knowledge partly as a way of distracting himself. Burton referred to himself as “Democritus Junior,” thereby alluding to the pre-Socratic philosopher mentioned in the title of the present chapter. Burton evidently did not know of King Duarte's writings, but there are interesting parallels in the lives and thinking of Democritus, Duarte, and Burton. Clinical psychologists might do well to consider all of them as mentors.

The physician Sydenham (1624–1689) wrote a treatise on psychological disorders that has been described as the most important one of the seventeenth century. Sydenham reportedly considered psychological disorders to be as common as physical ones. He was also something of a legend as a therapist, as the following incident reveals:

... in dealing with a patient who stubbornly refused to get well, Sydenham suggested a consultation with the wonder-working Dr. Robinson at Inverness. The patient made a long trip on horseback only to find that there was no such doctor, but, as Sydenham expected, anticipation, exercise, and anger effected a cure. (Magner, 1992, p. 222)

Foucault's (1961) account of the classical age asserts that the seventeenth century was an age of increased “confinement” involving not only the mad but also criminals, the poor, and the indigent ill all over Europe. On a more positive note, Foucault credits Thomas Willis with being the first to make the observation that melancholia and mania tended to alternate in the same individuals. Willis wrote about this in 1681.

Finally, some mention should be made of a late-seventeenth century philosopher, John Locke (1632–1704), and of his influential book, *An essay concerning human understanding* (Locke, 1689/1824). Locke is well known to psychologists as one of the British Empiricists and as an associationist (Hoeldtke, 1967). In this book, Locke argued that the mind is a blank slate at birth and that its contents are acquired through sensory experiences and by reflection upon these. One assertion of his that was influential regarding mental disorders was that whereas idiots were incapable of associating ideas, madmen associate ideas incorrectly.

### 1.01.7 THE EIGHTEENTH CENTURY, INCLUDING THE EMERGENCE OF PSYCHIATRY

At the beginning of the eighteenth century, Edinburgh was a major intellectual center. It was associated with the Scottish Enlightenment and with common sense philosophy and psychology. The University of Edinburgh was also a center of medical education, with its influence reaching across the Atlantic (Carlson & Simpson, 1969) and into France. Vienna was also respected as a center of medical thought as was Montpellier in France, but the German states were in a rather backward condition, perhaps due to the after effects of the 30 Years War. At this time the dominant figure in Scottish medicine was perhaps William Cullen (1710–1790), who founded a school of “neuropathology.” The modern sense of this word is somewhat misleading. It does not adequately convey the fact that Cullen was trying to represent the brain and the nervous system as the “master” influence on the health of the body (Riese, 1949) as an alternative to the “humoralism” still lingering from the ancient days of Hippocrates. Cullen's major work was a book,

*First principles of the practice of physic*, published in 1776–1784. Naturally, the “neuropsychology” approach had a lot to say about mental disorders.

This was also the time when the tradition of “mad doctors” emerged more fully in the British isles (Hunter & McAlpine, 1963). The first of these had been John Archer, who already in the seventeenth century became the proprietor of a private madhouse. The most eminent “mad doctor” of the eighteenth century was a man with the interesting name of William Battie (1704–1776), who published a *Treatise on madness* in 1758. According to Porter (1987), Battie’s book contains the key ideas of moral therapy as subsequently practiced by William Tuke (1732–1822). The number of private madhouses increased greatly during the late eighteenth and early nineteenth centuries. Before the advent of moral therapy, many of their proprietors used fear and restraint, including chains, to deal with the inmates (Scull, 1993). According to Bynum (1974), the first act aimed at licensing private madhouses was passed in Britain in 1774. The law did not require the proprietors to be medically qualified but did require a medical certificate for a patient to be admitted. Notably, 30 years earlier than this, the prevailing law had implied madness to be a condition that anyone could recognize and that any magistrate could formalize; the legal recognition of special knowledge about madness is part of what confirms the emergence of what we now call psychiatry during the eighteenth century in Britain. As Ackerknecht (1986) noted, the title “doctor” when used for the proprietor of a madhouse could refer to a university-trained physician, a surgeon, an apothecary, or simply a quack.

Although the history of psychiatry has somewhat indistinct beginnings in various European countries, the specialty was well established on both sides of the Atlantic before 1800. As Porter (1987) says concerning England:

Around 1700, the top practitioners for the disturbed were all-round physicians: within a century they were specialists. Mad-doctoring came of age on the day in 1788—5 December in fact—when the failure of the King’s physicians-in-ordinary to master George III’s delirium was acknowledged by the summons to the specialist, if very much despised, mad-doctor Francis Willis. (p. 175)

Some would probably not choose to honor Willis as the founder of this new professional specialty, since he represented the old-fashioned coercive approach rather than the newer one of moral treatment. In any case, the specialists known as “mad doctors” in Britain were called

“alienists” in France, and ultimately termed “psychiatrists” in the German states and then all over the world. By the time of the American and French revolutions, the individuals who stood out as pioneer figures in this field included Benjamin Rush (1745–1873) in the United States, Phillippe Pinel (1745–1826) in France, Vincenzo Chiarugi (1759–1820) in Florence, and Francis Willis (1718–1807) in England.

It appears that the leadership in medicine shifted from Edinburgh to Paris at about the time of the French revolution in 1789 (Ackerknecht, 1967). The French physician Philippe Pinel, who is often considered to be the founder of psychiatry, was much influenced by the doctrines of William Cullen of Edinburgh. Pinel actually translated one of Cullen’s books into French. Cullen’s writings also influenced Benjamin Rush, who incidentally was a signer of the American Declaration of Independence.

How were mental patients treated in the eighteenth century? In some respects, the ancient ways of Hippocrates prevailed. Benjamin Rush was especially well known for his propensity to bleed his patients, and may have inadvertently hastened the death of many of them. Many eighteenth century “mad-doctors” in Britain emphasized coercive methods, including chains and fetters. The important therapeutic innovation of this time is what was known as “moral treatment.” This phrase emphasized the attempt to use psychological and social influences and to treat the mentally disturbed with kindness rather than physical force, chains, or medications. The asylum began to be considered as a therapeutic intervention rather than simply a way to house and contain mental patients. In retrospect, moral treatment is usually portrayed as a tremendous advance, perhaps the very cornerstone of the new specialty of psychiatry. Indeed, it was obvious from the first that many patients did not require restraint after all, and often responded well to kindness and understanding. Who deserves the most credit for the development of moral treatment is somewhat unclear. Philippe Pinel became the focus of a legend, not only as a founder of psychiatry but also as the one who was the first to remove the chains from his patients at the Bicetre hospital in Paris. Actually, the evidence is that he was not the first to do this but served an apprenticeship to Citizen Jean B. Pussin in this respect (Weiner, 1979). Another contender for the credit of founding moral treatment is William Tuke, who was not a physician but a Quaker tea merchant. Tuke founded the York Retreat in 1796 as a way of providing a gentler approach than that offered by other available institutions. Tuke’s work was influential and was copied, for



example, by the founding of the Hartford Retreat in the United States. Finally, one must mention Vincenzo Chiarugi as one who opposed restraints and harsh measures. Chiarugi was born in Florence. His patron was Pietro Leopoldo, the Duke of Tuscany. Chiarugi directed the Hospital of Bonifacio in Florence and published three volumes on his work in 1793–1794, under the title, *Della Pazzia in Genere e in Specie* (“on madness in general and in specific”).

In any case, not all scholars view the advent of “moral treatment” as an unmitigated improvement. Foucault (1961) became famous for noting that “moral treatment” could be considered simply a different kind of oppression. In making the mad responsible for their own self-control, Tuke and Pinel may have merely substituted guilt for external coercion, and unduly imposed demands for a certain type of bourgeois respectability.

Even with all the emphasis on the absence of chains in the era of moral treatment, Pinel and his colleagues in other countries did use restraint when necessary, including strait jackets and isolation rooms (Woods & Carlson, 1961). Pinel avoided punishment and was reluctant to use the traditional Hippocratic treatments of bleeding, emetics, and purgatives. He observed the progress of the patient and tried to provide a humane environment, hoping that the healing forces of nature would prevail over the illness. In Britain, the physician who became most famous for a policy of nonrestraint was John Conolly, who had charge of the Hanwell Asylum in England in the nineteenth century (Scull, 1993).

A countervailing force at this time was the emerging movement of romanticism, associated particularly with the name of the philosopher and social contract theorist, Jean Jacques Rousseau (1712–1778). In his influential novel, *Emile, or education*, and in his other writings, Rousseau (1911) stressed the importance of what emerged naturally in human development as opposed to what is contrived by the educational system. He emphasized the heart over head, in contrast to what he considered the overintellectual emphasis of the Enlightenment.

Mesmerism emerged during the eighteenth century, more as a treatment for medical problems than for mental disorders. This development came from the work of Franz Anton Mesmer (1734–1815), a physician who was originally trained in Vienna but who later flourished in Paris. Mesmer’s theory about his therapy explained it in terms of magnetic forces being transmitted from the body of the doctor to that of the patient, curing all kinds of ailments. The theory was based on the study of electricity

including the role of electrical stimulation on the movement of frogs’ legs (the original “animal magnetism”). This was debunked as an explanation by more than one distinguished commission in Paris. The commissioners included such experts on electricity as Benjamin Franklin, then serving as ambassador from the United States. These scientific authorities tended to believe that the therapy was based merely on “imagination.” At the time, the effect on Mesmerism was quite negative in terms of the opinions of educated persons, although its practice continued not only in France but also in the German states and elsewhere (Gauld, 1992). Ellenberger (1970), in his book *The Discovery of the unconscious*, portrays Mesmer’s work as being an important link between the medieval use of priestly exorcism and the subsequent development of psychoanalysis by Freud.

It happened that the first cases of what was subsequently identified as neurosyphilis began to appear in the 1780s (its prevalence rose later, during the nineteenth century). This disease, which is known to have a latent phase lasting 10–15 years from the time of initial infection, was manifested in the form of the general paralysis of the insane (GPI) or paresis. Neurosyphilis included dementia paralytica, progressive paralyses, terminal convulsions, and death. Its early symptoms could include mania and locomotor ataxia (tabes dorsalis). Paresis most commonly affected middle-aged men.

Toward the end of the eighteenth century there were important new developments in the treatment of congenital mental handicaps. One innovator was Giacobbo (Jacob) Rodriguez Pereire (1715–1780). Pereire (or Pereira as his name is sometimes spelled), living in France at the time, developed methods for teaching the deaf to speak. He never revealed the particulars of his methods but was obviously successful in doing what he claimed, as revealed by the fact that his deaf pupils did learn to speak. A famous disciple of Pereire was the physician Jean Marc Gaspard Itard (1775–1838), who attempted to use some of the same approaches to train the Wild Boy of Aveyron. Itard considered his attempts to socialize this feral child and to teach him language to be unsuccessful, but in retrospect they have proven to be a model for educators working with children with mental retardation or autism. Itard’s work was thus influential in the development of fields as diverse as otolaryngology, child psychiatry, special education, and clinical psychology.

As noted by Shorter (1997), in the late eighteenth century patients with minor nervous complaints tended to seek out treatment at spas. Thus, there grew up a cadre of “spa doctors” to

care for them. In addition to bathing in or drinking natural mineral waters, such patients might also be subjected to actual “hydrotherapy,” special diets, or to mild shocks from some special electric apparatus. One can see in the practice of such physicians much of what was more likely to be identified as clinical “neurology” for many of the years of the following century.

### 1.01.8 THE NINETEENTH CENTURY, INCLUDING THE EMERGENCE OF CLINICAL PSYCHOLOGY

Paris continued to dominate the medical scene throughout the first half of the nineteenth century, with its emphasis upon the hospital as the site of clinical investigation (Ackerknecht, 1967). It was there that treatment was carried out and there that the correlation of clinical symptoms with the findings of pathological anatomy were observed. Pinel was if anything as famous in France for his work in internal medicine as in psychiatry. Pinel’s most famous pupil, Jean-Etienne-Dominique Esquirol (1772–1840), unlike his teacher, was able to devote his entire professional career to psychiatry.

Berrios (1996) identifies the second decade of the nineteenth century as a time when the previous global descriptions of types of mental disorders began to be elaborated into detailed symptom lists and definitions, or what is now called descriptive psychopathology. This process was essentially complete by the time of the World War I. Emil Kraepelin (1856–1926), the leader in this domain, published the final volume of the eighth edition of his textbook of psychiatry in 1915.

The romantic movement within German psychiatry continued to flourish in the 1820s and in places perhaps until 1848. An example of such a psychiatrist was Johann Heinroth (1773–1843), who was appointed to the first known chair of psychische Heilkunde (“psychotherapy”) at the University of Leipzig in 1819. The type of psychotherapy favored by Heinroth made considerable use of Christian precepts such as sin, repentance, and religious conversion. The romantics, influenced by German nature philosophy, were “Psychikers” who battled against the “Somatikers.”

Formal organizations of psychiatrists began at about the same time in France, Britain, and the United States during the 1830s and 1840s, when public mental asylums began to be constructed in these countries. In France, a law was passed in 1838 creating a national system of asylums (Goldstein, 1987). It was supported by liberals (who tended to be antic-

lerical) and to some extent replaced the previous system run by the Catholic orders. Even after the secularization of mental hospitals, many of the psychiatric nurses in the nineteenth century were supplied by Catholic religious orders such as the Sisters of St. Vincent de Paul (Santos & Stainbrook, 1949). The French psychiatric journal, *Annales Medico-psychologiques*, began publication in 1843, and in 1848 the Société medico-psychologique was founded (Dowbiggin, 1989). In England, which passed a similar law in 1845, there had previously been founded in 1841 an Association of Medical Officers of Asylums and Hospitals for the Insane (Scull, MacKenzie, & Hervey, 1996). In 1853 the Association’s journal began, with Sir John C. Bucknill (1817–1897) as editor. Called the *Asylum Journal*, it eventually became the *British Journal of Psychiatry*. In the United States, Dorothea Dix (1802–1887) presented her famous Memorial to the Massachusetts legislature, urging the state to take responsibility for the insane by building and staffing proper asylums for them (Gallaher, 1995). In 1844 the forerunner of the American Psychiatric Association was founded and began to publish the *American Journal of Insanity*, eventually called the *American Journal of Psychiatry* (Grob, 1962).

In Paris, Edouard Seguin (1812–1880), a physician who was a pupil of Itard, in 1837 opened an innovative training school for imbeciles. Seguin later emigrated to the United States and became the most influential person in the founding, in 1876, of the Association of Medical Officers of American Institutions for Idiotic and Feeble-minded Persons.

In Switzerland, in 1841, the physician Johann J. Guggenbuhl (1816–1863) founded an institution for Cretins at the Abendberg (Kanner, 1959). Guggenbuhl received praise from all over the world for this innovation, but the Abendberg was closed down after only a few years because of mismanagement. Theodor Kocher (1841–1917), a Swiss surgeon, discovered the cause of Cretinism (Schlick, 1994), despite his lack of any initial interest in the problem of mental retardation. Kocher, in ablating the thyroid gland of certain of his patients with goiters (especially the children among them), noticed that he had inadvertently created a sort of artificial cretinism. He later made a visit to the Valais and the mountains near Bern in Switzerland to examine some natural cases of cretinism there to compare them with his surgical cases. Of 32 alleged cretins referred to him for examination, he concluded that only six fit the refined criteria his surgical experience led him to develop to diagnose this disease. The other patients, though mentally handicapped,

failed to meet his criteria for such a thyroid-based disorder. Later, Kocher developed techniques for transplanting thyroid glands, the first such successful organ transplant. Thus, he discovered a curative therapy for cretinism as well as its cause. He won the Nobel prize for these discoveries after the turn of the century, in 1909.

In England, physician J. Langdon Down (1828–1896) wrote a notable paper on the mental retardation syndrome now associated with his name. He misinterpreted the condition as representing some kind of an evolutionary throw-back to “Mongol” ancestors.

In the late nineteenth century, let us say after 1848, the dominant role in medicine and psychiatry shifted to the German states (Ackernect, 1967). The unified country of Germany emerged in 1871 after the Franco-Prussian war. German medicine was dominated by laboratory approaches rather than by work in the clinic or the hospital. As the German physician Bernard Naunyer (1839–1925) was reported to have said: “Medicine will be a science, or it will not exist at all” (Magnus-Levy, 1944). The leaders in German psychiatry would surely include Wilhelm Griesinger (1817–1868) and later Emil Kraepelin (mentioned above in relation to descriptive psychopathology). Griesinger published an influential book, *Mental pathology and therapeutics*, in 1845. He was the director of psychiatry at Berlin’s Charite Hospital and held the chair in psychiatry at the University of Berlin, beginning in 1864. Unlike other psychiatrists of the time, Griesinger did not work in a mental hospital out in some isolated rural area. He tried to make psychiatry an integral part of a major urban university. Griesinger has become infamous among the psychodynamic psychiatrists and psychologists of our day for his rather dogmatic statement that mental diseases are brain diseases. What is not often realized by such critics is that Griesinger was quite sophisticated in the psychology of his time. He made extensive use of the concepts of the psychologist Johann F. Herbart (1776–1841) concerning the unconscious, the threshold of consciousness, and the “apperceptive mass” used by people in interpreting their experiences. Griesinger founded a new journal with the title [English translation] *Archives for Psychiatry and Nervous Diseases* in 1867 to focus on the type of neurologically oriented field of psychiatry he envisioned. However, he died the next year, with his vision still unrealized.

One German psychiatrist who took the road laid out by Griesinger was Theodore Meynert (1833–1892), a professor in Vienna, who was incidentally one of the teachers of Sigmund Freud (1856–1939). This was in the days when

Freud still intended to become an academic neuroanatomist. Meynert was the investigator who distinguished the various layers of the cerebral cortex in terms of cell types. Although he had a poor reputation as a clinician, Meynert trained several students who themselves made contributions to neuroanatomy. In 1865 the French physician Pierre P. Broca (1824–1880) had developed his influential hypothesis that expressive aphasia results from damage to the third frontal convolution of the left hemisphere of the human brain. At that time, Broca had only a single case with an autopsy to report. One of Meynert’s pupils, Carl Wernicke (1848–1905), later described a type of receptive aphasia that was quite distinct from Broca’s aphasia, anatomically as well as functionally. The patients Wernicke identified had lesions in the posterior perisylvian region of the left hemisphere, were unable to understand spoken words, and spoke in a fluent but incomprehensible jargon. Julius Eduard Hitzig (1838–1907), another Meynert pupil, carried out research on dogs indicating that the brain responds to local electrical stimulation.

Franz Joseph Gall (1759–1828), a German who received his M.D. from Vienna, also began his career with well-respected research on the topic of the localization of function in neuroanatomy (Temkin, 1947). His viewpoint was opposed by Pierre Flourens (1794–1867), who did not find evidence for such localization in his research on the cerebral cortex of pigeons. Gall and a colleague named Spurzheim went on to try to establish a correlation between human “neuroanatomy” (as indexed by bumps on the skull) and psychological characteristics. Spurzheim called this field phrenology, which translates simply as the study of the mind. Phrenology went on to considerable popular and commercial success in England and the United States and at first was supported by a significant element within the medical and psychiatric community. For example, John Conally (1794–1866), the prominent British alienist and champion of the doctrine of nonrestraint, was also a supporter of phrenology. Some supporters managed to combine phrenology and hypnotism (Gauld, 1992). Thus, they developed a professional approach that included a sort of prototype of neuropsychological assessment and psychological treatment. Ultimately, however, phrenology was consigned to the status of a pseudoscience and its practice condemned as quackery.

Late nineteenth century Germany was also one place where the new discipline of psychology emerged. It was at first modeled upon experimental physiology, for example, the work of Ernst Weber (1795–1878) and Gustav Fechner

(1801–1887) leading up to Fechner's 1860 book, *Elements of psychophysics*. The founders of psychology as an independent academic discipline in the 1870s are generally considered to have included William James (1842–1910) at Harvard University in the United States and Wilhelm Wundt (1832–1920) at the University of Leipzig, Germany. Of these founders of psychology, the one who made the greatest contribution to the study of psychopathology was William James, as shown, for example, by his 1896 Lowell lectures on exceptional mental states (Taylor, 1982).

The American Psychological Association was founded in 1892 by Granville Stanley Hall (1844–1924). Hall tried his best to facilitate a relationship between psychology and psychiatry. A Ph.D., he served at one point as the superintendent of the Bayview mental hospital in Maryland. Later he taught psychology to psychiatry trainees at Worcester State Hospital in Massachusetts. After the turn of the century, in 1909, he was to invite Freud and Jung to America, the only time Freud ever visited the US.

Emil Kraepelin, ultimately a professor at the University of Munich, may have been the leading German psychiatrist of his day. He received his MD at Wurtzburg and served as a resident under Bernhard von Gudden (1824–1886) in Munich (Danek, Gudden, & Distel, 1989). He also studied psychology under Wundt at Leipzig. In his 1893 textbook, Kraepelin first introduced the concept of "dementia praecox." This term combined a heterogeneous group of symptoms including social withdrawal, hebephrenia (silly, childish language), paranoia (delusions), and catatonia (paralysis with waxy flexibility upon passive movement). The reason for combining these symptoms was Kraepelin's hypothesis that many persons with such symptoms tend to become chronic mental patients. Kraepelin considered this syndrome to be a kind of dementia in a young person that predicted the individual's ultimate mental deterioration. He wrote extensively about the entire spectrum of psychopathology and divided the syndromes into 13 major groups.

In 1826 the French physician Antoine L. Bayle (1799–1858) had found an association of GPI with chronic inflammation of the meninges of the brain. In 1851, two French psychiatrists simultaneously came up with the concept we now term bipolar disorder, confirming the statements of Thomas Willis in the seventeenth century. Jean-Pierre Falret (1794–1870) called this condition "folie circulaire," whereas Jules Baillarger (1809–1890) called it "folie a double forme" (Bourgeois & Geraud, 1997). Incidentally, Falret was also the first to coin the term

"folie a deux" for a sort of psychosis of association in which an otherwise normal person acquires symptoms from being around someone with a mental disorder. Thus, a wife might adopt her paranoid husband's view that the house is surrounded by agents bent on harming them.

In England, the most influential scientific figure of the nineteenth century was Charles Darwin (1809–1882), who published his book *Origin of the species* in 1859. His cousin, Francis Galton (1822–1911), was the author of a book on *Hereditary genius*, published in 1869 (Galton, 1978), in which he developed a new field he called "eugenics" attempting to use the principles of genetics to improve the human species. It was Galton's American student, James McKeen Cattell (1860–1944), who first coined the term "mental test" (Cattell, 1890).

French psychiatry had a lengthy involvement with a concept later considered discredited, namely the theory of degeneration of Benedict A. Morel (1809–1884). This theory was a variation of Lamarck's theory of the inheritance of acquired characteristics (Walter, 1956). It led to the expectation that the offspring of disturbed or profligate persons would become worse in each generation. One follower of Morel was Cesare Lombroso (1835–1909) at the University of Turin in Italy. In 1876 Lombroso published a book, *L'uomo delinquente*, concerning what he described as the born criminal, with numerous "stigmata" of degeneration. Richard von Krafft-Ebing (1840–1902), a professor of psychiatry at the University of Vienna, also endorsed a version of degeneration theory as applied to sexual perversions. Krafft-Ebing published a book, *Psychopathia sexualis*, in 1886.

In the United States, George M. Beard (1839–1883), a New York neurologist (i.e., a specialist in nervous and mental diseases), published in 1880 a book about his concept of "neurasthenia," meaning simply weak nerves, with a vast array of symptoms. Beard was interested in electrotherapy and had been involved in the scientific study of hypnosis in the 1870s. Neurasthenia became an extremely popular medical diagnosis for people with chronic fatigue and vague nervous complaints (Rosenberg, 1962). The American Neurological Association had just established in 1871 its *Journal of Nervous and Mental Diseases*. The "neurologists" of those days were in competition for outpatients with psychiatrists, who were still for the most part in institutional practice. According to Shorter (1997, p. 130), neurasthenia was the "standard diagnosis for all functional nervous diseases" up to the time of World War I. These were the kinds of patients for whom the Philadelphia physician Silas Weir

Mitchell (1829–1914), of the Jefferson Medical College, prescribed his famous “rest cure,” or as he tended to say, “Dr. Diet and Dr. Quiet.” The American Neurological Association was founded in 1874. In 1894, Mitchell gave a famous address before the 50th Annual Meeting of the American Medico-Psychological Association (forerunner of the present American Psychiatric Association) in Philadelphia. As a neurologist who worked mainly with outpatients, Mitchell severely criticized his “alienist” colleagues for their lack of connection to the rest of the field of medicine and their lack of research:

Where, we ask, are your annual reports of scientific study, of the psychology and pathology of your patients. . . . Seriously we ask you experts, what have you taught us of the 91 000 insane whom you see and treat? (Mitchell, 1894, p. 101)

This talk, though harsh, facilitated the later integration of these two parts of present-day psychiatry.

In 1880 in New York, a rather short-lived organization was founded called the National Association for the Protection of the Insane and the Prevention of Insanity (NAPIPI). The membership consisted largely of neurologists and social workers who were critical of the asylum psychiatry of the day. In a sense this organization could be regarded as a forerunner of the antipsychiatry movement of the twentieth century (Dain, 1994).

Charles Locock, a London internist, was responsible for initiating the common use of bromides as a sedative in psychiatric practice. According to Shorter (1997, p. 200), Locock happened to mention at a medical meeting in 1857 that he had given 10 grains of potassium bromide per day for two weeks to a patient with “hysterical epilepsy,” with the apparent effect of suppressing the epilepsy. Otto Liebreich, a professor of pharmacology in Berlin, discovered in 1869 that chloral hydrate could relieve insomnia in patients with anxiety and depression (Shorter, pp. 198–199). This is the substance known as “knock-out drops” or “Mickey Finns” and indeed does what it is supposed to do. It is considered more reliable than morphine and can be taken orally (though it has a bad taste). It was widely used in mental hospitals thereafter, despite having some danger of addiction.

The Russian psychiatrist Sergei Korsakoff was the first to describe the syndrome named after him: chronic psychosis and memory loss in patients with alcoholism. Carl Wernicke in Germany had described the acute form of this condition in 1881—mental confusion and staggering gait. These conditions were subse-

quently identified as being due to a dietary deficiency in thiamine, often found in poorly nourished alcoholics.

The French physician Jean-Martin Charcot (1825–1893) was a key figure in the history of several modern specialties, including neurology, psychiatry, psychoanalysis, and clinical psychology. Charcot, the chief physician at the Salpêtrière hospital in Paris, was known for his work on clinical-neuropathological correlations as well as on hysteria and hypnosis. Charcot was responsible for elucidating the neuropathology of amyotrophic lateral sclerosis, now known in the United States as Lou Gehrig’s disease (in France, it is called “Charcot’s disease”). Hypnosis, meanwhile, had been rehabilitated somewhat by the Englishman James Braid (1795–1860) under new terminology. Braid’s 1843 book was entitled, *Neurypnology, or the rationale of nervous sleep*. Charcot successfully defended hypnosis before the French Academy of Sciences on February 13, 1882 (Gauld, 1992, p. 311) and used it in his “experimental therapeutics” of hysteria. Charcot considered hypnotism to be an abnormal process in itself, characteristic only of persons with hysteria. Charcot appointed Pierre Janet as head of a newly established psychology laboratory at the Salpêtrière Hospital in Paris in 1890.

Charcot’s contemporaries in Nancy, France, A. A. Liebault (1823–1904) and Hippolyte Bernheim (1840–1919), considered hypnosis to apply to normal people as well as to those with hysteria. Sigmund Freud translated Bernheim’s book on hypnosis into German. Freud also visited Charcot in Paris. He returned to Vienna and in 1895 published a book that was the beginning of his development of psychoanalysis as a treatment for hysteria that went beyond hypnosis (Breuer & Freud, 1895/1986).

Spiritualists and mental healers throughout the nineteenth century had been interested in psychology and often identified themselves with psychology in their writings. One development in the United States in the nineteenth century was the emergence of Christian Science as a religion, based on the writings of Mary Baker Eddy (1821–1910). She had experienced improvement in her own state of illness in the hands of the healer Phineas P. Quimby (1802–1866) (Gauld, 1992).

Toward the end of the nineteenth century a coalescence began of the traditional “alienists” or psychiatrists, who worked mostly in isolated mental hospital settings, and physicians who were neurologists (or “nerve specialists”). The latter had an outpatient, office clientele.

Late in the nineteenth and early in the twentieth century, a field of “abnormal psychology” emerged in France and was continued

in the United States. This was the result of the work of physicians, physiologists, philosophers, and psychologists including Theodule Ribot (1839–1916), Pierre Janet (1859–1947), William James (1842–1910), Granville Stanley Hall (1844–1924), and Morton Prince (1854–1929). Much of the research done by these workers can be considered to represent a nonpsychoanalytic offshoot of some of Charcot's work with hypnosis and hysteria. Ribot, who was trained as a physiologist, became chair of the department of experimental psychology at the College de France, where he specialized in abnormal psychology. Ribot was among those who influenced Charcot. Charcot's pupil Janet, later a professor at the College de France, interpreted neurotic complaints such as hysteria in terms of his concepts of dissociation and fixed ideas. Janet wrote an influential book on the psychological treatment of hysteria. William James, the author of the classic textbook on psychology in 1890, also lectured on abnormal psychology (Taylor, 1982).

Historian Eugene Taylor (personal communications, January 13–14, 1998) mentions several MD-Ph.D.s as being among those who "defined the interface between psychology and psychiatry" in this era and subsequently, Boris Sidis, Hugo Munsterberg, and Pierre Janet. According to Taylor, there were already clinics in France, Germany, the Netherlands, and the United States by the 1890s devoted to "medico-psychological" treatment. Boris Sidis did psychotherapy with adult outpatients at the Massachusetts General Hospital beginning in 1885. Caplan and Caplan (1969, pp. 304–305) considered Boris Sidis to have been the first psychologist to speak to the American Medico-Psychological Association. In this 1900 address, his opening words were:

Medical men and especially alienists seem to observe less strictly the law "not to admit an alien into the congregation of the Lord," and are willing to listen even to a psychologist. (Caplan, 1969, p. 305)

At the time, Sidis held the position of associate in psychology and psychopathology at the Pathological Institute of the New York State Hospital. Thus, there is justice to Taylor's claim that these developments constitute some of the roots of clinical psychology.

The above developments were independent of the work of Lightner Witmer, a Wundt Ph.D., who founded the first psychology clinic at the University of Pennsylvania in 1896 and began to train a whole generation of Ph.D. clinical psychologists there in Philadelphia. He emphasized the role of clinical psychologists in helping

others rather than only studying them (McReynolds, 1997; Routh, 1996). Unlike the members of the Boston school, Witmer took his cues from Itard, Seguin, and Montessori rather than from Charcot and Janet. He emphasized work with children rather than adults and focused on academic and cognitive problems more than emotional ones.

### **1.01.9 THE TWENTIETH CENTURY: GUILD-RELATED ISSUES**

The twentieth century has been a time of diversification in the number of professional groups providing services to the public in the area of mental health. These include psychiatry, psychoanalysis, psychology, social work, and newer groups such as marriage and family therapy. It has also been a time of impressive scientific advances in the study of psychopathology, assessment, and treatment. The scientific advances seem to cut across these disciplines and therefore can be discussed in a unified way. The professional developments of the century seem to require separate treatment for each competing guild. Let us therefore discuss each profession separately before proceeding to an account of scientific developments.

#### **1.01.9.1 Psychiatry**

Psychiatry began the century with most of its practitioners working in institutions. After it began to take over work from neurologists, its outpatient practice steadily increased. Neurology was a primary care specialty in the United States up until World War II but after that withdrew from such activities to become a field of consulting specialists, researchers, and teachers (Bluestein, 1992).

Philanthropist Henry Phipps was encouraged by psychiatrist Adolf Meyer to endow a psychiatric clinic at Johns Hopkins University. It was the first university psychiatric clinic of its kind in the United States and opened in 1913. In England psychiatrist Henry Maudsley (1835–1918) in 1907 offered funds to the London Council to establish a teaching hospital. Completed in 1915, the Maudsley Hospital ultimately became the locale of the Institute of Psychiatry at the University of London. In 1917 Emil Kraepelin founded the German Research Institute for Psychiatry in Munich. Shorter (1997, p. 262) calls this "the first comprehensive center for investigating brain, mind, and their disorders." Kraepelin's organization was later made a Kaiser Wilhelm Institute by the German Empire. It subsequently received funds from the Rockefeller Foundation and today is one of the Max Planck Institutes supported by the

German government. The Rockefeller Foundation began funding liaison psychiatry at the Massachusetts General Hospital and about 10 other hospitals beginning in 1925.

In 1934, the American Board of Psychiatry and Neurology was established and began to grant “board certification” in psychiatry to those who passed its examination. This provided to psychiatrists greater parity with their colleagues in other medical specialties who had long since achieved the possibility of “board status.”

Psychiatry was especially influenced by psychoanalysis during and after World War II. The first psychiatrist to attain the rank of General in the US Army was William C. Menninger, who had a psychoanalytic orientation. According to Shorter (1977, p. 307), by 1945 most chairs of departments of psychiatry in US medical schools were psychoanalysts. In 1946, an organization called the Group for the Advancement of Psychiatry (GAP) was founded within the American Psychiatric Association, under the leadership of Menninger. This group favored psychoanalytic approaches. By the mid-1970s, very few American departments of psychiatry selected psychoanalysts as chairs. The instruction of psychiatrists in intensive psychotherapy was also on the wane. According to some observers, an era of biological psychiatry and psychopharmacology had begun.

Of course psychiatry is anything but a monolithic field and accommodates many viewpoints and approaches to treatment. For example, psychiatrist Victor Frankl was among those incarcerated in a Nazi concentration camp during World War II and believed that he had been able to survive the experience because he was able to find some meaning in life (Frankl, 1962). Accordingly, he developed a treatment approach he called Logotherapy aimed at helping his patients to find meaning in their own lives.

The “antipsychiatry” movement is also worth mentioning in discussing the history of twentieth century psychiatry as a profession. Psychiatrist and psychoanalyst Ronald D. Laing came to voice strong criticism of both psychiatry and of the families of persons with schizophrenia (Laing, 1960). His view was that parents tended to tie their children in mental knots and threaten their identity. Laing thought of schizophrenia as being in many cases not so much a breakdown as a breakthrough, a necessary stage on the path of development. In 1967 Laing reviewed Michel Foucault’s book, *Madness and civilization* (Foucault, 1961) in the *New Statesman* and thereafter considered Foucault to be a sort of ally. Foucault spoke of the construction of so many public mental hospitals after the

French revolution as “the great confinement” and tended to view this as a sort of conspiracy by psychiatry against human freedom. Of course, at this time, Foucault was hardly alone in being critical of residential treatment for psychopathology. This type of criticism, together with the existing disillusionment with institutions, and the availability of effective neuroleptic medications, hastened the general trend toward deinstitutionalization. By this time most psychiatrists had established an outpatient practice in any case.

### 1.01.9.2 Psychoanalysis

Psychoanalysis virtually swamped the field of psychotherapy early in the century, relying on the treatment principles of free association and interpretation by the analyst. Freud’s writings attracted many disciples. Also, outpatients were willing to pay for this kind of treatment for periods of months and years, if so advised. Psychoanalysis soon began organizing itself in a formal way. The first international psychoanalytic congress was held in Salzburg in 1908. Freud and Jung were invited to the United States in 1909 to speak at Clark University. Psychoanalysis also soon began a process of fission, with Alfred Adler (1870–1937) breaking away in 1911. Then in 1913 Carl Jung (1875–1961) left the fold. In that same year, Freud and certain colleagues set up a small group called “the Committee” to maintain more oversight of psychoanalytic orthodoxy. Interestingly, each of its offshoots seemed to follow Freud’s pattern of elaborating a theory of psychopathology and treatment and establishing formal organizations that its adherents could join.

Freud thought of psychoanalysis as an independent field, separate from psychiatry and medicine, and was willing to train analysts (including psychologists) who lacked a medical background. The American Psychoanalytic Association, founded in 1911, had ideas different from Freud’s about the relation of psychoanalysis to medical practice and after 1938 for 50 years restricted its accredited institutes to offering training to medical candidates only. In 1926, on the other side of the Atlantic, psychologist Theodore Reik had been tried for practicing medicine without a license, that is, for doing psychoanalysis. Reik was acquitted. In fact, Freud himself came to Reik’s defense (S. Freud, 1927). In 1985 Bryant Welch (a psychologist–attorney) and three psychologist colleagues filed a lawsuit against the American Psychoanalytic Association for this practice as a violation of federal antitrust law. In 1988 the organization settled the suit out of

court and began allowing social workers and psychologists to become trainees. According to Shorter (1997, p. 310), the number of non-medical candidates in training soon rose to above 20%.

In France, psychoanalysis emerged as a highly influential approach, but a number of years after it had done so in the United States (Roudinesco, 1990). Psychoanalysis in France began with the work of Marie Bonaparte and her colleagues in the 1920s (Bertin, 1982). This included the founding of an institute and a professional journal as well as the translation of some of Freud's writings into French. Psychiatrist and psychoanalyst Jacques Lacan (1901–1981) was active on the French psychoanalytic scene beginning in the 1930s but began his influential seminars in Paris only in 1953. The legal issue of lay vs. medical analysis also arose there. After the student disturbances of 1968, psychoanalysis assumed a greater representation in French university curricula. In 1973 Lacan published a book on what he considered the four fundamental concepts of psychoanalysis. Lacan's "deconstructionist" approach was popularized in literary circles by Jacques Derrida. Even today, if one examines the shelves of a bookstore in Paris, the section on psychoanalysis is highly visible, but there may be no sections labeled psychology or psychiatry.

The influence of psychoanalysis in Britain and the United States had been evident well before that in France. The Rockefeller Foundation provided some funding for the Chicago Psychoanalytic Institute in 1925. Franz Alexander (1891–1964) was appointed professor of psychoanalysis at the University of Chicago in 1931, the first such academic recognition for the field (Mora, 1994). The role of psychoanalysis in the United States and Britain increased during the 1930s, with the immigration of many analysts from continental Europe fleeing Hitler. As the American interest in psychoanalysis cooled somewhat in the 1970s, Roy Schafer (1976) and others began to reconsider whether psychoanalysis ought to model itself after the natural sciences. Perhaps, Schafer suggested, it would be more appropriate to consider psychoanalysis to be part of the humanities. Its use of hermeneutic approaches in interpreting patients' utterances were seen as similar to those of literary scholars in interpreting poems and fictional narratives. The philosopher of science Adolph Grunbaum (1984, 1993), an outsider to psychoanalysis, criticized its use of a patient's subsequent behavior as a way of confirming the correctness of psychoanalytic interpretations. Other criticisms came from insiders, such as psychoanalyst–historian J. M. Masson (1984). Nevertheless, psychoanalysis continues as a

viable profession and as an influence on scholars over much of the world. Its influence continues to spread in Latin America and elsewhere during the late twentieth century.

### 1.01.9.3 Clinical Psychology

The Boston physician Morton Prince viewed abnormal psychology as a subject belonging more to philosophy or the liberal arts than to medical school. He was influenced by Pierre Janet and William James and published a book on "Christine Beauchamp," a famous case of multiple personality disorder he had encountered. Prince founded the *Journal of Abnormal Psychology* in 1906 and later gave it to the American Psychological Association. He also founded the Harvard Psychological Clinic in 1926. It was a research facility rather than one for providing services to the public and later received funding from the Rockefeller Foundation. Another of Prince's projects was the American Psychopathological Association, open to membership for both psychiatrists and psychologists. If Prince had had his way, the fields of psychology and psychiatry would probably be more unified than they have turned out to be. His aims have been fulfilled much more in the domain of research than that of practice.

Eleven years after his clinic had been established, Lightner Witmer founded a journal called the *Psychological Clinic* in 1907 and in its inaugural issue elaborated on his concept of what the field should involve. This was the first known use of the term clinical psychology. Witmer's journal continued to publish about 23 volumes, well into the 1930s, mostly case histories of children treated in his own clinic at the University of Pennsylvania. Witmer's idea of clinical psychology focused on intervention but resembled what we now think of as special education more than psychotherapy.

According to Eugene Taylor (personal communication, January 13–14, 1998), the first official Ph.D. clinical psychologist at the Massachusetts General Hospital was L. Eugene Emerson, who as early as 1911 was using a type of psychotherapy derived from the work of Freud and Jung. Another Ph.D. psychologist, F. Lyman Wells (1884–1964), was doing psychotherapy with inpatients at the McLean Hospital at the same time. The theories of Freud, Jung, Adler, and other psychodynamic views also influenced academic psychology, especially in the clinical area but also in the areas of personality, developmental, and social psychology.

James McKeen Cattell (Ph.D. Leipzig, 1886) was one of the strongest supporters of the idea



of psychology as a profession but did not have much interest in clinical issues. He founded the Psychological Corporation in 1921 as a non-profit organization to promote applications in psychology, mostly psychometric testing. His interests soon turned to science at large. He bought *Science* magazine from Alexander Graham Bell, and served as its editor for many years. Cattell also published *American Men of Science* (later known as *American Men and Women of Science*) and inaugurated the practice of highlighting the relative merit of scientists' contributions by putting a star beside the names of those considered the most notable. In this way he carried forward Galton's idea of the importance of individual differences.

In the era before World War II, clinical psychologists were known more as mental testers than as psychotherapists. The mainstay of their early activities in this area was the Binet test, first published in France in 1905, translated into English in 1908, and eventually standardized in the United States. Psychologist Henry Goddard, who first had the Binet translated, was the research director at the Vineland Training School in New Jersey (an institution for persons with mental retardation). He set up the first internship program in clinical psychology in that same year, 1908. Internships involving a year of full-time supervised experience soon became a part of the expected training of clinical psychologists. Goddard's research was supported financially by Samuel S. Fels, the soap maker, and some friends from the Ethical Culture Society of Philadelphia. Fels and Goddard were part of an informal group that met at Vineland for some years that called itself humorously, "the Feeble Minded Club" (Doll, 1988).

In 1917, the American Association of Clinical Psychologists was founded. It was the first organization in the field of clinical psychology and came about because its founder J. E. W. Wallin (1876–1969) wished to begin to establish some professional standards. In particular, he was concerned that Binet testing was being done by school teachers with no background in psychology on the one hand, and by academically trained psychologists with no clinical experience on the other. Only two years later, this organization disbanded itself in favor of a new Clinical Section within the American Psychological Association. For a brief time, the American Psychological Association tried to develop a procedure for certifying what were at the time called "consulting psychologists," meaning those who were adequately prepared to offer their services to the public. The person who was in charge of this effort was F. Lyman Wells, already mentioned as an influential figure

in early clinical psychology. The APA's efforts in this domain did not succeed (only 25 persons were ever so certified) and were discontinued in 1927.

Psychologist Leta Stetter Hollingworth (1886–1939) published a little-cited paper in 1918 that was the first to mention the possibility of a different type of degree for professional psychologists, the "doctor of psychology" instead of the more usual Ph.D. (doctor of philosophy). The Psy.D. was discussed as a possibility for many years but did not come into much actual use until the 1960s. One of the first programs that granted the Psy.D. began in 1968 at the University of Illinois. In the United States, Britain, and some other European countries, the Psy.D. and other similar degrees such as the "doctor of clinical psychology" (D.Clin.Psy.) are now more often offered by training programs for practitioners. Research-oriented graduate programs still give the Ph.D. in clinical psychology.

By the time of World War I, self-report questionnaires began to be developed for the assessment of personality and psychopathology. By the 1930s, projective tests such as the Rorschach inkblot test and the Thematic Apperception Test joined what was becoming the clinical psychologist's standard test battery.

In 1937 the American Association of Applied Psychologists (AAAP) was founded. The Clinical Section within the American Psychological Association disbanded itself, and its members formed the Clinical Section of the AAAP. In 1945 Division 12 of the American Psychological Association was founded, the present Division of Clinical Psychology. It was based on a merger of two groups: one of these was the psychologists from the Clinical Section of the AAAP, who in a sense represented the tradition established by Lightner Witmer of Philadelphia. The other group briefly in 1945 carried the label of the Division of Abnormal Psychology and Psychotherapy and was to have been APA Division 11. This group could perhaps be seen as the descendants of the Boston school of abnormal psychology. Division 11 was never officially established; instead its potential members became part of Division 12, which was initially called the Division of Abnormal and Clinical Psychology.

In 1945, the American Psychological Association appointed David Shakow (1901–1981) to head a committee to discuss how clinical psychologists should be trained. The impetus for this committee's work was provided by the promise of massive funding of such training in all mental health fields by the Veteran's Administration (VA) and the new National Institute of Mental Health (NIMH). James Grier Miller who had been trained in both psychology and psychiatry was chief of the VA

Division of Clinical Psychology and Neuropsychiatry at the time. The Shakow Committee's recommendations were later endorsed by the so-called Boulder Conference in 1949. Shakow's model called for training clinical psychologists at the Ph.D. level as "scientist-practitioners," including a required one-year predoctoral internship, usually obtained during the trainee's third year of graduate school. As Levy (1962) later pointed out, many psychologists trained as "scientist-practitioners" never went on to publish any research; many did not even publish their Ph.D. dissertations. Thus, this training model did not seem to function as well as had been hoped. As time went on, more of the graduates of such programs also went into private practice, an outcome that no doubt would have horrified David Shakow and his colleagues.

The first state law certifying psychologists for independent practice was passed in Connecticut in 1945 (Heiser, 1945), and by 1977 all states had such certification or licensing laws. The distinction between these two types of legislation is that certification usually protects only the title (in this case "psychology"), whereas licensing is more likely to specify the actual content of the practice. Similar legislation was passed in each of the Canadian provinces. It is the goal of most national psychological associations in European countries to achieve full legal recognition for professional psychology, if indeed they had not already attained it (McPherson, 1986).

In 1947, the American Board of Examiners in Professional Psychology (ABEPP, later simply ABPP after the word "Examiners" was dropped from the Board's name) was founded. This board soon began to grant diplomas in clinical psychology and was modeled after boards that were well established in various medical fields including psychiatry. In 1948, Nicholas Hobbs (1915–1983) carried out a project initiated by the American Psychological Association to develop an explicit set of ethical standards for psychologists. Revisions of these ethical principles continue up to the present.

In London after World War II, psychologist Hans J. Eysenck set up a Department of Psychology within the Institute of Psychiatry at the University of London. This department became a major center for behavior therapy in Britain in subsequent decades.

In his 1959 book, psychologist Alan Ross (1921–1993) noted the fact that after the end of World War II, clinical psychology had moved far from some of its origins focusing on work with children. This was partly because of all the government funds that encouraged work with adults such as military veterans. Ross wished to reclaim what he viewed as the original heritage

of the field. He stimulated the formation of a new subgroup of persons who identified themselves as clinical child psychologists. Psychologist Nicholas Hobbs was one of these and was particularly effective in suggesting how support and treatment could be arranged for disturbed children, coordinating work with them individually, with their families, and with the schools (Hobbs, 1966).

In 1962, a conference on behavior modification was held in Charlottesville, Virginia. It was instigated by psychiatrist Joseph Wolpe, behavior therapist Andrew Salter, and psychologist Leo Reyna, who published the proceedings as a book (Wolpe, Salter, & Reyna, 1964). The American Association of Behavior Therapies (AABT) was founded relatively soon after this landmark conference, in 1966 (in 1969 the "Therapies" in the organization's name was changed to "Therapy"). Despite the interdisciplinary origins of this field, most behavior therapists were psychologists. Until 1972 the AABT met with the American Psychological Association; after that, it met independently.

Like other professional groups (including psychoanalysts), behaviorally-oriented practitioners showed some tendency toward fission. Applied behavior analysis since its beginning tended to isolate itself from behavior therapy with separate professional organizations, journals, and so on (Krantz, 1971). This branch of behavioral psychology dates its own history from a 1947 research conference in Bloomington, Indiana under the aegis of B. F. Skinner. Unlike other behaviorists, the followers of Skinner avoided using dispositional concepts, preferred single-subject experiments to those with group designs, and emphasized the "three-term contingency" involving setting events, response, and environmental consequences.

In 1963, a new component of the National Institutes of Health was created, called the National Institute of Child Health and Human Development (for which the usual acronym is NICHD). Part of the impetus for this new research institute was the interest of President John F. Kennedy and his family in the problem of mental retardation. Research grants related to this problem were shifted from the NIMH to the NICHD, which also had the practical effect of divorcing psychiatry from the medical aspects of mental retardation, which were henceforth considered to be more in the domain of the pediatrician. The creation of this agency also propelled psychology into the role of being the primary discipline concerned with psychopathology associated with mental retardation.

The California School of Professional Psychology was founded in 1969, with multiple campuses not affiliated with any university. The

founder was psychologist Nicholas Cummings, who had the backing of the state psychological association in California. From the standpoint of those who founded this and other free-standing schools of professional psychology that soon grew up all over the United States, the existing graduate schools' programs were too elitist and not sufficiently concerned about producing practitioners. A conference of psychologists at Vail, Colorado in 1973 urged the acceptance of a practitioner model of training alongside the now traditional scientist-practitioner model (Korman, 1974). The Vail recommendations also endorsed the trend toward schools of professional psychology.

With the growing dominance of the US health-care system by insurance and HMOs, psychologists were having trouble being reimbursed by third parties for their professional services. The insurance companies complained about the fact that psychology licensing was usually generic and did not distinguish between those qualified to deliver "health care services" and others (such as industrial psychologists) who might not have such training. For this reason, in 1975 psychologists established a National Register of Health Service Providers in Psychology. Each psychologist on this register had to have not only a valid state license but also training in providing health services, such as a formal clinical internship.

In 1988 the American Psychological Society (APS), consisting mostly of research and academic psychologists, split off from the American Psychological Association, which was increasingly dominated by professional practitioners. Many research and academic clinical psychologists felt torn in their loyalties between these two competing organizations.

#### 1.01.9.4 Social Work

In 1917, the first National Conference on Social Work was held in the United States. In 1917 Mary Richmond (1861–1928), a prominent figure in social work, published a book, *Social diagnosis*, which attempted to define the nature of social case work. The existing 15 schools of social work in the United States joined a new association that was established in 1919. At the time, nine of them were linked to universities and the other six to operating social agencies instead, which resembled the long-standing pattern for hospital-based schools of nursing (Leiby, 1978). Schools of social work agreed among themselves in 1939 that the standard educational pattern in the field would be two years of graduate study, resulting in a Master's degree in Social Work (MSW).

By 1945, an organization of social workers in the United States already had 2000 members. In 1955, the National Association of Social Workers (NASW) was established as a combination of several groups. According to Shorter (1997, p. 293), the NASW increased to 80 000 members by 1990, about one-quarter of whom were in private practice.

#### 1.01.9.5 Religious Counseling

Many mental health workers have tended to distance themselves from religion, although they might admit, if pressed, that their work was not unrelated to some of the counseling done by members of the clergy or of religious orders. One twentieth century exception to the attempt to keep mental health and religion separate was the Emmanuel Movement. The Rev. Elwood Worcester (1863–1940), who had a philosophy Ph.D. from Leipzig in 1889, in 1904 became rector of the Emmanuel Church in Boston. In 1905, he and his colleagues attempted to integrate a mental health clinic into the activities of this church, attracting wide publicity in the press. The medical community of Boston reacted somewhat adversely and uncooperatively. However, historian Eugene Taylor notes that the Emmanuel Movement was but "chapter one" of the Clinical Pastoral Education Movement.

Much more recently, science-fiction writer L. Ron Hubbard (1950) launched "dianetics" as an alternative to traditional psychotherapies. In some respects it seems to have been modeled after psychoanalysis. Hubbard went on in 1954 to establish Scientology as a church, at least in terms of its legal status. Shorter (1997) points out that Hubbard opposed ECT and that he appointed Thomas Szasz as Scientology's "consulting psychiatrist." Scientologists mounted campaigns against the use of the drug Ritalin for children with attention deficit disorder. They are equally against the use of Prozac.

#### 1.01.9.6 Interdisciplinary Activities and Advocacy

Mental health professionals cannot justify their existence simply as a way to make a living. They must demonstrate that their activities benefit their patients and society at large. One of the earliest effective consumer advocates was Clifford Whittington Beers (1876–1943), who published the book, *A mind that found itself* in 1908, about his own experiences as a mental patient. A graduate of Yale, Beers had a mental breakdown at age 24 in which he attempted suicide and was hospitalized at the Hartford Retreat. Subsequently, he experienced a manic

episode and was an inmate at the Connecticut State Hospital. The book he wrote later described these experiences, not without some bitterness about the way he had been treated at the time, and suggested the need for better practices in this field. His advocacy was endorsed by such influential figures as William James at Harvard and Adolf Meyer at Johns Hopkins, and in 1909 Beers became one of the founders of the National Committee on Mental Hygiene, an organization that still exists. The Rockefeller Brothers supplied a grant to assist the fledgling movement.

A child guidance clinic, attached to the local juvenile court, was a new innovation begun in Chicago in 1908 by physician William Healy (1869–1963) and psychologist Grace Fernald (1879–1950). The Commonwealth Fund (established by the Harkness family) in 1919 made a grant to the National Committee on Mental Hygiene that supported the founding of a series of child guidance clinics around the United States and in other countries. The Commonwealth Fund also provided support to several child guidance clinics in England beginning in the 1920s (Wooldridge, 1994). The typical staffing of a child guidance clinic was an interdisciplinary team consisting of a psychiatrist, psychologist, and social worker. The usual role of the psychiatrist was diagnostic interviewing and, when feasible, psychotherapy. That of the psychologist was mental testing, and that of the social worker was dealing with family members and other community agencies.

As an outgrowth of the child guidance movement in the United States, a new organization was formed in 1924 called the American Orthopsychiatric Association. The Greek word “ortho” means straight, and juvenile courts and child guidance clinics were intended to help youth “go straight” and avoid crime. The first president, not surprisingly, was William Healy. Membership was at first restricted to psychiatrists but in 1926 was broadened to include all mental health professionals. For many years “Ortho” has served as an interdisciplinary meeting place for practicing psychiatrists, psychologists, and social workers. It has also served as an organization advocating for the needs of those, especially the disadvantaged, who were in need of mental health services.

After World War II, the National Institute of Mental Health in the United States, created in 1946 and up and running in 1949, began a program of training grants for all mental health fields (Rubinstein, 1992) that lasted into the Reagan era of the 1980s (Benjamin, 1986). These funds were responsible for huge increases in the number of clinical psychologists and psychiatrists who were trained. Similar in-

creases in funding were also seen in Britain, the Scandinavian countries, and elsewhere in Western Europe as a part of various national health systems. The success of the Soviet Union in putting the satellite Sputnik in orbit in 1957 spurred the US government to even greater efforts in funding research and training in all scientific fields.

In London in 1947, the National Society for Mentally Handicapped Children was founded. It was the first of many groups founded after World War II in various countries primarily by parents of children with mental retardation, cerebral palsy, and many other handicapping conditions. These groups proved to be quite effective in advocating that services be provided for their children, particularly in the area of education. They also advocated that research be funded, including research on prevention. One of the most effective of the parent-advocates for persons with mental retardation was Elizabeth M. Boggs (1915–1996). Besides being the parent of a handicapped son, she was a Cambridge-trained Ph.D. in chemistry and readily trained herself to follow the intricacies of government legislation related to services and research. She was therefore highly effective as a lobbyist for several decades.

In 1963 the Kennedy administration sponsored a bill to establish a nationwide network of interdisciplinary Community Mental Health Centers (CMHCs), which indeed came into being and still exists. The federal government did not plan to continue the funding of these centers indefinitely and relatively quickly shifted their support to states and local communities. It is by now widely acknowledged that these centers were not able to provide as much community support as was hoped for by sponsors of the legislation. Many of the mental patients being discharged from mental hospitals around the country were thus thrown back on their own resources and those of their families. The administration of a CMHC can be in the hands of persons trained in any of several different mental health-related disciplines.

The “antipsychiatry” movement that began in the 1960s was critical not only of psychiatry proper but of the mental health establishment in general. The novel by Kesey (1962), *One flew over the cuckoo’s nest*, criticized mental hospital treatment, focusing specifically on ECT and lobotomy. The psychiatric nurse was portrayed simply as part of an oppressive hospital team. The novel was made into a movie in 1975, which no doubt reached an even larger audience. At about the same time, sociologist Erving Goffman published his influential critique of the mental hospital as a “total institution” in which patients suffered numerous abasements,

degradations, humiliations, and profanations (Goffman, 1961). Goffman's book was based on field work at St. Elizabeth's Hospital, a government-supported institution in Washington, DC at a time when about 6000 mental patients were housed there.

In 1968 Bengt Nirje, director of the Swedish Parents Association for Mentally Retarded Children, developed the concept of normalization, which was first applied in the field of mental retardation. The idea meant that the goal of the service system is to assist the individual to live in as normal a way as possible, in spite of the presence of any handicapping condition: living at home, going to an ordinary school, having some kind of job or responsible role in the household, shopping at regular stores in the community, being treated by the usual primary physicians, etc. Along with this concept went the idea of "self-advocacy" that individuals with physical or mental disabilities should themselves try to better their situation and not wait for some other group such as parents or professionals to do this for them (Shapiro, 1993).

Advocacy is not just the business of parent groups or of those with mental problems themselves. It is also the business of lawyers, and the mental health bar became very active in the latter half of the twentieth century. One important case was the suit of Wyatt v. Stickney, originating in Alabama in 1971. In this case, the court declared that mental patients had a right to treatment, and that they could not be confined in a state mental hospital without proper treatment for their condition. One side effect of such cases was simply to speed up the course of de-institutionalization. This unfortunately meant that many mental patients not only did not get much treatment, they were sometimes released and had to fend for themselves as best they could, on the streets (Isaac & Armat, 1990).

Behaviorally-oriented token economies also sometimes ran into legal trouble. In a token economy, points are assigned to patients or clients contingent on certain predefined appropriate behaviors, and these points (or tokens) can be exchanged for preferred items or privileges. Kazdin (1978) describes one such situation in a Missouri program sponsored by the Federal Bureau of Prisons in 1972:

[This project] was terminated because of pending litigation that challenged the involuntary assignment of prisoners to the program without due process, deprivation of privileges normally provided in prisons, aversive conditions used as part of treatment, and other aspects of contingencies. (p. 292)

In a related US Supreme Court decision on the case of Doe v. Bolton (1973), Justice William O. Douglas spoke at length about the right of an individual to be let alone, plan one's own affairs, shape one's own life, do what one pleases, be free from bodily restraint or compulsion, and be free to work, stroll, or loaf. Such words bring back memories of the Bicetre Hospital in Paris in 1789.

In 1975, federal legislation was passed in the United States guaranteeing all children a free appropriate education in the public schools, regardless of handicapping condition. This law was of tremendous importance especially to parents whose handicapped children had been excluded from the public schools or who had not been provided with educational accommodations to their special needs. As an unanticipated side effect, this legislation fueled a tremendous amount of attention to assessing children to determine their eligibility for special education programs (Milofsky, 1989). Also, such special programs at times could seem very expensive to school boards, giving them reason to set up procedural barriers against such placements. This law was both a meal ticket and a major source of frustration to clinicians working with children and to those working within the schools. These laws were revised as recently as 1997 under the heading of the Individuals with Disabilities Education Act, which had much more emphasis on mainstreaming and inclusion. It is too early to see what effects and side effects these changes will have.

The National Alliance for the Mentally Ill (NAMI) was founded in 1979 as a Washington-based national advocacy group, consisting largely of the parents and friends of people with major mental illness such as schizophrenia, depression, or bipolar disorder. As of 1996 it had about 130 000 members. The NAMI view of mental illness as a type of "no fault brain disorder" successfully influenced Congress and the NIMH to give greater emphasis to biologically-oriented research in psychiatry and psychology.

#### **1.01.10 RESEARCH ON PSYCHOPATHOLOGY IN THE TWENTIETH CENTURY**

##### **1.01.10.1 General Paralysis of the Insane**

In 1913, bacteriologist Hideo Noguchi (1876–1928), working at the Rockefeller Institute, discovered *Treponema pallidum* (the organism causing syphilis) in the brains of paretics and patients with tabes dorsalis,

confirming his hypothesis concerning the cause of the disturbance (Clark, 1959). While such discoveries were welcomed, they did have the effect of making the diagnosis and treatment of syphilis and of general paralysis of the insane (GPI) the province of internal medicine rather than that of psychiatry. The psychosocial aspects of syphilis, like those of other sexually transmitted diseases including the AIDS virus, remain of interest to the behavioral sciences under the new guises of behavioral medicine and health psychology.

### 1.01.10.2 Dementia

In 1901 the physician Alois Alzheimer, working at a Frankfurt hospital, encountered a patient with a dementing condition who upon autopsy had characteristic tangles and neuritic plaques in the brain. This condition is now known to us as Alzheimer's disease (O'Brien, 1996). According to Shorter (1997, p. 103), in 1995 a gene was discovered on chromosome 14 and another one on chromosome 21 that were linked to Alzheimer's disease.

The current focus within the behavioral sciences is probably more on the difficult situation of family caregivers of Alzheimers patients than on the dementing condition itself. There is a high stress situation, and much remains to be discovered about how to offer optimal support to them.

### 1.01.10.3 Dementia Praecox/Schizophrenia

The eminent German jurist Daniel Schreber (1903) wrote an autobiographical account of his nervous illness which has usually been thought to be schizophrenia. Schreber's story was used by Freud (1911/1959) to outline his concepts of the role of homosexuality in the genesis of paranoia (with statements such as "I love him" being transformed mentally into their opposites, such as "he hates me"). Shorter, a biologically oriented historian of psychiatry, reinterpreted the evidence to argue that Schreber may have actually suffered from neurosyphilis (Shorter, 1997, p. 80).

The name schizophrenia was suggested as a substitute for dementia praecox by Eugen Bleuler (1908, 1911), who was professor of psychiatry at Zurich and director of the Burgholzi hospital there. In contrast to Kraepelin, Bleuler preferred to define this group of disorders in terms of their symptoms rather than relying so much on the chronic course of the psychosis. He listed the core symptoms as including loose associations, flat affect, autistic

thinking, and ambivalence, with the symptom of loose associations being the most influential concept. Psychiatrist Carl Jung was originally trained by Bleuler at the Burgholzi and early in his career participated in research on word associations in schizophrenia.

Kurt Schneider (1887–1967), a professor of psychiatry at Heidelberg, came up with an influential list of "first rank symptoms" which he considered to be characteristic of schizophrenia. An example of such a symptom would be mentally hearing one's voice spoken aloud.

One of the first genetic studies of schizophrenia was carried out by Hans Luxenburger in 1928. He was a psychiatrist who worked at Kraepelin's Institute at Munich. He searched through 16000 birth records in Bavaria and compared them with a patient list, finding 211 patients who were part of a twin pair. Of those who were monozygotic, the concordance rate for schizophrenia was higher than that for dizygotic twins (Shorter, 1997, p. 241).

In contrast to this approach based on genetics, the German refugee psychoanalyst Frieda Fromm-Reichman (1899–1957) emphasized the role of experiences provided the developing child by the "schizophrenogenic mother." Fromm-Reichman was noteworthy for her skill as a psychotherapist for patients with schizophrenia. She began her work at Chestnut Lodge in Maryland in 1935 and was portrayed in the novel *I never promised you a rose garden* as "Dr. Fried." She was part of a group of neo-Freudians in the United States that included psychiatrist Harry Stack Sullivan (1892–1949).

Psychiatrist Franz J. Kallmann (1897–1966), who was trained at Kraepelin's Institute, fled Hitler in 1935 to work at the New York State Psychiatric Institute, associated with Columbia University. He continued his genetic research, finding higher concordance rates for schizophrenia in monozygotic than dizygotic twins, with the corresponding percentages being approximately 85% vs. 15% (Kallmann, 1938, 1946, 1953).

Seymour S. Kety and his associates were involved in a Danish adoption study that was more refined in its methods than previous research, in part because of the quality of public record keeping in Denmark. For Copenhagen adoptees with schizophrenia, this disorder was found to be 10 times more common in adoptees' biological relatives than in their adoptive families (Kety et al., 1968). These findings were essentially replicated in the rest of Denmark (Kety, Wender, Jacpben, Ingraham, et al., 1994).

Psychologist Paul Meehl (1962) presented an integrative theory of how schizophrenia might

come about, under the title of “schizotaxia, schizotypy, and schizophrenia.” Schizotaxia refers to the hypothetical genetic predisposition that is considered necessary but not sufficient to produce the disorder. Schizotypy is the behavioral phenotype produced by this genotype—not necessarily abnormal, but a condition of vulnerability to the disorder. Perhaps schizotypes find interpersonal relationships a bit more aversive than others do, or manifest some subclinical cognitive slippage on occasion, but nothing symptomatic. Full blown schizophrenia is what is seen when a schizotype is subjected to certain stressful physical or psychological conditions.

Psychiatrist Manfred Bleuler, the son of Eugen Bleuler (who had coined the term “schizophrenia”), was like his father a professor in Zurich. M. Bleuler published a study of 208 patients with schizophrenia whom he had followed up for over 20 years (M. Bleuler, 1972). This study confirmed the belief that the disorder was quite variable in its course and outcome. He was not, however, able to realize his ambition of uncovering a Mendelian etiology for schizophrenia, which continues to be elusive.

Psychologist Philip Holzman and his colleagues showed that patients with schizophrenia and their first-degree relatives were more likely to have abnormal smooth pursuit eye movements than control subjects (Holzman et al., 1973). In this procedure, patients are simply asked to try to follow a moving pendulum with their eyes. The abnormality consists of a jagged pattern of eye tracking rather than a smooth one. The finding has been replicated by other investigators and turns out to be a “trait marker” that does not change when an individual decompensates into psychosis or recovers from schizophrenia. The research provides further support for some kind of genetic hypothesis concerning schizophrenia.

For a time, the “dopamine hypothesis” concerning schizophrenia was a serious scientific contender (see e.g., Snyder, 1976). The appeal of this notion was that virtually all of the effective neuroleptic medications seemed to have in common the property of blocking dopamine receptors in the brain. When other equally effective antipsychotic drugs such as clozapine were discovered that did not block dopamine, the idea lost some of its attractiveness.

#### **1.01.10.4 Schizoaffective Disorder**

This term was coined by Russian psychiatrist Jacob Kasanin in 1933 to describe a condition with symptoms of both schizophrenia and

depression. Its prognosis tended to be intermediate, that is, better than that of schizophrenia but worse than that of depression.

#### **1.01.10.5 Depression**

It was thought at first that phenothiazine medications might be rather specific to schizophrenia rather than depression, supporting the validity of the distinction between these two groups of disorders. However, it turned out that depression also responds well to such medications (Klein & Fink, 1962).

Psychiatrist A. T. Beck was one of the first to elaborate a theory explaining how the way a person thinks could contribute to depression, for example, negative thoughts about oneself, the world, and the future, and cognitive distortions such as making internal, global, and pervasive attributions for negative events (“this happened because of me,” “I am that way about everything,” “that is just the way I am”) (A. T. Beck, 1963).

Psychologist Martin Seligman linked up his research on experimentally produced learned helplessness in dogs with similar phenomena seen in humans and moved into research on helplessness and human depression (Seligman, 1975).

#### **1.01.10.6 Neurosis, Including Anxiety**

Janet and Raymond (1903) in France wrote about obsessions and psychasthenia (which literally translates as “mental weakness”). Janet considered psychasthenia to be characterized by pathological feelings of inadequacy.

One interesting new development was the discovery of the phenomenon of “experimental neurosis.” According to Kazdin (1978), in 1912, Mariya N. Yerofeyeva (1867–1925) was doing research in the St. Petersburg laboratory of Ivan Pavlov (1849–1936), conditioning a dog. As usual, the unconditioned stimulus was food-powder and the unconditioned response was salivation. In this case the conditioned stimulus was a mild electric shock on the dog’s skin, which might be expected to elicit defensive reactions competing with salivation. Still, the conditioning was going well up until the point when the shock was administered to one new location after another on the animal’s skin. When this was done, conditioning broke down, and the animal developed a lasting disturbance of its overall behavior. In 1913 another of Pavlov’s co-workers made a similar discovery. Nataliya R. Shenger-Krestikovnikova (1875–1947) was conditioning a dog to discriminate

between a circle and an ellipse. The dog was able to do so at first, but conditioning broke down when the discrimination became too difficult and was replaced by enduring disturbed behavior (Pavlov, 1927).

John Watson (1878–1958) and Rosalie Raynor (1898–1935) published in 1920 their study of “little Albert” that is by now well known (Watson & Raynor, 1920). The child was repeatedly presented with a furry animal in the presence of a loud noise and evidently learned to fear the animal and other objects similar to it as a result of this experience. The authors presented the study as a demonstration of how phobias might originate. Watson (1913) had presented behaviorism as a “purely objective experimental branch of natural science. Its theoretical goal is the prediction and control of behavior” (Watson, 1913, p. 158).

Behaviorists were not the only ones contributing to the study of the phenomena underlying neurotic behavior in this era. Anna Freud (1895–1982) published a well-received book on *The ego and the mechanisms of defense* in 1937. Neurotic symptoms were interpreted as functioning as defensive maneuvers to ward off anxiety.

The psychoanalyst J. H. Masserman (1943) did experiments with cats, putting them into approach-avoidance conflict situations. This work contributed to the thinking of subsequent behavioral researchers concerned with neurotic behaviors who, however, did not necessarily accept some of Masserman’s psychoanalytic speculations about his findings. Indeed, both psychoanalysis and behaviorism remain for the most part mutually isolated systems of thought to this day.

Howard S. Lidell (1895–1962) at Cornell University in 1956 reported research producing “experimental neurosis” in dogs, sheep, goats, and rabbits. This showed that the phenomenon was not specific to dogs nor to the particular procedures used in Pavlov’s laboratory.

After World War II, the psychoanalytic approach to neurosis took a turn toward “ego psychology,” recognizing the existence of a relatively conflict-free sphere of functioning that was little affected by neurotic disorders (Hartman, Kris, & Loewenstein, 1946).

In 1969 British psychiatrist Isaac M. Marks published an influential article suggesting that there might be important genetic factors in fear and anxiety disorders including agoraphobia, panic disorder, and obsessive-compulsive disorder.

Psychiatrist Donald F. Klein found in his research on psychopharmacology that anxiety patients (unlike those who were depressed) did not get better on chlorpromazine. Panic

disorder, as a condition distinct from depression, also did not respond to chlorpromazine (Klein, 1964; Klein & Fink, 1962).

#### 1.01.10.7 Shell Shock/Post-traumatic Stress Disorder

W. H. R. Rivers (1864–1922), a British physician, experimental psychologist, and anthropologist, became involved in treating the war neuroses referred to as “shell shock” during World War I and soon developed the view that they were psychologically based rather than being simply a mechanical result of combat trauma (Ackerknecht, 1942). This view is preserved in the present concept of post-traumatic stress disorder (PTSD).

#### 1.01.10.8 Stress

Stress is not in itself considered a type of psychopathology but is considered a risk factor not only for psychopathology but also for various types of physical illness. One of the more influential researchers in this field is psychologist Richard S. Lazarus (1966). His writings define stress as a set of demands that exceed the adaptive capacity of the person. It is important to know not only the physical characteristics of the stressful situation but how these are appraised by the individual, and what coping strategies are adopted to try to deal with the stress.

#### 1.01.10.9 Psychosomatic Disease

Physician Flanders Dunbar (1902–1959) wrote a book on the personality factors she considered to underlie ulcers, colitis, asthma, and coronary heart disease (Dunbar, 1938). The theory that certain “psychosomatic” illnesses exist (e.g., the concept of “the ulcer personality”) was subsequently rejected by many. It would be more accurate to say that this notion was broadened into the idea that behavioral and psychosocial factors probably have some importance in most medical conditions. In a later era, Drotar (1981) summarized some of the new approaches taken by “pediatric psychologists” in studying and helping children with chronic medical illness.

#### 1.01.10.10 Anorexia Nervosa and Bulimia

In the 1960s and 1970s there seemed to be a marked increase in the prevalence of self-starvation and other eating disorders, particularly among young women from socially advantaged backgrounds in the United States



and Europe. Scholars unearthed a case history of this kind of anorexia nervosa dating back to the seventeenth century, written by Richard Morton (1637–1698), physician to King James II. An eating disorder known as bulimia nervosa also was characterized during the 1970s, consisting of “binging” episodes in which large quantities of food were eaten, followed by “purging” or self-induced vomiting (Russell, 1979). The abuse of laxatives is also sometimes a part of bulimia.

#### 1.01.10.11 Borderline Personality Disorder

In former times the phrase “borderline” referred to an individual with difficulties resembling schizophrenia but not being quite severe enough to deserve that label. Subsequently, borderline came to refer to a personality trait in which an individual had an impaired sense of identity and problems with the boundaries of self. Suicide threats were observed to be common in this condition. Psychoanalyst Otto Kernberg (1967, 1975) was one of the first influential writers on what he called “borderline personality organization.” In the *Diagnostic and statistical manual of mental disorders* (American Psychiatric Association, 1980), the condition was labeled as borderline personality disorder (BPD).

#### 1.01.10.12 Antisocial Behavior

An early example of basic research on aggressive behavior was that of John Dollard (1900–1980), Leonard Doob, Neal E. Miller, O. H. Mowrer (1907–1982), and R. R. Sears, reported in their 1939 book, *Frustration and aggression*.

On the clinical side, psychiatrist Hervey M. Cleckley published in 1941 an influential book, *The mask of sanity*, trying to define the characteristics of a psychopath. Cleckley felt that one could not reliably diagnose this condition on the basis of an interview because many psychopaths were so skilled at deception. Instead, one had to look at the way the person behaved in life, disregarding rules and exploiting other people.

A classic study on the stability of antisocial behavior from childhood to adulthood was published in 1966 by sociologist Lee Robins under the title, *Deviant children grown up*. She followed up, approximately 30 years later, a cohort of children originally seen for diagnostic purposes in a St. Louis child guidance clinic. A relatively crude classification was made on the basis of the presenting complaint, so that there were those who presented with antisocial

behavior, and others. Also included in the study were subjects who had been children in St. Louis at the same time but were not referred to such a clinic. The follow-up was exceedingly thorough and involved a personal interview using standardized questions and a coding system that later served as a model for the Diagnostic Interview Schedule (DIS) to be used with *DSM-III*. Also, data from the subjects’ military service records, Social Security earnings records, the county sheriff’s office, and many other such databases were accessed. The main finding of the study was that antisocial children tend to become antisocial adults, many of them qualifying for classification as psychopaths (*DSM* Antisocial Personality Disorder) in adult life.

The stability of aggressive behavior from childhood to adulthood was confirmed in a prospective longitudinal study using peer-ratings of third-grade school children as a means of selecting the subjects. This was carried out by psychologist Leonard Eron and his colleagues (e.g., Huesmann et al., 1984).

#### 1.01.10.13 Substance Abuse

Some drugs have a dual history of being introduced as a treatment or as a means of studying psychopathology, and then later to become abused substances. Such was the fate of cocaine, which Freud to his lasting regret at one time recommended to a friend as a helpful treatment. The friend became addicted and died from an overdose (Gay, 1988). A more recent example would be lysergic acid diethylamide (LSD), discovered in 1943 and once considered promising as a method of gaining insight into the nature of psychotic hallucinations. As Shorter (1997, p. 265) comments, LSD had “no clinical payoffs” and became a “street drug of abuse.”

#### 1.01.10.14 Sexual Dysfunction

In 1966, gynecologist W. H. Masters and psychologist Virginia Johnson published their book on human sexual experience, including reports of direct observation of sexual activity and physiological recordings during such events. Their work elucidated aspects of normal sexual response that had previously not been so well understood, for example, female vaginal lubrication during sexual arousal. It also provided new understanding and treatment recommendations for various types of sexual dysfunction, for example, dyspareunia (Masters & Johnson, 1966).

### 1.01.10.15 Mental Retardation

H. H. Goddard (1912) wrote a book about a family he chose to call the Kallikaks (combining the Greek words for good and bad). The pedigrees of two branches of the family of "Martin Kallikak" were presented, one with considerable mental retardation among its members, the other without this pattern. The book has been derided as an example of a lack of rigor in its attempted analysis of genetic patterns ("look and say Mendelism"). The charge was even made that some of the published photographs were altered to reveal their physical stigmata more clearly. It is also possible that the changes in the photographs were made, in all innocence, to correct the fuzziness of some of the pictures (Fancher, 1987).

Eugenic approaches to mental retardation, considered anathema today, were widely endorsed by professionals and the public earlier in this century. In a famous case, *Buck v. Bell* (1927), the US Supreme Court even endorsed the use of involuntary sterilization for this purpose. Oliver Wendell Holmes, the chief justice, was quoted as saying "three generations of imbeciles is enough."

Torsten Sjögren, a Swedish geneticist, established in 1931 that Tay Sachs disease is a Mendelian recessive condition (Sjögren, 1931). This disorder involves blindness, deteriorating mental functioning, and death commonly by three years of age. The British geneticist Lionel S. Penrose (1898–1972) was responsible for summarizing for his readers material on both biometric and molecular genetics as related to mental retardation (Penrose, 1951). Philip Levine (1900–1987) and Alexander S. Wiener (1907–1976) elucidated the mechanisms by which Rh factor incompatibilities in the blood between the mother and fetus can produce fetal death or mental retardation.

Psychologist Edgar A. Doll and his colleagues published research implicating birth injuries as being responsible for a certain proportion of the cases of mental retardation (Doll, Phelps, & Melcher, 1932).

In the 1930s, psychologist Harold M. Skeels (1901–1970) and his colleagues published the well-known "Iowa studies" in which infants from an orphanage experienced large positive changes in IQ after being transferred to an environment where they received more attention and stimulation. These were not controlled studies and elicited strong criticism from Terman and others in the hereditarian camp. Nevertheless, the studies were the beginning of a long tradition of research on the effects of early experience on intellectual development

and retardation. Skeels later published long-term follow-up data on some of these children, which indicated that they tended to retain their intellectual gains in adult life.

Psychiatrist George Tarjan (1912–1991) and his colleagues at Pacific State Hospital in California, an institution for persons with mental retardation, collaborated with colleagues at University of California at Los Angeles and the University of California at Riverside to develop a federally funded research program on the social epidemiology of mental retardation. This work continues today well after the end of Tarjan's life.

In support of the role of experience in long-term intellectual development, British psychologists A. D. B. Clarke and Ann M. Clarke published some interesting findings in their 1958 book. Their work suggested that individuals from highly deprived backgrounds may be quite retarded in their early development but have the potential for considerable gradual improvement when placed in more favorable surroundings (Clarke & Clarke, 1958).

The publication of psychologist Norman R. Ellis's 1963 edited book, *Handbook of mental deficiency: Psychological theory and research*, catalyzed the development of new research in the behavioral sciences relevant to mental retardation. In 1968, Ellis organized the annual Gatlinburg Conference as a meeting place, mainly for research psychologists interested in mental retardation and developmental disabilities.

In 1959, physician Jerome Lejeune (1926–1994) reported his discovery that Down's syndrome was generally characterized by an extra copy of chromosome number 21 (Hecht, 1994). This was but the first of a long list of chromosomal anomalies to be reported over the next few years, many of which were associated with mental retardation.

A team of researchers at the University of Washington in Seattle established an association between a particular syndrome of malformation in the offspring of alcoholic women (Claren & Smith, 1978; Jones, Smith, Ulleland, & Streissguth, 1973). The syndrome, which was dose-related in severity, is known as fetal alcohol syndrome (FAS) or (when milder) fetal alcohol effect (FAE). FAS is associated with mental retardation and other learning problems as well as behavior disorders in childhood and later life.

Anthropologist Robert Edgerton in 1967 published an ethnographic study of a cohort of persons released after institutionalization for diagnosed mental retardation. Virtually none of them accepted such a self-label. Their method of coping with the stigma of this diagnosis

involved drawing around themselves a protective “cloak of competence” and enlisting what Edgerton called “benefactors” in the community to help them do so.

The book, *The bell curve*, written by Herrnstein and Murray (1994), repeated Jensen’s arguments concerning the genetic basis of racial differences in intelligence test scores. Once more, the wrath of both professionals and journalists was elicited. The general consensus in the field is that such conclusions are not warranted by available data.

#### 1.01.10.16 Autism

The child psychiatrist Leo Kanner (1894–1981) was the first to describe a syndrome he originally called infantile autism, characterized by extreme social withdrawal and impairments in cognition and language. His basis for doing so was a series of 11 case histories of children he had seen in the Harriet Lane Home of Johns Hopkins University Medical School, where he was a professor (Kanner, 1943). Kanner and Eisenberg (1956) followed up, nine years later, 63 children who had been diagnosed as having early infantile autism. They found the child’s language at age five to have great prognostic significance. Those who had no language at age five rarely improved, while those who were able to speak at age five did much better at outcome.

Psychologist Bruno Bettelheim (1903–1990) viewed infantile autism in psychoanalytic terms and tended to “blame” the parents for causing it, even comparing the autistic child’s early experiences to those of concentration camp victims (e.g., Bettelheim, 1950, 1967). Such views are part of the explanation for his advocacy of separating these children from their parents in order to facilitate their treatment.

#### 1.01.10.17 “Minimal Brain Dysfunction” (MBD)/ADHD

Neurologist Alfred A. Strauss (1897–1957), who was associated with a residential treatment center for children at Northville, Michigan, carried out research in the 1930s and 1940s on what was originally labeled as endogenous vs. exogenous mental retardation. The term “endogenous” referred to children who functioned at a delayed level but appeared to be physically normal, whereas “exogenous” meant that there was some kind of evidence suggesting neurological dysfunction. Subsequently, the emphasis changed toward children without mental retardation but who had subtle difficulties that were presumed to be of neurological origin.

They were first referred to as “brain injured” (Strauss & Lehtinen, 1947), but later were said to suffer from minimal brain dysfunction (MBD).

In 1957 psychiatrist Max Laufer and pediatrician Eric Denhoff redefined a part of what had been considered to be MBD as a hyperkinetic behavior syndrome in children (Laufer & Denhoff, 1957). They advocated the treatment of this behavior disorder with amphetamines. An early description of such behavior problems by British physician G. F. Still (1902) was soon rediscovered. Psychologist Virginia Douglas at McGill University in Canada was soon to argue convincingly in the 1960s and 1970s that the main problem for these children was one of inattention rather than hyperactivity. Thus, it came to be that the current terminology for this common childhood problem is attention deficit hyperactivity disorder (ADHD) (Barkley, 1990).

#### 1.01.10.18 Learning Disabilities

In England, physician James Hinshelwood (1900) published a case of a child with what he called “congenital word blindness,” interpreted as a developmental variation of the adult neurological disorder of alexia. Psychologist Edmund B. Huey (1908) wrote a classic monograph on the psychology and pedagogy of reading (reprinted by the MIT Press). Samuel T. Orton (1928, 1937), a psychiatrist and neuropathologist at the University of Iowa, reviewed similar cases he had seen of children with “specific reading disability,” which he hypothesized to result from “strephosymbolia,” a lack of proper lateralization in the brain that leads the child to confuse mirror images such as b and d with each other. Orton also noted the association of reading, writing, and speech and language problems in children.

Educator Samuel A. Kirk (1904–1996) was the person who coined the term “learning disabilities” which served as a rallying point for parent groups as well as for researchers and practitioners since the 1960s. He was also among the first to do experimental research in special education (Kirk, 1952).

#### 1.01.10.19 Child Abuse and Neglect

The problem of abuse and neglect of children, including sexual abuse and psychological abuse, is ancient. It had long been the special concern of child welfare social workers, who are responsible for attending to the needs of family preservation and foster home placement when abuse is detected. The “battered baby” syndrome was rediscovered by the medical community in the

early 1960s, and many state laws were soon passed requiring professionals to report suspected abuse. Eventually other professionals such as psychologists and psychiatrists were drawn into professional activities related to abuse. One influential and scholarly book in this area was that written by sociologist David Finkelhor (1984).

### 1.01.11 RESEARCH ON ASSESSMENT IN THE TWENTIETH CENTURY

#### 1.01.11.1 Intelligence

James McKeen Cattell was a key figure in the early development of psychological assessment in the twentieth century. A disciple of Francis Galton, he originally tried to promote brief measures of basic psychological processes: sensory, motor, reaction time, and the like. His project to demonstrate the utility of such measures in college admissions at Columbia University failed, however.

In trying to demonstrate the presence of a general factor (*g*) underlying academic and intellectual abilities, Charles E. Spearman (1863–1945) originated the mathematical procedure that came to be known as factor analysis (Spearman, 1904). Godfrey Thomson (1881–1995) effectively critiqued Spearman's mathematical procedures by showing that one could obtain similar evidence for a *g* factor based on a matrix of random numbers (Thomson, 1919). Although Spearman's particular approach to factor analysis was soon superseded by others, his concept of *g* remains somewhat influential.

The psychologist Alfred Binet (1857–1911) and his psychiatrist colleague Theodore Simon (1873–1961) developed an intelligence test (Binet & Simon, 1905) that successfully distinguished children with mental retardation from others whose mental development was normal. They did so by using tasks that resembled what children actually do in school, rather than simple sensory-motor processes. Binet and Simon originated the practice of representing a particular test score in terms of "mental age."

Psychologist Henry H. Goddard (1866–1957), who had been appointed research director at the Vineland Training School in New Jersey in 1906, learned of the Binet–Simon test on a European trip and by 1908 had it translated into English (Doll, 1988). He readily confirmed the ability of the test to discriminate the performance of persons with mental retardation from that of others.

The use of intelligence tests on a broad scale first began with the mass testing of military

recruits upon entry of the United States into World War I. Robert M. Yerkes (1876–1956) and his colleagues developed the Army Alpha and Beta tests for this purpose and tested 1.7 million men. After the war, the industrial and educational uses of mass testing were developed, and testing became big business. Reasoning that mental age scores would not be useful for evaluating adults, Yerkes and other colleagues had earlier developed a "point scale" in which each item passed was simply given unit weight (Yerkes, Hardwick, & Bridges, 1915).

Lewis M. Terman (1877–1956), a psychology professor at Stanford University, developed a more standardized version of the Binet–Simon test and collected normative data with it. Terman's revision, known as the Stanford–Binet (Terman, 1916), made use of ratio IQ scores derived by dividing mental age by chronological age and multiplying by 100. The use of such a mental ratio score had been suggested by German psychologist Wilhelm Stern in 1912 (English translation, Stern, 1914). Terman soon began his "genetic studies of genius" (Terman, 1925) in which he followed up children with high Stanford–Binet scores. While few of them really turned out to be geniuses, these children indeed had high levels of academic, vocational, and social accomplishments later in life, validating the test as a predictor of such long-term outcomes (Terman & Oden, 1947).

In England, the mathematician R. A. Fisher and others working in agriculture went beyond descriptive statistics to develop the inferential approach, which eventually revolutionized both psychometric testing and experimental psychology.

The issue of test scores and race, class, and national origin became a major one after World War I, with IQ being interpreted by many as being more inherited than learned. The US government restricted immigration, and many states passed "eugenic" sterilization laws. In 1936 the Soviet government reacted in the opposite direction and put forth a decree banning the use of intelligence tests and all such "pedological" measures. One objection to the use of such measures was that they appeared to discriminate against individuals from proletarian backgrounds as compared to children of bourgeois parents and against members of various ethnic minorities within Soviet society. Oddly enough, among the researchers whose writings were banned at this time was psychologist Lev S. Vygotsky (1896–1934). Vygotsky had done research on the influence of cultural factors on intellectual development and spoke of the "zone of proximal development" as the area for educational focus with a child. When

his complete writings were later published, Vygotsky became one of the more influential scientists arguing for environmental influences on intelligence (Kozulin, 1990).

It was ultimately psychologist David Wechsler (1896–1981) who developed the intelligence test into the standardized form best accepted in the United States and other countries in the West (Wechsler, 1939). Wechsler agreed with Yerkes and his associates that mental age scores were not useful for adults.

Psychologist Raymond B. Cattell (1905–1998) (not to be confused with J. McK. Cattell) attempted to develop “culture free” intelligence tests. His theory distinguished between “fluid” and “crystallized” ability. Fluid ability refers to one’s performance on relatively novel tasks such as the Raven’s Progressive Matrices, which has been found to fall off more steeply with age than crystallized ability, which refers to more practiced skills such as vocabulary.

In 1969, Arthur R. Jensen, a professor of psychology at Berkeley, published an article in the *Harvard Educational Review* that was widely reviled both by many of his professional colleagues and by the public (Jensen, 1969). Essentially, Jensen argued that efforts at compensatory education had failed. He implied that differences in IQ scores among different racial and ethnic groups might be hereditary in nature and difficult to change. Such interpretations of the data on IQ and race were not uncommon in an earlier generation of behavioral scientists but certainly had become politically unacceptable by the 1960s.

Psychologist L. J. Kamin (1974) counter-attacked and charged psychologists of the past such as Henry Goddard and L. M. Terman and present day psychologists such as Arthur Jensen and Cyril Burt with holding racist attitudes. Kamin also charged Cyril Burt with fabricating data in his widely cited research on intelligence in twins reared apart. Careful examination of Burt’s data by others suggested that these charges had merit.

### **1.01.11.2 Categorical Diagnosis of Mental Disorders**

Adolf Meyer (1866–1950) was probably the most influential American psychiatrist of his day. Trained in Switzerland in neuropathology and psychiatry, he began work in 1893 as a staff pathologist at Illinois Eastern Hospital in Kankakee (Winters, 1966). He was frustrated in his task from the beginning because of the poor quality of the case records. It was no use trying to establish the correlation of brain autopsy findings with clinical symptoms if the accounts of what the patient was like in life were

missing or flawed. Thus, Meyer began a lifelong concern with developing adequate methods for the psychiatric assessment and classification of patients. He continued this preoccupation in subsequent positions at the Worcester Asylum (now Worcester State Hospital) in Massachusetts and ultimately as professor of psychiatry at the Johns Hopkins University. Meyer’s standardized descriptions of psychiatric syndromes, or “reaction” patterns as he called them, became well known. Incidentally, Meyer was also interested in therapeutic methods and began applying psychoanalysis within a New York state hospital as early as 1902.

After World War II, many professionals were critical of psychiatric categorical diagnoses because of their low reliability (e.g., Stengel, 1959). Shorter (1997, p. 297) refers to Stengel’s article as “the opening cannon in the campaign to revise diagnosis.” The group of psychiatrists at Washington University in St. Louis were particularly influential in advocating a more rigorous approach to diagnosis. John P. Feighner, the chief psychiatric resident at Washington University at one point, published the standardized research criteria in use in the Department of Psychiatry there (Feighner et al., 1972). These “Feighner criteria” rapidly came into use by others, especially research psychiatrists and psychologists. An intermediate step between the use of Feighner criteria and the *DSM-III* was constituted by the “research diagnostic criteria” promulgated by Spitzer, Endicott, and Gibbon (1978).

British child psychiatrist Michael Rutter, in his Isle of Wight studies in 1975, demonstrated the applicability of rigorous epidemiological methods in this field. As part of this work he devised standardized psychiatric interviews for parents and children and standardized teacher rating forms. He showed a concern with assessing the reliability and validity of these methods and also was among the first to advocate the use of a multiaxial classification system.

Psychologist David Rosenhan published a striking study using “pseudopatients” who gained admission to mental hospitals on the basis of faked auditory hallucinations (Rosenhan, 1973). They began to act in their usual way, denying any such symptoms, immediately upon admission to a mental hospital. It typically took the hospital a matter of weeks to discharge them, and even so the hospital psychiatric staff tended to render discharge diagnoses such as “schizophrenia in remission.” For many, this brought the credibility of the traditional diagnostic system even more into question.

A task force of the American Psychiatric Association soon began work on what was to

become in 1980 a thorough revision of its *Diagnostic and statistical manual*. One issue that emerged in the discussion was the question of whether homosexuality should be included in the revised manual as a “mental disorder.” Gay advocacy groups were unhappy that homosexuality had been included in the DSM-II in 1968. There was evidence, for example, from the research of psychologist Evelyn Hooker (1907–1996) (e.g., Hooker, 1969), that homosexuality was compatible with the absence of symptoms of any disorder (unless one wished to regard sexual orientation itself as a symptom). To make a long story short, the *DSM-III* taskforce decided to delete homosexuality from the manual, and the American Psychiatric Association affirmed this in an unprecedented referendum in 1974.

Psychologist Theodore Millon lobbied in the *DSM-III* taskforce to include a separate axis for personality disorders, a suggestion that it ultimately implemented, though it did not define many of the personality disorders precisely as Millon may have wished.

According to Reisman (1991), the original *Diagnostic and statistical manual (DSM)* of the American Psychiatric Association in 1952 listed 60 disorders. The *DSM-II* in 1968 listed 145, and *DSM-III* in 1980 listed 230 of them. It remains to be seen how well justified this inflation of the number of categories was. It is certainly easier to diagnose the broader categories reliably than it is the narrower ones. Considerable information has been collected on the reliability of the newer *DSM* categories, and they do seem to be more reliable than their counterparts in the earlier versions of the *DSM*. Although the validity of categories of descriptive psychopathology is a perennial issue, it is clear that they can be made more reliable by standardizing the questions asked, the way the answers are coded, as well as the way the information is combined.

#### 1.01.11.3 Interviews

Informal interviewing surely goes back to the dawn of medicine as well as that of philosophy. A Socratic dialogue is after all a type of interview. This is no doubt the most common “assessment” procedure used by mental health professionals and is often the only one used. It was only recently, however, that standardized interviews began to be developed to increase the reliability and validity of clinical judgment. Psychiatrist Max Hamilton (1960) published a standardized, interview-based rating scale for depression that is still in common use. Sociologist Lee Robins and her colleagues developed the Diagnostic Interview Schedule (DIS), to be

used by lay interviewers, to generate *DSM-III* diagnoses. The corresponding standardized interview for use by clinicians is called the Structured Clinical Interview for Diagnosis (SCID). These procedures have unquestionably increased the reliability of diagnosis for research purposes, though the question of validity remains a perennial one.

#### 1.01.11.4 Self-report Measures of Personality and Psychopathology

One of the first measures of this kind was the Personal Data Sheet developed by psychologist Robert S. Woodworth (1869–1962), a professor at Columbia University, for military use during World War I (Woodworth, 1917). The items were rationally selected, mainly from psychiatric textbooks, and not initially subjected to empirical validation procedures. Thus, the content of this questionnaire was based on descriptive psychopathology, as developed over the entire preceding century.

The Minnesota Multiphasic Personality Inventory (MMPI), developed by psychologist Starke R. Hathaway (1903–1984) and psychiatrist J. C. McKinley in the 1930s and 1940s (e.g., Hathaway & McKinley, 1940, 1943), was innovative in several ways. For one thing, the clinical scales of the MMPI were empirically validated using psychiatric patients with particular diagnoses as criterion groups and visitors to the University of Minnesota hospitals as a control group. Also, the MMPI contained several innovative “validity scales” that could be used to identify subjects who might be too confused to give reliable answers, who could not read well enough to use the test properly, or who were trying to present themselves to the examiner in an unduly favorable or unfavorable light. The MMPI quickly became one of the most frequently used assessment procedures of its kind. The thousands of empirical studies conducted with it ultimately gave it even more credibility than it acquired from its initial method of validation.

Psychiatrist A. T. Beck published the first article describing the Beck Depression Inventory (BDI) in 1961 (A. T. Beck, Ward, & Mendelsohn, 1961). This theoretically based scale came into extremely wide use and has recently been revised.

Exploiting the conceptual distinction between “state” and “trait” measures, psychologist Charles Spielberger and his colleagues devised and validated separate pencil and paper questionnaires to measure these two different types of anxiety (Spielberger, Gorsuch, & Lushene, 1970). Part of the innovation in these questionnaires is simply in the way the items are

worded. For example, one might ask “do you have butterflies in your stomach at this moment?” (state) vs. “do you frequently get butterflies in your stomach?” (trait). A state anxiety measure might be more useful in assessing a person’s response to having blood drawn in a medical clinic while a trait measure would be a better choice in assessing neurotic anxiety.

#### **1.01.11.5 Rating Scales**

In 1962, psychologist Lester Luborsky published an article on his Mental Health/Sickness Rating Scale. This work was incorporated by Endicott and Spitzer (a psychologist–psychiatrist team) in their Global Assessment Scale (GAS), which was developed in 1976. In this scale, a score of 0 would be given if the patient had died by suicide and a score of 100 if the individual showed superior functioning in every area of life. The current *DSM* uses a further revised version of this called a Global Assessment of Functioning (GAF) scale, which has been shown to be quite reliable.

Psychologist C. Keith Conners developed a teacher rating scale in the 1950s primarily for use in research on the efficacy of stimulant medications with behavior disordered children. This scale soon came into wide use for diagnostic purposes as well as being commonly employed as a treatment outcome measure. It continues to be in use today. Conners also developed parallel scales for use with parents and for reports by the young persons themselves.

Psychologist Thomas Achenbach and his colleagues developed and validated an integrated set of rating scales for assessing child psychopathology, including the Child Behavior Checklist (for parent ratings), a Teacher Report Form, and a Youth Self-report scale. Many of the items of these scales are parallel, and thus, cross informant syndromes can be characterized by combining the scores from all three types of informants. The development of a sizable database using these scales provided a new perspective on the classification of child psychopathology as well as its measurement (Achenbach & Edelbrock, 1978). This approach has become widely influential, especially in the research community concerned with child and adolescent psychopathology.

#### **1.01.11.6 Infant Assessment**

Standardized procedures were soon developed for testing infants which superficially resembled those of intelligence tests for older children. Arnold L. Gesell (1880–1961), who

was both a psychologist and a physician, developed one of the earliest tests of this kind, with 195 items for infants ranging in age from 3 to 30 months and others for babies up to age 60 months (Gesell, 1929). However, scores on these infant tests proved to have very little long-term stability and thus were mainly of use for research purposes.

The most widely used method for assessing infants, developed by psychologist Nancy Bayley (1899–1994), is known as the Bayley Scales of Infant Development (BSID) (Bayley, 1955, 1969).

#### **1.01.11.7 The Rorschach Test**

Swiss psychiatrist Herman Rorschach (1884–1922) published the manual for his famous inkblot test in 1921, the year before his death, and did not have a chance to follow up on his initial work. He had tried out the procedure with patients suffering from various types of psychopathology as well as normal controls. Rorschach originally had a plan of using 15 blots but could only get the publisher to print 10 blots, and the blots unexpectedly were printed including varying shades of gray. Nevertheless, interpreters managed to capitalize on every serendipitous development of this kind. Many of Rorschach’s original interpretive hypotheses are still considered valid by many present users of the procedure, most of whom are psychologists of a psychodynamic persuasion.

Samuel J. Beck, who did his Ph.D. dissertation at Columbia on the Rorschach, went on to try to objectify the scoring methods most frequently selected for response and collected norms on the frequency of different perceptions of these areas. It was then possible to score “form quality” (perceptual accuracy, thought to be lowered in schizophrenia) on the basis of these norms (e.g., S. J. Beck, 1937, 1944).

In 1961, psychologist Wayne Holtzman and his colleagues published a new inkblot test that had a more standardized procedure for administration than the Rorschach test. The Holtzman test had clearer rules for scoring and provided norms for various patient groups and for the general population (Holtzman Thorpe, Swartz, & Herron, 1961). Despite its superior psychometric properties, this test was not frequently used in clinical evaluation. It was considered too lengthy and cumbersome.

#### **1.01.11.8 Human Figure Drawings**

During the 1920s, psychologist Florence L. Goodenough (1886–1959) at the University of Minnesota developed and validated a

standardized way of scoring a child's drawing of a man (Goodenough, 1926). This had some use as a brief cognitive screening procedure, for example, with kindergarten children and in cross-cultural research, but is not often considered to be of much practical clinical value.

In 1948, psychologist Karen A. Machover (1902–1996) wrote a book on the use of human figure drawings as a brief projective test. Unlike Goodenough, Machover did not formally validate her procedure but simply shared with readers her speculations about how such drawings might be interpreted. Human figure drawings of this kind have come into very common use, particularly in the assessment of children. Psychologists Loren and Jean Chapman (1967, 1969) demonstrated that people trying to interpret such human figure drawings are subject to the phenomenon of “illusory correlation” in which they read their biases into what they see. For example, one might judge drawings focusing on the eyes to indicate paranoid trends, when no empirical association between drawing eyes in this way and paranoia exists.

#### **1.01.11.9 Thematic Assessment Methods**

In 1935, Christiana Morgan and Henry A. Murray (1893–1988) reported a new procedure, the Thematic Apperception Test, in which the individual was asked to look at reproductions of paintings or photographs and to tell a story about the scene portrayed in each picture. In research in the Harvard Psychological Clinic, the diagnostic team concluded that this was the best procedure in their battery for identifying individuals who were seriously self-deceived concerning their motives (Murray et al., 1938).

#### **1.01.11.10 Sociometric Measures**

The “sociogram” based on people's choices of others with whom they would like to share activities or others whom they would like to avoid was developed by psychiatrist Jacob Levy Moreno (1892–1974). Moreno introduced sociometric techniques in a rather unusual book, *Who shall survive?* (Moreno, 1952).

#### **1.01.11.11 Assessment of Social Competence**

Psychologist Edgar A. Doll (1889–1969) once served as director of research at the Vineland School and maintained a lifelong interest in the problem of mental retardation. In 1935, Doll published the Vineland Social Maturity Scale, which assessed what an individual characteristically was capable of doing in everyday life, as

judged by a knowledgeable informant. Such information soon became a part of the way mental retardation was defined.

#### **1.01.11.12 Assessment of Marital and Family Relationships**

Psychologist Richard Abidin developed an innovative measure called the Parenting Stress Index (PSI) (Loyd & Abidin, 1985). This work recognized the increasing realization in the field that not only parents have effects on their children. Children's behavior also affects their parents as well, and a difficult child can be a major source of parental stress.

#### **1.01.11.13 Neuropsychological Measures**

As neurology developed increasing sophistication, there grew a need for psychological assessment procedures sensitive to the effects of neurological lesions. An early example of such a technique and one that continues to be used frequently is the Bender Gestalt Test, developed by child psychiatrist Lauretta Bender (1938). She used designs taken from the work of Gestalt psychologist Max Wertheimer. This test requires the copying of these nine geometric designs with pencil and paper. A second widely used neuropsychological test, the Visual Retention Test (Benton, 1945), involves memory as well as drawing skills. The Halstead–Reitan battery, which developed out of the work of Ward Halstead (1947), involved a combination of many different subtests and soon came into common use.

A neuropsychologist who became renowned in the field was the Soviet scientist, Alexander R. Luria (1902–1977) who often tended to use qualitative assessment techniques rather than standardized quantitative techniques.

#### **1.01.11.14 Biological Assessment Procedures**

A specific test for the presence of syphilitic antibodies in the blood was developed by August von Wasserman in 1906. About 90% of the patients with general paralysis of the insane (GPI) tested positive, confirming the syphilitic etiology of this disease.

The German neuropsychiatrist Hans Berger, after 15 years of trying, succeeded in 1929 in recording electrical activity from the intact human scalp. Thus, the electroencephalograph (EEG) was born. By 1934 the first such recording in the United States had occurred, at Harvard Medical School, and by 1939 the EEG was in general use. It proved to be especially valuable in the diagnosis of seizure



disorders and presumably in distinguishing hysterically-based “epilepsy” from the neurologically-based kind.

Physician Murray L. Barr developed a cytogenetic assessment procedure which an individual’s cells could be examined under a microscope to determine the number of X chromosomes present in each one. This would ordinarily indicate simply whether the person was male or female (since males have only one X chromosome, while females have two). There are rarer cases of females with only a single X (Turner’s syndrome), males with more than one X (Klinefelter’s syndrome), or other persons with multiple supernumerary X chromosomes. Each of these syndromes has its own profile of cognitive and behavioral characteristics (e.g., Money, 1963).

Physician Robert Guthrie (1916–1995) developed a blood test for detecting phenylketonuria (PKU) in newborn babies, making possible a program of early dietary treatment to prevent mental retardation and associated behavior disorders in this Mendelian recessive condition.

Geneticist Herbert A. Lubs (1969) published his findings on a “marker X chromosome,” later to be implicated in the discovery of the fragile-X syndrome, one of the most common genetic causes of mental retardation and various associated disabilities (de la Cruz, 1985). It is of interest that fragile-X is a disease that actually does become worse in successive generations as it is passed on because of the increasing number of trinucleotide repeat mutations on the X chromosome. Perhaps this provides at least limited vindication of the nineteenth century psychiatrist B. A. Morel’s concept of degeneration.

Endocrinologist Rosalyn S. Yalow developed techniques for the radioimmunoassay of peptide hormones. Among other uses, these procedures allowed the diagnosis and treatment of thyroid disease in newborn infants before any signs of cretinism appeared. She won a Nobel prize for this work in 1977.

New imaging technology is one of the fastest growing areas of assessment. Computerized tomography (CT), for example, is a noninvasive procedure that enables one to view the brain in cross-section and to visualize neuronal structures there as well as tumors, areas of hemorrhage, and so on. Johnstone et al. (1976) used CT relatively soon after it became available. They were able to document that persons with chronic schizophrenia tended to have enlarged cerebral ventricles. They also examined the relation of these to degree of impairment. Besides CT, the new techniques include magnetic resonance imaging (MRI), the positron emission technique (PET) for observing brain metabolic activities,

and the study of regional cerebral blood flow (RCBF)(Andreasen, 1988).

## **1.01.12 RESEARCH ON TREATMENT IN THE TWENTIETH CENTURY**

### **1.01.12.1 The Boston School of Psychotherapy**

According to historian Eugene Taylor, the first use of different psychotherapeutic methods began at the Massachusetts General Hospital between 1903 and 1905 and on Ward’s Island in New York after 1905. This work, which had its origins in developments in France, was subsequently overshadowed in the United States by developments in psychoanalysis.

### **1.01.12.2 Psychoanalysis and its Derivatives**

Psychoanalysis dominated the mental health scene for more than half of the century and remains of interest today. Psychoanalysis usually involves daily or at least relatively frequent meetings between the therapist and patient, with the patient lying on a couch and engaging in free association (“just say whatever comes to mind”). From the first, this approach to treating neurotic problems achieved good “consumer acceptance” and was sufficiently remunerative that its practitioners could make a living. Admittedly, psychoanalysis was not only a method of treatment but a theory of psychopathology and had important implications for assessment as well. Detailed coverage of all of this is well beyond the scope of this chapter, since there are hundreds of books on this topic.

Freud had broken with his colleague Josef Breuer before the turn of the twentieth century and henceforth referred to psychoanalysis as his own creation. He published books on the interpretation of dreams in 1900, on the psychopathology of everyday life in 1904, on jokes and their connection with the unconscious, on psychosexual development, on sexuality in general in 1905, and many more in later years.

Although psychoanalysis was considered mainly as a treatment for neurotic symptoms, some practitioners tried it with psychotic patients. For example, psychoanalyst Marguerite Sechehaye did psychotherapy with schizophrenic patients at the Burgholzli hospital in Zurich and described her experiences in a 1947 book.

Otto Rank (1884–1939) was encouraged by Freud to obtain a Ph.D. in psychology from the University of Vienna and was for many years a member of the inner circle of psychoanalysis.

Rank's book on the *Trauma of birth* was but the first public example of his departure from strict orthodoxy. Part of Rank's historical importance is as a liaison man between psychoanalysis, social work, and psychology. At a later point in his career, Rank taught at the School of Social Work in Philadelphia and supervised the clinical work of Jessie Taft (Robinson, 1962), who even became his biographer (Taft, 1958). Jessie Taft was, in turn, one of the early supervisors of clinical psychologist Carl Rogers (1902–1987). Thus, there are indirect links between psychoanalysis and Rogers' client-centered or person-centered therapy.

In Germany in 1936, obituaries recorded the death of Bertha Pappenheim (1859–1936), a woman who was only three years younger than Freud. She was a prominent social worker and was perhaps the only one to have her likeness featured on a postage stamp. It eventually became known, via Ernest Jones' biography of Freud, that Bertha Pappenheim was the actual name of the patient in Freud's case history of "Anna O." Breuer and Freud (1895) had credited this patient with inventing the "talking cure." Records subsequently unearthed have documented that Pappenheim was not cured by psychoanalysis after all. She subsequently spent time as a patient in a sanatorium. Incidentally, Pappenheim lived on to become an effective opponent of the use of psychoanalytic methods for children under her care.

The psychoanalyst Eric H. Erickson (1902–1994) was trained by Anna Freud. Although he always presented himself as orthodox in his theoretical beliefs, he nevertheless managed to produce some of the most original and influential contributions. For example, in *Childhood and society*, Erikson (1950) transformed Freud's psychosexual stages into psychosocial ones that many readers found more palatable and added something all of his own about the stages beyond childhood of identity development, intimacy, generativity, and integrity. His writings on identity development have in particular generated much research related to development and the types of psychopathology that attend failure at negotiating it. In terms of treatment activities, Erickson was an acknowledged master at psychoanalytic play therapy.

In the 1950s, psychoanalysts began to be concerned about the absence of research evidence concerning the efficacy of this type of therapy. In 1953 the American Psychoanalytic Association appointed a Central Fact Gathering Committee to address this question. According to Shorter (1997, p. 311; see also Knight, 1953), the discussions of this committee were inconclusive, its report was kept

confidential, and the committee was disbanded in 1957.

One of the more recent variants of psychoanalysis is psychoanalytic self-therapy, developed by psychiatrist Heinz Kohut (1913–1981), an emigrant from Vienna to Chicago. Kohut is reported to have been particularly sympathetic to narcissistic and borderline patients, both of whom have notable difficulties with the "self."

With the emergence of managed care in the United States, it became more and more difficult for psychoanalysts and others doing long-term psychotherapy to be reimbursed for their services by third-party payers. Nevertheless, this type of therapy maintains its appeal to some who are willing to pay for it out of their own pockets. Historian Eugene Taylor (personal communication, January 13, 1998) ventured the following prediction:

psychologists opposed to the medicalization of psychotherapy and the imposition of managed care and its required treatment protocols will split off and develop autonomously, possibly launching an entirely new generation of deregulated philosophical psychologists whose job is to teach people how to live, as psychotherapy itself becomes demedicalized, de-pathologized, and more oriented toward education for transcendence.

### 1.01.12.3 Play Therapy for Children

Psychoanalytic methods were first adapted for use with children by Hermine von Hug-Hellmuth (1921). Children generally are not able to lie on a couch and free associate but prefer to play with toys, and thus what became play therapy was a necessary modification. The next developments in the psychoanalytic treatment of children were due to Melanie Klein (1882–1960), who was known for her boldness in making "deep" interpretations of children's play activities (e.g., Klein, 1932). The principal historic importance of Klein's work may lie in its role in the emergence of the "object relations" school of psychoanalysis that was especially influential in Britain. One of Klein's pupils was the pediatrician and psychoanalyst Donald Winnicott (1896–1971). His clinical concepts included the idea that "transitional objects" such as blankets and teddy bears are used by children as mother substitutes.

Child psychiatrist Frederick Allen (1890–1964) had obtained his M.D. from Johns Hopkins University after previously spending three years as a school psychologist. He became Director of the Philadelphia Child Guidance Center and contributed to the literature on play therapy. His work was influenced by that of Otto Rank.

#### 1.01.12.4 Japanese Therapeutic Approaches

The Japanese psychiatrist Shoma Morita (1874–1952) developed psychotherapeutic procedures based on Zen Buddhism that were named after him. Although such approaches are commonly used in Japan, they have not become widely adopted elsewhere (Moriyama, 1991). There continue to be major language and cultural barriers between East and West in the mental health field, as has been illustrated every time this topic has come up in the present chapter.

#### 1.01.12.5 Behavior Therapies

In 1924, psychologist Mary Cover Jones published a study of the case of “Peter” in which she showed how the child could be induced to overcome his fear of a furry animal by gradually increasing exposure to it in the presence of competing activities such as eating. Her supervisor in this research was psychologist John B. Watson, well known as one of the founders of behaviorism and a strong advocate of this theoretical position (Watson, 1913). At the time he worked with Jones, Watson had already been dismissed from his academic position at Johns Hopkins University and had entered the world of business, working for an advertising firm in New York. As Taylor remarks (personal communication, January 13, 1998), “behaviorism met Madison Avenue in the 1920s.”

The name of William H. Burnham (1855–1941) is not well known, but Kazdin (1978) says that he was an important pioneer in the development of behavior therapy. Burnham (1924) published a book on *The normal mind: An introduction to mental hygiene and the hygiene of school education*.

Physiologist Edmund Jacobson published an influential procedure for systematically teaching people to relax (Jacobson, 1929). This procedure was later incorporated into behavioral approaches to desensitizing phobias and had many applications in behavioral medicine.

Another behavioral psychologist, Knight Dunlap (1875–1949), a professor at Johns Hopkins University, wrote a book about the use of “negative practice” in order to get rid of unwanted habits such as stuttering or thumb sucking (Dunlap, 1932). To get rid of such a habit, the individual was encouraged to perform it voluntarily over and over, until it became unpleasant to do so.

In 1935, physician Walter L. Voegtlin and his colleagues from the Shadel Sanitarium in Seattle proposed the use of aversion therapy to help alcoholics achieve abstinence. Voegtlin was a

gastroenterologist. In the 1950s a follow-up study was published of over 4000 patients who had received this type of treatment. The results were promising though not definitive.

The “bell and pad” method for treating bed-wetting in children (still in use today) was first popularized by Mowrer and Mowrer (1938). Their work was based on a method originally developed by pediatrician M. Pfaundler in 1904. A pad containing electronic moisture sensors was placed in the child’s bed. If the child urinated during the night, a bell rang to awaken the child, who was then supposed to get up and use the bathroom to finish urinating. Repeated use of this procedure was observed to decrease and then eliminate enuresis, although relapses were a problem. The Mowrers explained how this procedure worked in terms of a Pavlovian conditioning process. Many years later, Azrin and Foxx (1974) developed and field tested a more elaborate version of the Mowrers’ behavioral procedures that could even enable parents to toilet train their child in “less than a day.”

One of the earliest practitioners to make a living doing behavior therapy was Andrew Salter (1914–1996), from New York City. He had done his undergraduate work at New York University but had no graduate or professional degree. Salter was influenced by Clark Hull’s behaviorally oriented writings on hypnosis (Salter, 1941) as well as Pavlov’s work on conditioned reflexes (Salter, 1949). His concept of “assertion training” influenced the work of Joseph Wolpe.

According to Kazdin (1978), the psychoanalyst Alexander Herzberg moved to London during World War II and developed a modification of psychoanalysis that he called “graduated tasks” (Herzberg, 1945). Kazdin notes that psychologist Hans J. Eysenck (1916–1997) worked with Herzberg in the 1940s and was influenced by him, thus providing part of the context for Eysenck’s later emergence as an advocate for behavior therapy.

Eysenck received his Ph.D. at the University of London in 1940 and in his early days in the field viewed clinical psychology as involving only research and diagnostic testing, not treatment. That he left to psychiatry. Eysenck spent some time in the United States and changed his mind about the role of psychologists as therapists. In 1952 he published a review of the effects of traditional psychotherapy which had the effect of a bombshell on psychotherapists who read it. He concluded that there was no evidence that psychotherapy was effective, because about two-thirds of the patients improved with or without therapy. Eysenck went on to pioneer the field of behavior therapy in Britain.

In 1958, psychiatrist Joseph Wolpe (1915–1997) published an influential book about the use of systematic desensitization for phobias (Wolpe, 1958), claiming a 90% success rate with this type of problem. In its most common variant, Wolpe's treatment involved teaching the patient to relax (using the procedure of Jacobson, 1929). Then the patient learned to associate this state of relaxation with a series of imagined scenes that were arranged in a hierarchy of increasing anxiety related to the object of the patient's phobia. The reasoning was that relaxation and anxiety are incompatible responses, and anxiety can be counter-conditioned in this way. Wolpe had received his M.D. from Witwatersrand in South Africa. In 1956 he came to California to spend his sabbatical at the Center for Advanced Study in the Behavioral Sciences, and it was there that he wrote his 1958 book. In 1962 he took a position at the University of Virginia and then moved to Temple University in 1965, where he spent most of his career.

Systematic desensitization soon attracted the attention of behavioral psychologists who began to carry out empirical research on this topic (e.g., Rachman, 1959; Lang & Lazovik, 1963). Controlled research was supportive of Wolpe's claim that such treatment was effective. However, critics of this research (e.g., Breger & McGaugh, 1965) pointed out, with good reason, the fact that this procedure did not seem to be based purely on behavioral principles. In particular, the role of the hierarchy of imagined scenes in the therapy seemed very cognitive. It is thus not a complete surprise that behavior therapy, as it developed, tended toward a greater and greater rapprochement with cognitive approaches.

#### 1.01.12.6 Behavior Analysis

The basic research of psychologist B. F. Skinner (1904–1990) on the functional analysis of the behavior of rats and pigeons ultimately created a clinical specialty of "applied behavior analysis" that separated itself from other behavioral and certainly from cognitive-behavioral approaches to treatment. Skinner received his Ph.D. from Harvard in 1931 and published his first book in 1938. Psychologist Sidney W. Bijou, who was once a colleague of Skinner's at Indiana University, was one of the first to apply this type of behavior analysis to children and individuals with mental retardation or autism (Bijou, 1996). Others followed suit. For example, using a single subject design, Carl D. Williams (1959) illustrated the use of extinction procedures to eliminate tantrum

behaviors in a child at bedtime. Such advice about how to deal with tantrums is now routine in behavioral parent-training programs.

Behavior analysis also proved to be useful with psychotic adult inpatients, as shown by Ayllon and Michael's (1959) paper on "the psychiatric nurse as a behavioral engineer." In 1962, psychologist Montrose Wolf went a step further to design a token economy at Ranier School, an institution for persons with retardation, in Washington. Ayllon and Azrin (1968) did the same thing for all types of state hospital patients. In the 1970s Wolf designed what came to be called the Achievement Place model, a type of family group home for youngsters considered to be at risk for delinquent outcomes. In an Achievement Place home, the live-in foster parents used behavioral principles to encourage various types of prosocial behavior, everything from cleaning up the bathroom, to doing homework, to avoiding aggressive outbursts.

By the 1960s, psychologist O. Ivar Lovaas and his colleagues at University of California at Los Angeles (e.g., Lovaas, 1987) had become engaged in intensive treatment efforts for children with autism, using what came to be called "discrete trial" operant conditioning methods. These efforts eventually attracted great interest and even generated demands from parent groups around the country for their local school systems to fund behavior analysts to work with their children.

Psychiatrist Stuart Agras and his colleagues (Agras, Barlow, & Chapin, 1974) published data showing that anorexia nervosa could be treated using behavior modification. In this and subsequent research it was shown that weight gain could be used as the basis for presentation of contingent reinforcement such as the opportunity to watch television or to see a boyfriend. Anorexic patients exposed to such contingencies gained weight, at least while the contingencies were in effect.

#### 1.01.12.7 Cognitive Therapy

Although not labeled as such at the time, one can see in retrospect that the "personal construct" therapy and the related assessment procedures developed by George A. Kelly (1905–1966) could be considered to be an early variant of cognitive therapy (Kelly, 1955). Kelly thought of every person as a sort of scientist who developed particular ways of construing other people and events as a result of experience. He tried to assess these personal constructs and to help the person modify them so reality would be represented as accurately as possible. Another early example of cognitive therapy

was the “rational emotive” treatment developed by psychologist Albert Ellis (1958), a prolific writer on this topic.

In terms of basic research, one could find few more effective advocates of the integration of cognitive and behavioral approaches than Albert Bandura, a professor of psychology at Stanford University (e.g., Bandura, 1971). Early in his career, Bandura published research showing the effects of modeling or imitation on children’s behavior, including aggression. Subsequently, Bandura’s research incorporated the influence of cognitive variables in such processes. For example, he found that children’s behavior would only be influenced by models if they directed their attention toward what the model was doing and engaged in mental rehearsal in order to remember what they saw. Seeing the model rewarded for certain behaviors might not influence the child observer at the time, but would do so later if rewards became available to the child for demonstrating the behaviors later. Bandura’s theory also emphasized the importance of “self-efficacy” or the belief that one would be able to accomplish some goal. Even though Bandura did not do treatment outcome research, his theory provided a framework for cognitive-behavior therapy.

Beck’s model of cognitive therapy for depression has been influential (Beck, 1976). Among other strategies, it encourages patients to become aware of their negative “automatic thoughts” and to learn to dispute them.

One of the more promising new cognitive-behavioral treatments to emerge is the one developed by Marsha Linehan for borderline personality disorder (Linehan, 1993). It is called “dialectic behavior therapy” in that among other strategies it encourages patients to explore possible alternative explanations for certain distressing events they describe. This treatment has already been shown to be effective in reducing the number of self-destructive acts engaged in by patients with BPD.

#### **1.01.12.8 The Interpersonal Approach**

One of America’s most innovative psychiatrists was Harry Stack Sullivan (1882–1949). He was especially known for his ability as a therapist working with young male schizophrenic inpatients (Sullivan, 1931). On occasion he encouraged such patients to become intoxicated on alcohol for 3–10 days in order to “open them up” to psychotherapy. Although influenced by psychoanalysis, he defined the field as being primarily concerned with interpersonal relationships (Sullivan, 1953). Sullivan coined the term “significant other” for the person to whom

one is most attached and emphasized the importance of “consensual validation” not only by one’s therapist but by peers or “chums.” Thus, Sullivan thought it important to encourage close, confiding social relationships, especially among youth.

#### **1.01.12.9 Research on Psychotherapy**

From the time of Hippocrates down to World War II, one would be hard pressed to come up with many examples of controlled experimental research on psychotherapy or even on many of the medical approaches used to treat mental disorders. It is to the credit of psychologist Carl R. Rogers that he not only developed a new approach to therapy (Rogers, 1951), he also began to use some of the research methods he had learned about in graduate school to study both the process and the outcome of the therapy he had developed. Rogers tape-recorded therapy sessions, had them transcribed, and used coding procedures to try to quantify what was happening. He used control groups, for example, persons who were asked to stay on a waiting list for a time with the promise of receiving the same therapy at a later time. He and his colleagues used standardized measures such as Q-sorts to try to get at psychotherapy outcomes.

Of course, Rogers’ therapy tapes and transcripts could also be used by those with theoretical views quite in contrast with his own. Psychologist Charles Truax (1966), for example, managed to demonstrate that Rogerian therapists were not as nondirective as might have been supposed. They were more likely to pay attention, and thus presumably to reinforce, some types of client statements than others.

Psychologist A. E. Bergin is known as both a researcher and as a reviewer of psychotherapy research. One theme he focused on was the fact that psychotherapy can harm as well as help (Bergin, 1966). Researchers and clinicians therefore need to be concerned about “side effects” as well as “effects.”

One interesting twist on psychotherapy research presented data suggesting that the provision of psychotherapeutic services in an HMO setting could actually reduce patients’ inappropriate utilization of nonpsychiatric medical care (Cummings & Follette, 1967). Such conclusions are rather self-serving to the mental health professions, of course, and more research is needed to confirm or disconfirm these findings.

Another innovation in psychotherapy research was the trend toward using meta-analysis, begun by Smith and Glass (1977). In a meta-analysis, the existing research literature

is reviewed quantitatively rather than only in the traditional narrative fashion. Summary statistics such as “effect sizes” are calculated to pool the results of many separate investigations. The best known conclusion of the Smith and Glass (1977) review is simply that psychotherapy “works” as compared to various control conditions. Scholars criticized some authors of meta-analyses, for averaging in flawed studies with those that were well designed. In the familiar language of the computer world, this amounts to “garbage in, garbage out.” Meta-analysis was soon applied to child therapy as well (Casey & Berman, 1985; Weisz, Weiss, Alicke, & Klotz, 1987), with much the same conclusions.

The neglected issue of the clinical significance of psychotherapy research was addressed by Jacobson and Revenstorf (1988). It is important in reading a research article in this domain to look not just for “statistically significant” findings, which in the case of studies with large samples may be trivial in size. Instead, one should ask whether the amount of change observed would actually be important to a therapy client. For example, did the client come into therapy with a particular psychological disorder and move into the normal range as a result of treatment?

#### **1.01.12.10 The Psychotherapy Integration Movement**

One problem for psychotherapy researchers was the sheer number of “new” therapies being developed. It was widely estimated in the 1980s that there were more than 130 different types of psychological therapies. There was thus a need to try to specify what these diverse approaches might have in common that could account for their effectiveness, as well as to work toward designing specific psychological therapies for specific disorders (Beutler, 1979). A classic work on this topic was psychiatrist Jerome Frank’s (1973) book, *Persuasion and healing*. Frank examined not only different types of formal psychotherapy but also traditional religious or shamanistic approaches to healing (for an updated version of this book, see Frank & Frank, 1991).

#### **1.01.12.11 Group Therapy**

According to historian Eugene Taylor, group therapy began with the work of Joseph Hersey Pratt in Boston in 1904, with the Class Treatment of patients with tuberculosis. The term “group therapy” was used by psychiatrist J. L. Moreno in 1932. Moreno also developed a

version of this approach known as psychodrama and in 1942 established a Psychodramatic Institute.

According to Shorter (1997), psychiatrist Joshua Bierer (1901–1984) introduced the use of group psychotherapy at Runwell Hospital, London, and then at other London hospitals. This eventually took the form of a Social Club, which elected its own members. Bierer founded a Social Psychiatry Center in Hampstead in 1946 and set up the first day hospital in England in 1948.

During the 1960s and 1970s, many new forms of group therapy were developed. Encounter groups became a fad (especially in California). Gestalt Therapy (not to be confused with Gestalt Psychology) and other approaches associated with the human potential movement or humanistic psychology thrived (Buhler, 1971).

#### **1.01.12.12 Self-help Groups**

The first and most famous of these groups, Alcoholics Anonymous (AA), was founded in 1935 by “Bill” Wilson and Bob Smith. To this day, persons who attend AA receive social support for their sobriety and turn their attention away from themselves toward helping others. AA was from the first a religiously oriented group, in a sense an offshoot of Frank Buchman’s Oxford Group within charismatic, evangelical Christianity. Participants were thus encouraged to confess their allegiance to a “higher power” and their inability to overcome their difficulties without spiritual help. There is, however, a nonreligious alternative approach known as Rational Recovery. Treatment for substance abuse has been a rather separate enterprise from that of traditional psychiatry or psychology. Many of the therapists involved in such work are themselves former alcoholics, and the ideology of AA is strong among them.

#### **1.01.12.13 Marriage and Family Therapy**

One pioneer in the development of family therapy was psychologist John Elderkin Bell. According to Bell’s own recall of events (personal communication, Iowa City, Iowa, early 1980s), he read in a journal in the 1950s some accounts of the work of colleagues in England who were seeing parents and children in therapy, which he mistakenly interpreted to mean seeing them together, or what is now labeled “conjointly.” Thus he began doing this in his own practice, only to discover that he had evidently been the first in the world to do so. He published a book on this topic (Bell, 1961).

The field of marriage therapy grew up as one that was rather separate from the mainstream of either psychiatry or psychology. Many social workers had a special interest in this domain. Gradually, psychologists began to be interested in working with married couples. Jacobson (1989) was a pioneer in carrying out controlled research on behavioral marital therapy.

#### **1.01.12.14 Community Interventions and Prevention**

Within both psychiatry and psychology after World War II, some were interested in moving beyond interventions with individuals or small groups. Their desire was to go out into the neighborhoods and the cities to make larger changes. Psychologist Seymour B. Sarason is an example of such an individual. He and his colleagues set up a "psychoeducational clinic" at Yale, not as a place for the evaluation and treatment of individual school children but to exert an impact on the whole school system and on the city. When asked about his motives for such activities, Sarason mentioned the facts that he was Jewish, that he had polio as a child, and that as a youth he had been an adherent of Trotsky. Thus, he was disposed to view problems such as discrimination and economic injustice as community matters rather than simply individual ones.

In England, psychologist Jack Tizard (1919–1979) developed a sort of action research program to move adults from institutions for the retarded out into the community. He and his colleague Neil O'Connor (1917–1997) demonstrated that with suitable instruction these individuals could perform as well as other workers employed in competitive jobs (O'Connor & Tizard, 1956). In part because of research of this kind, deinstitutionalization eventually went from a trickle to a flood. The use of "job coaches" to help handicapped persons adapt to competitive employment is now widespread.

Psychologist George Albee (1968) engaged in extensive research on the cost effectiveness of mental health treatments and the number of personnel required to deliver them. He came to the conclusion that there were simply never going to be enough therapists to do the job. To him, prevention was the only answer. Albee (1970) proceeded to make himself very unpopular with his colleagues by speaking and writing about "the uncertain future of clinical psychology" (he thought the same thing about the future of psychiatry). He suggested on a later occasion that since its clientele mostly consists of relatively well-to-do persons, insurance coverage of psychotherapy might represent a subsidy to the rich from the poor (Albee, 1979).

#### **1.01.12.15 Special Education**

The physician and educator Maria Montessori (1870–1952) in Italy began her work by familiarizing herself deeply with the writings of J. M. G. Itard and Edouard Seguin and translating their works into Italian (Kraemer, 1988). She developed educational techniques for young children with delayed development living in the slums of Rome that used structured play-like tasks to engage children's natural attention. As things turned out, her approaches came into common use throughout the world in private preschools for well-to-do children with no particular developmental delays.

Psychiatrist Samuel T. Orton (1937) not only came up with neurological hypotheses about the origins of reading disability in children but also, together with colleagues in education such as Anna Gillingham and Bessie Stillman, devised remedial techniques involving intensive drills on analyzing words into sounds and learning letter-sound associations.

#### **1.01.12.16 Rehabilitation**

The psychologist Shepherd Ivory Franz (1874–1933) was presented with an honorary M.D. by medical colleagues at George Washington University for his research contributions. He was also made an honorary member of the American Psychiatric Association. He was an early advocate of rehabilitation for patients with neuropsychological disorders (e.g., speech training for aphasics) as well as for those with severe psychopathology.

Psychologist Marc Gold (1939–1982) was responsible for developing innovative approaches to teaching specific vocational skills to persons with mental handicaps. For example, Gold carried out a task analysis specifying the details of how to put bicycle coaster brakes together and showed that adults with mental retardation could learn to master it. For those of us who as school children had the experience of taking our bicycles apart and unsuccessfully trying to put them back together, Gold's accomplishment is most impressive.

#### **1.01.12.17 Drug Therapies**

In terms of developing medications for treating mental disorders and understanding how they work, a crucial discovery was of the existence of neurotransmitters, the chemical substances that mediate the synaptic transmission of neural impulses. According to Shorter (1997), the first neurotransmitter to be discovered was acetylcholine by Otto Loewi in 1926. By the mid-1990s, over 40 neurotransmitters had been identified.

The use of neuroleptic drugs for psychotic patients beginning in the early 1950s greatly reduced the census of mental hospitals worldwide. Reisman (1991), for example, reports that the US mental hospital population went down from about 600 000 in the 1940s to about 150 000 in the 1970s. Shorter (1997, p. 248) gives some details of how this all happened, as follows: Henri Laborit, who was a surgeon in the French navy, in 1951 developed the drug chlorpromazine. The chemist who synthesized it was Paul Charpentier. At the time, Laborit was looking for something to potentiate anesthetics. He noted that surgical patients given this drug became rather indifferent to the world. By 1952 the drug began to be used in Paris (Delay, Deniker, & Harl, 1952) and by May 1953 had transformed the disturbed wards there. The same kind of thing then happened in Montreal, London, and Boston later the same year. Psychiatrist Heinz E. Lehmann, who introduced chlorpromazine to Montreal's Verdun Protestant Hospital in 1953 (Lehmann & Hannahan 1954; Lehmann, 1989), of the first to note that the drug had some side effects, some known as extrapyramidal or Parkinsonian, and others subsequently described by the term tardive dyskinesia. The Parkinsonian side effects were temporary and readily treatable with other medications, but this was not so for the dyskinesia, which were sometimes enduring ones.

With regard to the use of lithium in mania, Shorter (1997) gives the following account: in 1949, John Cade, the superintendent of a mental hospital in Australia, injected urine from manic patients and from controls into guinea pigs. The guinea pigs died. To try to make the urine more soluble, Cade mixed lithium with it. On a whim, he decided to inject lithium alone into the guinea pigs. To his surprise, instead of the usual frantic struggling, they lay there placidly. He tried injecting himself with lithium and then injected several mental patients, including 10 with mania, six with schizophrenia, and three with depression. Interestingly, the manic patients tended to improve. It so happened that the *Journal of the American Medical Association* reported in the same year two deaths associated with attempted lithium therapy, so the research community understandably became very cautious about the topic. Nevertheless, Mogens Schou, a Danish psychiatrist, confirmed Cade's results, using a double-blind crossover research design (Schou et al., 1954). It was not until 1970 that the Food and Drug Administration (FDA) in the United States approved the use of lithium, and then only in response to the threat of civil disobedience by an Oregon psychiatrist. Now lithium is in common use with manic patients,

though with caution due to its known adverse side effects.

In 1970 psychiatrist Jules Angst (director of the Burgholzli Hospital in Zurich) and his colleagues demonstrated that lithium had similar prophylactic effects in unipolar and bipolar depressed individuals (Angst, Weis, Grof, Baastrup, & Schou, 1970). In other words, lithium has effects on depression as well as on mania.

According to Shorter (1997), meprobamate was first synthesized in 1950 by Bernie Ludwig, an organic chemist for Carter Products, with the consultation of physician Frank Berger. It was tested as an anti-anxiety drug and marketed by Wallace Labs in 1955 under the labels of "Miltown" and "Equanil." It became the first psychiatric drug to become a popular fad. The comic writer S. J. Perelman in 1957 published a book with the title, *The road to Miltown*. Comedian Milton Berle began referring to himself as "Miltown Berle."

Antidepressants were the next medications to emerge, in a somewhat circuitous way. Selikoff, Robitzek, and Ornstein (1952) published a report that iproniazid (Marsilid), a drug used in the treatment of tuberculosis, seemed to have some antidepressant properties. A related drug, named Tofranil, was then developed, tested, and marketed by Geigy (Kuhn, 1957). It was the first of the tricyclic antidepressants, for which 10 million prescriptions were written in 1980 in the United States alone. Swiss psychiatrist Roland Kuhn, who was responsible for this work, had undergone a training analysis under Ludwig Binswanger but later fell away from psychoanalysis (Shorter, 1997, pp. 258–262).

The advent of the benzodiazepines in the 1950s and 1960s is very much a commercially motivated story. Once more, I will depend on the account given by Shorter (pp. 316–319). In 1954, the Hoffman-LaRoche company wished to develop a sedative or anxiolytic drug that would be able to compete with Miltown and Equanil. The chemist involved was Leo Sternbach, and in 1955 he synthesized Librium. Then in 1959 he came up with diazepam (Valium), which was marketed in 1963. Even though these drugs were placed on Schedule IV by the FDA because of their addictive potential, benzodiazepines accounted for about half the prescriptions written for psychiatric office patients by 1990. Later in the 1990s the hottest drug in this group was alprazolam (Xanax). Shorter (1997, p. 320) describes panic disorder (for which this drug was often prescribed) as "the Upjohn illness."

By the 1970s, drug therapy had become quite standard for many types of psychopathology. In 1982, Rafael Osheroff, a 42-year-old physician



who had been a patient at Chestnut Lodge in Maryland in 1979, sued the hospital for malpractice because he had been treated only by means of psychotherapy, not medications. The case was settled in his favor by an arbitration panel (Klerman, 1990). This case was noteworthy also because of the fact that Chestnut Lodge was so well known for the use of psychoanalytic treatment for psychosis.

In the 1950s and 1960s child psychiatrist Leon Eisenberg and psychologist C. Keith Conners began to carry out placebo controlled double-blind studies of the effects of stimulant medications on the behavior of disturbed children (e.g., Conners & Eisenberg, 1963). This research supported the efficacy of such medications. The clinical use of Ritalin (methylphenidate) with behavior problem children increased accordingly, at least in the United States. British and other European psychiatrists did not quickly follow suit, however. It is estimated that over two million children in the United States receive Ritalin at present (Shorter, 1997, p. 290).

In France, Lambert (1966) carried out research demonstrating the antimanic properties of valproic acid, which had originally been synthesized as an organic solvent in 1882 and in 1963 discovered, more or less by accident, to act as an anticonvulsant.

In the 1980s, it was discovered that clomipramine (Anafranil) caused improvement in the symptoms of obsessive-compulsive disorder (OCD) in children as well as adults (Rapaport, 1988). This disorder had previously proven quite refractory to treatment by any method, whether psychoanalytic, behavioral, or medical.

A syndrome involving motor and verbal tics, often including curse words, was described by the nineteenth century French physician Charcot and named after his colleague, Georges Gilles de la Tourette (1857–1904), who had been the first to report such a case. Tourette syndrome (as it has come to be called) was rediscovered by psychiatrist E. Shapiro, who found that the tics could be controlled by the drug haloperidol (Haldol), a neuroleptic (Shapiro et al., 1989).

At the time of writing, the biggest story in medications for psychopathology is that of Prozac (fluoxetine) and the general class of drugs of which it is a part, selective serotonin reuptake inhibitors (SSRIs). In the 1990s, Prozac was the drug most commonly prescribed by psychiatrists. By 1992 it was the second most frequently prescribed drug in the world. It is typically employed as an antidepressant. Once more we will depend on the account of Shorter (1997), who even included Prozac in the subtitle of his book on the history of psychiatry. The development of Prozac had its origins in work

with 5-hydroxytryptophan (5HT), or serotonin, in the 1950s. In 1957 Bernard Brodie at the National Institutes of Health discovered that reserpine could greatly reduce bodily stores of 5HT. Alec Coppen at the Medical Research Council in Britain in 1963 showed that drugs which were serotonin equivalents could relieve depression. It was Ray Fuller, a senior pharmacologist at Eli Lilly in Indianapolis, who synthesized Prozac (Lilly 110140), working with biochemist David Wong. By 1980 the field testing of this new drug was underway, and in 1987 it was released to the public. Shorter notes that by 1993 almost half of the patient visits to psychiatrists were for mood disorders and typically included a prescription for Prozac.

#### **1.01.12.18 Psychosurgery**

The Portuguese physician Egas Moniz (1874–1955) and a surgical colleague developed techniques for lobotomy, in which neural fibers connecting part of the frontal lobes to the rest of the brain were severed (Moniz, 1937). He won a Nobel prize for his work, but this type of surgery is now rarely used as a treatment for psychopathology. In the peak year of 1949, over 5000 lobotomies were performed on mental patients. In the same year, however, controlled research found that the rate of favorable changes did not differ between patients with frontal topectomies and controls (Mettler & Columbi-Greystone Associates, 1949). The frequency of psychosurgery declined greatly thereafter.

#### **1.01.12.19 Electroconvulsive Shock Treatment**

Electroconvulsive shock treatment (ECT) was developed by psychiatrists Lucio Bini and Ugo Cerletti (1877–1963) in the 1930s. In initial experiments on dogs, placing the electrodes on the animal's mouth and anus, the investigators found that about half of the subjects died. Later they discovered that if the electrodes were placed on the dog's temples, electric current could be delivered safely (Shorter, 1997, pp. 218–219). Next, they tried the same thing on pigs at a Roman slaughterhouse. Finally, on April 18, 1938, they tried ECT on the first human patient (Cerletti & Bini, 1938). The clinical use of ECT spread rapidly to England, France, and the United States. This therapy is still in use, especially for the treatment of severe depression.

#### **1.01.12.20 Other Biological Treatments and Preventive Measures**

In 1910 Paul Ehrlich developed an arsenical compound later called Salvarsan that proved to

be an effective treatment of syphilis. This received a mixed reception but by about six years later had been made the basis of a government-supported public health intervention in Britain (Ross & Tomkins, 1997). Surely the most unusual treatment for psychopathology so far developed was that of treating neurosyphilis by infecting the patients with malaria—the fever had the requisite antibiotic effect. This treatment was used by the psychiatrist Julius Wagner-Jauregg (1857–1940), a professor at the University of Vienna. In fact, he won a Nobel prize for this work in 1927. Now there are safer antibiotics available to treat syphilis.

Interestingly, Wagner-Jauregg had also been responsible previously for encouraging the Viennese government to attempt to prevent cretinism and other hyperthyroid conditions by requiring that salt be iodized (Wagner-Jauregg, 1894), an effective public health measure that is now in wide use (Whitrow, 1993).

One of the most curious episodes in the medical treatment of mental disorders was the one involving extracting all the teeth of patients with psychoses, on the theory that focal infections were maintaining their disturbed mental condition. This approach was carried out by Cotton (1922) and was endorsed for a time by prominent figures in the field, including psychiatrist Adolf Meyer. This proved to be nothing more than an unfortunate fad.

Insulin coma was introduced as a treatment for schizophrenia by psychiatrist Manfred Sakel (1900–1957) in Berlin in 1933. The rationale for this treatment was the notion, later considered quite in error, that there was some incompatibility between schizophrenia and seizures. Thus, the reasoning was that if one induced seizures, perhaps these could interrupt the psychosis. The use of insulin coma was discontinued after about two decades in favor of ECT, which was much more reliable in its production of seizures, without so many unwanted side effects, and even ECT was generally used in treating depression, not schizophrenia.

Metrazol (Cardiazol) convulsive therapy for schizophrenia was introduced in 1934 by Ladislaus von Meduna (1896–1964). The rationale for it was similar to that for insulin coma therapy. Meduna was trained as a neuropathologist and had done research comparing the brains of persons with epilepsy to the brains of those with schizophrenia. Thus, he bore major responsibility for the erroneous hypothesis elaborated above about the incompatibility between these two conditions. Meduna published an article on this new treatment in 1935 and a book on it in 1937. Metrazol therapy was also superseded by ECT.

Karl Landsteiner (1868–1948) and A. S. Wiener published their first paper on the Rh factor in human blood in 1940 (Wiener, 1952). This work ultimately led to the ability to prevent fetal malformations and mental retardation due to Rh factor incompatibility in mother and infant (Zimmerman, 1973).

Pediatricians Harry M. Meyer, Jr., chief of the Laboratory of Viral Immunology at the National Institutes of Health (NIH) in Bethesda, Maryland, and Paul D. Parkman developed a rubella vaccine that proved to be an effective preventive measure. It undoubtedly prevented a large number of fetal malformations, stillbirths, and cases of mental retardation, deafness, and other severe disabilities.

Finally, pediatricians Porter W. Anderson, Jr. and David Smith, together with immunologists John Robins and Rachel Schneerson, developed a vaccine against *Hemophilus influenzae* type b (Hib), the agent causing bacterial meningitis and responsible for death, deafness, and mental retardation among children (Holden, 1996).

### 1.01.13 EPILOGUE

This chapter was begun in search of the forerunners of present day psychiatry and clinical psychology. It was acknowledged in Section 1.01.1 that Hippocrates could not really be considered a psychiatrist. Perhaps he is best regarded as the ancestor of the many physicians who have to deal not only with physical illness but also with the emotional and behavioral problems that commonly present themselves in medical practice. Primary care physicians to this day provide reassurance, dispense advice, and prescribe medications for many such patients and are responsible for the care of those who are not referred to mental health specialists. Such physicians are the mainstay of the managed care system in the United States and of the national health systems of many other countries.

Similarly, Democritus was hardly a clinical psychologist. In his role as a scientist trying to investigate the fundamental causes of mental disorder, he could perhaps be most aptly viewed as the ancestor of persons such as Hideo Noguchi, the bacteriologist working at the Rockefeller Institute. There he discovered syphilis spirochetes in the brains of paretics.

The actual specialty of psychiatry did not develop until the late eighteenth and early nineteenth centuries. A transition took place early in the field in which the role of such physicians changed from that of simply restraining troublesome patients to engaging in “moral treatment” of them. Only a few of the

early psychiatrists, such as Esquirol, Griesinger, and Kraepelin, were able to combine their clinical activities with those of a university professor and scientist. During the late nineteenth and early twentieth centuries, psychiatry expanded its scope of practice and for the most part superseded neurology in the care of outpatients with milder functional complaints. Psychiatry was then almost taken over by psychoanalysis, especially in the United States in the era preceding and immediately following World War II. Beginning in the 1950s, psychiatry was revolutionized by the development of psychotropic drugs, first the neuroleptics and subsequently the anxiolytics, antidepressants, and antimanic medications. It is currently undergoing some contraction of its activities due to managed care. Primary care physicians are taking over an increasing amount of the responsibility for the medical management of mental disorders, and other mental health practitioners are carrying out more of the psychotherapy.

Clinical psychology had its origins toward the end of the nineteenth century, with roots in the work of Pierre Janet and others in France and in Witmer's psychology clinic in Philadelphia. Its early development was much influenced by Binet's intelligence test and certainly by psychoanalysis, although psychologists for several decades had many barriers to overcome if they wanted to become psychotherapists. By the time of World War II, clinical psychologists were known primarily as mental testers, and for a time were well accepted into the "clinical team" (e.g., Menninger, 1950). Gradually in the postwar era psychologists became more active as psychotherapists in private practice. They were prominent in the development of behavioral therapy and behavior analysis and were responsible for the origins of formal research on psychotherapy. Along with their colleagues in various academic specialties within psychology, clinical psychologists such as Paul Meehl and Hans Eysenck also contributed significantly to research on psychopathology. Having fought their battles to become psychotherapists, clinical psychologists then saw much of this role usurped in turn by social workers and master's-level counselors.

At present, it seems that one likely scenario for both psychiatry and clinical psychology might be provided by what happened to neurology at the end of the nineteenth century. Neurology continued to be an essential specialty in terms of research, teaching, and specialized consultation. Indeed, in this "decade of the brain" the neurosciences have become a major scientific frontier, attracting luminaries such as Francis Crick.

But after the 1890s, neurology tended to lose its "market share" in the everyday care of persons with functional nervous ailments. Similarly, psychiatry today remains an essential discipline in terms of its knowledge about areas such as psychopathology and psychopharmacology. Clinical psychology has comparable expertise in fields such as psychometrics and behavioral approaches to treatment. One cannot imagine that either of these fields will disappear, but it is likely for the moment that much of the everyday clinical work will be carried out by professionals with more modest levels of training.

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# 1.02

## The Scientific Status of Clinical Psychology

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Psychologists work to develop a valid and reliable body of scientific knowledge based on research. (from the Preamble to the Ethical Principles of Psychologists and Code of Conduct; American Psychological Association, 1992)

### 1.02.1 INTRODUCTION

Psychology grew out of the fields of philosophy and experimental physiology (Boring, 1950) and is typically described as the scientific study of behavior (Sternberg, 1995; Zimbardo & Gerrig, 1996). This includes both animal and human behavior with the term “behavior” typically so broadly defined as to include physiological acts, overt behaviors, and thoughts. While clinical psychology can be considered as

the applied discipline of psychology that focuses on the application of psychological principles to adjustment problems of individuals (Rotter, 1971; Shakow, 1976), the domain of clinical psychology includes “assessment, diagnosis, and treatment of clinical dysfunction; issues in the application of treatment to individuals or society at large; factors that influence adjustment and functioning in everyday life; development across the life span; personality; the family; delivery of psychological services; training of psychologists; and ethical and professional issues” (Kazdin, 1994, p. 1). While science plays a role in each of these areas, this chapter will focus on the scientific status of clinical psychology with respect to assessment and diagnosis, clinical interventions, and clinical training.

From its inception, clinical psychology has been described both as a science and a profession (Peterson, 1976). The flagship journal of clinical psychology, *Clinical Psychology: Science and Practice*, has chosen a name which highlights the dual identity of clinical psychology. Likewise, many of the activities of clinical psychologists do not fit our historical conceptualizations of science. For example, clinical psychologists are less likely to be engaged in laboratory-based “bench” research, rather they are more likely to serve as a consultant to an individual or agency to evaluate and develop a treatment plan relevant to a problem presented by an individual or group of individuals (family, couple, or other system). Depending on the results of the evaluation and plan, the clinical psychologist might also be engaged in the delivery of an intervention (psychotherapy) that has potential for reducing the distress identified by the individual or group being evaluated.

This chapter has several goals. First, it will identify the similarities, while acknowledging the differences, of the science and practice roles in clinical psychology. These differences will be focused on through the discussion of the different roles of clinical psychologists (science and practice) with an emphasis on why training in the science and practice of clinical psychology is critical for these dual roles. To accomplish this goal, a brief review of the history of clinical psychology is presented, followed by a discussion of the philosophy and definition of science. Concepts from behavioral psychology will be presented which will emphasize the role of different types of learning experiences and the importance of the scientist–practitioner training model on the behavior of the clinical psychologist primarily engaged in (i) clinical research—the clinical scientist or (ii) clinical practice—the clinical practitioner. These behavioral concepts will be used to reframe the “scientist” vs. “practitioner” debate in terms of the need to maintain a science-based practice of clinical psychology.

Second, by focusing on the similarities and contributions that clinical scientists and clinical practitioners make to the field of clinical psychology, it will be shown that the important question is not whether or not clinical psychology is a science but, rather, the question is, “What is the current status of science in clinical psychology?” This question will be addressed through a brief discussion of the current scientific status of diagnosis, assessment, and intervention.

Finally, changes in the health care system are likely to influence, or be influenced by, the science-based practice of clinical psychology. Potential issues and change will be discussed in

terms of public policy and the science of clinical psychology.

### **1.02.2 BRIEF HISTORY OF CLINICAL PSYCHOLOGY IN THE UNITED STATES**

Historically, the differentiation of psychology from philosophy was the emphasis which psychology placed on science and the scientific method (Sokal, 1992). However, there was little agreement as to which aspects of science were most important. Specifically, two distinct types of science emerged: (i) laboratory-based activities more in line with experimental physiology, and (ii) applied research activities which focused on individual differences and mental measurement (Goodwin, 1983). This division has been identified as a foreshadowing of the science–practice split in psychology through an emphasis of the difference between experimental and applied psychology (Rice, 1997).

Clinical psychology as a specialized area of training began with the establishment of the first psychology clinic by Lightner Witmer in 1886 (Brems, Thevenin, & Routh, 1991; Watson, 1953). Following World War I, many of the psychologists interested in the measurement of individual differences began to consult with organizations outside academia. This led to the first attempt to develop a professional identity complete with accreditation procedures for “consulting” psychologists. Although this movement for accreditation was not supported by the American Psychological Association (APA) Council (Wallin, 1960), consulting was viewed by the APA leadership as a form of applied research with all the components of an emerging professional identity (Rice, 1997).

By 1919 the clinical section of the APA was formed (Edelstein & Brasted, 1983), and several position papers (cf., Poffenberger, 1938; Rogers, 1939; Shakow, 1938) appeared that delineated the distinct requirements necessary to become a “clinical psychologist” (Derner, 1965; Edelstein & Brasted, 1983; Norton-Ford, 1982; Reisman, 1981).

World War II served to provide much of the emphasis for the emergence of the profession of psychology. First, the APA developed new bylaws which gave recognition to the science and profession of psychology (Capshaw & Hilgard, 1992). Second, the Veterans Administration sponsored a large program to train clinical psychologists. This program incorporated university-based training in psychological theory with clinical practice training which included therapy in addition to psychological assessment. This model cumulated in the Boulder Conference (Raimy, 1950).

Since that time numerous national conferences have been held to address these issues (e.g., Stanford, Strother, 1956; Miami Beach, Roe, Custard, Moore, Ross, & Skodak, 1959; Hoch, Ross, & Skodak, 1966; Vail, Korman, 1976; West Virginia, Foster, Berler, & Collins, 1982; Gainesville, Belar & Perry, 1992; Ann Arbor, Belar et al., 1993; to name but a few), which resulted in the training guidelines followed today.

The early conferences (Boulder, Stanford, Miami Beach, Chicago, and Vail) focused on clinical training in general, with an emphasis on a broad-based background in psychology. Recommendations resulting from these conferences led to specific evaluation criteria and processes by which the APA accredits clinical training programs. Accreditation by the APA is presumed to be documentation that students receive certain minimum training experiences believed necessary to ensure that they have been appropriately trained as clinical psychologists. Each of these conferences reaffirmed the need for training in both the science and practice of clinical psychology.

The West Virginia Conference focused on the essential ingredients for training psychologists in a single school of thought, behavioral psychology. As an organizer of this meeting, I was sure that this “working conference” would specify objective criteria that would be necessary and sufficient for training in clinical psychology. Much to my surprise there was absolutely no consensus among this group of clinical psychologists, all who were engaged in behavioral training. “Indeed, after 6 hours of discussion among a group of prominent clinical directors, the only critical component of behavioral training that was unanimously endorsed was training in an empirical approach to the study of behavior” (Collins, Foster, & Berler, 1986, p. 304).

This emphasis on the importance of science training was the primary theme of the National Conference on Scientist–Practitioner Education and Training for the Professional Practice of Psychology (Belar & Perry, 1992). Recommendations from this conference will be discussed in greater detail later in this chapter; however, it is important to note that, while science plays a major role in the field of clinical psychology, the practice of clinical psychology has not always been integrated with science.

However, many authors (e.g., Rice, 1997; Schneider, 1990) have expressed concern that the division between the science and practice of psychology may lead to an irreversible division in psychology. The goal of this chapter is to show the importance of science in clinical psychology and the need to adhere to a science-based practice of clinical psychology.

First, however, definitions and models will be presented to set the foundation for a discussion of the critical need for a science-based practice of clinical psychology.

### 1.02.3 SCIENCE: DEFINITIONS AND MODELS FOR CLINICAL PSYCHOLOGY

Defining science is not easy. One approach is to identify activities that we can all agree are *not* science (Sullivan & Collins, 1996). For example, most would agree that religion, art, and mysticism are not scientific fields. Physics, chemistry, physics, and biology are easy examples of science fields, and many of us would agree that sociology, anthropology, and psychology are scientific approaches. However, scholars might well engage in the scientific study of religion (theology), music, painting, etc. Are these individuals engaged in “science?” Does science provide unique perspectives to problems of behavior that are different or distinct from the perspectives provided by religion, art, or mysticism? Are there alternative conceptual models that can be used to describe how scientific behaviors develop? Why do scientists do what they do?

#### 1.02.3.1 The Behavior of Scientists

At the most basic level, scientists engage in problem-solving activities that may include observation, conceptualization, and experimentation. Walker (1963) suggests that “the scientific method is a survival technique that developed during the biological evolution of living things. Any organism or device that includes a suitably connected memory unit can “learn by experience,” and this learning by experience contains the basic elements of the scientific method” (p. 15). While the basic elements of the scientific method may account for all learning, contemporary science is a bit more complicated.

Kuhn (1970) offers a somewhat more complex definition of science. For Kuhn, science

means research firmly based upon one or more past scientific achievements, achievements that some particular scientific community acknowledges for a time as supplying the foundation for its further practice. Today such achievements are recounted, though seldom in their original form, by science textbooks, elementary and advanced. These textbooks expound the body of accepted theory, illustrate many or all of its successful applications, and compare these applications with exemplary observations and experiments. (p. 10)

Thus, science is more than merely learning from experience. A science must be based on

information, theory, models, and methods that guide the actions of individuals engaged in science. In addition, knowledge about the behavior of the individual scientist can provide a more complete understanding of science. Sidman (1994) provides an interesting example of the importance of knowing about the scientist and the effect of his or her experiences on the development of theories and models. In the epilogue, he provides excerpts of correspondence between himself and Willard Day. This dialogue provides an interesting perspective of the influence that Day had on Sidman's approach to science.

The behavior of scientists can be broken down into conceptual or decision-making behaviors and experimental method behaviors. The conceptual process used in science is the hypothetico-deductive method (Walker, 1963). This method is a combination of induction, inspiration, and deduction (Walker). Using induction and inspiration, scientists postulate models or theories (Ackermann, 1970). From these models or theories, he or she can then deduce predictions or hypotheses which can then be verified by further observation.

Experimental methods include (i) identification of measures of interest, (ii) developing the methodology to conduct the measurements, and (iii) developing predictive models for analyzing the measures. Identification of measures has historically required specific definitions as to the operations used in measuring the quantity. For example, the weight of an object is the number read from a scale when the object is placed on the scale. The term *operational definition* was coined by P. W. Bridgman, who felt that it was the hallmark of science (Walker, 1963). While it has been argued that concepts which cannot be adequately defined using operations are not in the realm of science (Walker), most philosophers today have abandoned the doctrine of operational or explicit definition (Suppe, 1977).

This is an important point. Technological advances such as the invention of a more powerful microscope can change science by allowing for the operationalization of concepts that were previously seen as "not subject to empirical validations," or for the development of new operationalizations of a concept that may result in different outcomes. Concepts once thought to be unmeasurable become measurable with increased technology. Thus, the operationalization of a concept is quite arbitrary.

This historical focus on operationalization is perhaps one factor that resulted in the perception that science is value-free or a measure of the "truth." Today, there is general agreement that science, and the results of science, are influenced by the beliefs, expectations, and values of the researcher and the research community he or

she belongs to for both science in general (Habermas, 1975; Holton, 1973; Polanyi, 1958) and for the science of psychology (Campbell, 1984; Fishman, 1988; Gergen, 1985; Howard, 1985; Koch, 1976, 1981; Krasner & Houts, 1984; Scarr, 1985). At the extreme, science is seen as nothing more than a construction of the human mind. As Scarr put it, "Facts do not have an independent existence" (p. 499). While total discussion of this issue is beyond the scope of this chapter, some general comments are necessary.

The belief that researchers are neutral, value-free truth seekers is probably based on the philosophical principle called logical positivism. Logical positivism is based on two assumptions: "(1) there is an external world independent of human experience, and (2) objective knowledge about this world can be obtained through direct sense experience" (Fishman, 1985, p. 5). Thus, logical positivism suggests that all observations are the ultimate forms of truth since observations are objective (Staats, 1988).

An alternative to logical positivism has been labeled by Gergen (1985) as social constructionism. This model suggests that all knowledge, including scientific knowledge, is a construction of the mind:

Sensory data are filtered through the knowing apparatus of the human senses and made into perceptions and cognitions. The human mind is also constructed in a social context, and its knowledge is in part created by the social and cultural world. Knowledge of the world is therefore always constructed by the human mind in the working models of reality in the sciences. If this is not evident, consider for a moment the vast differences in our concepts of the world before Galileo, Darwin, Einstein, and Freud. (p. 499)

While there is no doubt that data are a product of the observed and the observer, science can, and will, continue to make major contributions to our understanding (no matter how limited it may be) of behavior.

Hayes (1995) offers an alternative definition of science:

Science is a human endeavor that has as its purpose the development of increasingly integrated systems of verbal rules that allow us to accomplish analytic goals with precision, scope, and depth, based on verifiable experience . . . Science is a special kind of word-producing, rule-making enterprise. (p. 52)

Hayes's definition is consistent with Kuhn (1970) in that both see science as the accumulation of information designed towards a better understanding of the world. Unlike other perspectives, however, Hayes suggests that there

are aspects of the world that can be studied and measured which are beyond the scope of rule-governed science.

### 1.02.3.2 Science vs. Art

As mentioned previously, science must be understood in terms of both the factual theories and method *and* the behavioral experiences of the scientist. Facts are more likely to be learned from reading and studying a subject but experiences are a critical part of any learning endeavor.

It is widely accepted that behavior is influenced by rules and experiences (Hayes, 1989; Hayes, Zettle, & Rosenfarb, 1989). These different influences are often labeled as “rule-governed behavior” (Hayes, 1989, 1995) and “contingency-shaped behavior” (Hayes, 1995). Another way of expressing these ideas might be the difference between what is often thought of as didactic “academic” information vs. practical “experience.” In clinical psychology, this translates roughly into what one learns from reading the literature or going to class compared with what one learns from supervised clinical and research opportunities.

There are important differences between those behaviors acquired through rule-governed processes and behavior acquired via contingency-shaping:

Rule-governed behavior tends to be somewhat more rigid, less modifiable by its direct consequences, more precise in its initial forms, and more subject to arbitrary social contingencies. Contingency-shaped behavior is generally more moldable and modifiable, but it is also more variable, and subject to chance contingencies. (Hayes, 1995, p. 52)

Science focuses primarily on rule-governed behaviors. However, as Hayes (1995) points out, while “science is the best rule-generating institution ever invented, it is wrong to think that all behavior can be *directly* rule-governed . . . Any instance of rule-governed behavior stands ultimately on contingency-shaped behavior, at least to a degree” (pp. 52–53). Thus, experience plays a critical role in science.

An example might be helpful. Articulation of rules that govern behavior is the first step in the development of science and critical to the understanding of the status of a scientific discipline. If we were to develop a science of baseball, we might first want to articulate rules to describe how a person should swing a bat to hit a ball approaching at speeds of nearly 140 kilometers per hour. The rules might include a discussion on stance, placement of the bat, choice of the type of bat, etc. This verbal set of

descriptors might be quite elaborate and, combined with some video clips of someone engaged in “batting,” we may have the fundamentals for the science of baseball. However, this “science of baseball” will never be an exact replica of baseball experienced by the professional, unless it incorporates the contingency-shaped skills learned through the interaction of the ballplayer and his or her experiences at playing ball.

Likewise, an understanding of the science of clinical psychology requires an understanding of the known rules of clinical psychology, combined with experiences that allow contingency-shaped learning. While the study of science involves both components, it is difficult to document those critical experiential aspects of science training. Most often we focus on issues such as mentoring and directed research as a mechanism to acquire contingency-shaped aspects of science.

There has been a push to evaluate the critical importance of hands-on training in the laboratory for teaching science. For example, Ertepinar and Geban (1996) demonstrated that the addition of a laboratory-based method which focuses on conducting science studies resulted in a greater mastery of science knowledge than merely providing students with science information. Thus, it appears that active involvement in behaviors relevant to science may be an important part of science training.

### 1.02.3.3 Clinical Scientists and Clinical Practitioners

As mentioned previously, clinical psychologists engage in many different activities with two of the most identifiable being the science and practice roles. Psychologists who emphasize clinical research can be identified as clinical scientists, while individuals who emphasize clinical practice are often described as clinical practitioners. While it is clear that these roles are arbitrary and that science and practice often go hand in hand, it is also clear that some training programs emphasize training for clinical science careers and others emphasize training for clinical practice careers. Likewise, many clinical psychologists engage in both scientific and practice careers simultaneously; however, there is much to be gained from differentiating clinical science training from clinical practice training.

Clinical scientists and clinical practitioners need to be well grounded in the knowledge base of clinical psychology (this will be discussed in greater detail in Section 1.02.4). Such training would allow both types of trainees comparable exposure to the rules of the science of clinical

psychology. In addition, basic contingency-shaped experiences in clinical research and clinical practice are critical for both career paths. To become a sound clinical researcher, basic training in clinical practice is critical. Likewise, sound clinical practitioners must have basic experience in clinical research. However, the types of contingency-shaped experiences and the amount of contingency-shaped experiences may be quite different for success as a researcher or practitioner.

The ideal method for gaining contingency-shaped experiences in research would be through supervised research with an experienced mentor who can guide and direct the activity. Such experiences would provide contingency-shaped behaviors critical for science practice in clinical psychology that are unique from the rule-governed science behaviors. These activities are typically formalized in academic training programs in terms of thesis and dissertation requirements; however, successful completion of a dissertation is probably minimal for both clinical scientists and clinical practitioners and more extensive supervised research experiences (such as one or two years of postdoctoral experience with a senior scientist) would enhance the development of contingency-shaped research skills critical for a career as a clinical scientist.

Practice training requires extensive contact with clinical populations. The ideal is supervised clinical work with an experienced professional who can guide and direct the activity. Like research experience, clinical experiences will provide contingency-shaped behaviors critical for the practice of clinical psychology that are unique to the contingency-based research behaviors or the rule-governed aspects of clinical psychology. Academic programs require a minimal amount of clinical practice and a year of full-time clinical training (internship). However, these experiences are minimal basic training. As with research training, postdoctoral experiences in a clinical setting may be critical for the development of sophisticated contingency-shaped practice skills.

At first glance, this may seem to be merely an attempt to redefine the existing model of clinical psychology training using new jargon. However, this redefinition can greatly influence the manner in which science and practice are taught and may provide a useful model for the integration of science and practice. It should come as no surprise that there will be a great deal of variability with respect to contingency-shaped behaviors. By their definition, these experiences are somewhat unique. Unlike rules, where everyone can be taught the same concepts, contingency-shaped behavior will be

much more variable and likely to be influenced by chance contingencies (Hayes, 1995). However, there are certain activities that will enhance training in science or practice. The development of contingency-shaped practice skills can be enhanced through an emphasis on practical training clinical service delivery with a broader range of clients and treatment methods. Science training can be enhanced with an emphasis on practical research training, again with a broader range of methods and problems. While time constraints in a training program will limit the amount of experiential training in both the practice and science aspects of clinical psychology, the time spent engaged in an activity does not guarantee more or better contingency-shaped behavior. It is likely that the quality of the supervised experience (either in research or practice) will have a greater influence on outcomes.

Given that an emphasis on training students for roles as clinical scientists or clinical practitioners may result in unique behavioral repertoires owing to differential rule-governed and contingency-shaped behaviors, it should come as no surprise that both career paths offer unique and important contributions to the science of clinical psychology. A recognition of the different contributions may be critical to our advancement of the science of clinical psychology, but beyond the scope of this chapter. However, several papers (e.g., Clement, 1988, 1996; Davison & Lazarus, 1995; Fensterheim, 1993; Fensterheim & Raw, 1996; Kanfer, 1990; Marten & Heimberg, 1995; Nezu, 1996; Woolfolk & Lazarus, 1979) will provide an overview of this area for interested readers. An understanding of these unique contributions requires better identification of the rule-governed and contingency-shaped aspects of clinical psychology.

Much of the controversy over the science and practice of clinical psychology comes from a failure to recognize the importance of contingency-shaped behaviors and the unique perspective gained from mentored research and mentored clinical training. It has been suggested that if the split between the science and practice of clinical psychology is not resolved, we may see a dissolution between the science and practice of clinical psychology (Rice, 1997). Failure to resolve this split might result in clinical training being removed from academic psychology departments and relegated exclusively to professional schools. In this scenario, Rice has predicted that clinical health psychology would become an allied health profession, rehabilitation psychology would ally itself with occupational therapy, and counseling psychology would evolve into

“spiritual counseling.” Academic departments would see a decline in graduate students and the blending of traditional psychology departments with other academic units such as neuroscience, human development, computer science, and social science departments.

Rice’s (1997) dissolution scenario is by no means the future of psychology, but a probable outcome if science and practice are not emphasized in clinical psychology training. Later in this chapter, Rice’s “science-based practice” scenario will be discussed as the ideal outcome for clinical psychology. First, however, the current status of science in clinical psychology is reviewed with an emphasis on identifiable rule-governed and contingency-shaped aspects of the science of clinical psychology.

## 1.02.4 THE CURRENT STATUS OF SCIENCE IN CLINICAL PSYCHOLOGY

### 1.02.4.1 Diagnosis and Assessment

The publication of the third edition of the *Diagnostic and statistical manual of mental disorders (DSM-III)* (American Psychiatric Association, 1980) changed the manner and approach to diagnosis and classification used in clinical psychology. The system was widely adopted (although not uniformly supported, see Kirk & Kutchins, 1992, for a review) by all mental health professions. *DSM-III* (and its successors *DSM-III-R*, American Psychiatric Association, 1987; *DSM-IV*, American Psychiatric Association, 1994) has become the standard classification system for clinical psychology and is represented in almost every major textbook on the topic (Klerman, 1984).

The shift from *DSM-II* (American Psychiatric Association, 1968) to *DSM-III* was profound. *DSM-II* was heavily influenced by psychodynamic theory and used concepts such as neurosis and psychosis as major classifications. *DSM-III* attempted to develop a more empirical, and to some, more scientific classification. This change had great influences on both the science of clinical psychology and psychiatry. Maxmen (1986) described this shift as:

Psychoanalytic psychiatry bases truth on authority; something is true because Freud said so. Scientific psychiatry bases truth on scientific experimentation . . . The old psychiatry derives from theory, the new psychiatry derives from fact. (p. 31)

Changes in the *DSM* (from the third to the fourth edition) have continued to emphasize

empiricism and science. Categories were changed (or added) only if there were sufficient data to warrant such a change. Symptoms of disorders likewise were modified, only with considerable evidence that the existing criteria were inadequate and that new criteria were more accurate.

While there is a strong scientific emphasis in the *DSM* system, it is not without its critics (cf., Kaplan, 1983; Kirk & Kutchins, 1992; Miller, Bergstrom, Cross, & Grube, 1981; Schacht, 1985; Singerman, 1981). As with any scientific endeavor, there are some assumptions in which science may have appeared to play little or no role. Spitzer (1985) identified several nonscience aspects involved in the development of the *DSM*, two of which will be summarized here. First, the *DSM* is based on a descriptive approach which emphasized classification on the basis of shared clinical features rather than presumed etiology. Science has not shown that descriptive approaches are superior to other approaches. In fact, there is no compelling reason to believe that a similar disorder would not show up in different individuals as different symptoms. The *DSM* deals with these differences by identification of a list of descriptive criteria, and the clinician merely tallies the number of symptoms presented by the client. A diagnosis is given if the number of symptoms meets or exceeds a predetermined criteria.

Second, Spitzer points out that subjective judgments played a major role in the development of the *DSM-III*. As noted in the introduction to *DSM-III*, “most of the diagnostic criteria are based on clinical judgment, and have not yet been fully validated by data.” While the revisions have continued to search for empirical support and less subjective data, clinical judgment continues to play a major role in this diagnostic system.

For the scientist–practitioner and the clinical scientist, the *DSM-III* and its successors provide a significant step towards the development of a strong science. The rules are clearer than other diagnostic systems; however, the system continues to utilize clinical judgments (contingency-shaped behaviors) necessary given the existing knowledge base. As the field develops and more is learned about specific disorders, the *DSM* system allows for correction and changes in the diagnostic rules. It is anticipated that changes will occur and mechanisms have been built in to facilitate and foster change.

The scientific status of psychological assessment is difficult to evaluate. On the one hand, with regard to testing and test development, there are “180 guidelines covering 16 categories pertaining to the activities of test developers,



test users, and test takers” (Cone, 1995, p. 204). However, there is considerable debate as to the best use of tests in clinical psychology (cf., Collins & Thompson, 1993; Cone, 1989; Hayes, Nelson, & Jarrett, 1987, 1989; Haynes & Uchigakiuchi, 1993).

With respect to guidelines, the majority focus on (i) technical standards for test construction and evaluation, such as validity, reliability, norming, etc.; (ii) professional standards for test use, such as employment testing, educational testing, etc.; (iii) standards for particular applications, such as testing of individuals for whom English is a second language, visual impairments, etc.; and (iv) standards for administrative procedures, such as scoring and reporting issues, and the rights of test-takers (Cone, 1995).

While the existing standards are important and useful to the science of psychology, Cone (1995) recommends that new standards be developed to improve the utility of psychological assessment methods. Cone’s detailed rationale for each of these recommendations will not be presented in this chapter; however, the new standards should:

- (1) Recognize that different subject matters might require modifications in standards.
- (2) Make room for the concept of accuracy, and distinguish it from reliability and validity.
- (3) Distinguish between representational and elaborative validity, and require purveyors of new measures to show the former and some evidence of the latter before publishing the measure.
- (4) Require psychologists to evaluate what they do.
- (5) Develop standards dealing with the process of that evaluation.
- (6) Consider specific standards dealing with functional analysis.
- (7) Include standards focusing on idiographic or local use assessment activities. (Cone, 1995, p. 220)

One of the most important steps for improving the science in clinical assessment is to get practicing psychologists to evaluate what they do. While everyone agrees that evaluation is important, it is difficult to get consensus on the systematic application of psychological methods and practices to clinical assessment (Kazdin, 1996a). Clement (1996) offers a model of practice assessment which focuses on diagnosis, treatment plans, compliance with treatment plans, and assessment of treatment outcome. While this plan may be criticized as relying heavily on self-report measures (Nezu, 1996), the spirit of the plan is an important step for improving the scientific status of assessment in clinical psychology (Hayes, 1996; Lambert & Brown, 1996).

Managed health care may directly influence the use of sound clinical assessment strategies (Nelson-Gray, 1996). The current trend is to develop outcome measures applicable to all clients (Nelson-Gray). While the development of uniform measures may be useful, measures unique to diagnostic categories may be far more helpful because of the differences in success rates with different clinical problems (Nelson-Gray). For example, treatment of social phobia may be best evaluated using the Index of Social Phobia Improvement (Turner, Beidel, & Wolf, 1994); depressed individuals might best be evaluated using the Beck Depression Inventory (Beck, Ward, Mendelsohn, Mock, & Erbaugh, 1961), and anxiety disorders might be evaluated using the State-Trait Anxiety Inventory (Spielberger, Gorsuch, & Lushene, 1970). For many situations, however, individualized (or idiographic) assessment methods may be needed (Collins & Thompson, 1993; Nelson-Gray, 1996).

#### **1.02.4.2 Scientific Status of Clinical Assessment**

A great deal is known about the psychometric properties of tests (Cone, 1988). Clinical psychology has developed sophisticated measures of intelligence (Kaufman & Harrison, 1991) and personality (Ben-Porath & Butcher, 1991). In general, we have a fairly sophisticated, sound set of rule-governed behaviors as a foundation for the science of clinical psychology. The use of assessment in clinical settings (contingency-shaped experiences) is less clear cut. Since the mid-1970s, there has been a noticeable decline in emphasis on clinical assessment (Garfield & Kurtz, 1973, 1976; Levitt, 1973; Shemberg & Keeley, 1970).

One factor that has contributed to the decreased use of clinical assessment in the field is that clinicians have not always found these data to be useful in treatment (Adams, 1972; Moore, Boblitt, & Wildman, 1968). The behavioral alternative to traditional assessment, behavioral assessment, was developed in part as a reaction to more traditional assessment approaches (Hersen, 1976). More recently, even among behavioral psychologists, reliance on behavioral observations for assessment has given way to more moderate approaches involving the use of psychometrically sound trait measures (Collins & Thompson, 1993; Gross, 1990; Watkins, Campbell, & McGregor, 1990).

Cone (1988) points out that there are two “contrasting models of behavioral assessment” (p. 46): a nomothetic-trait approach and an idiographic-behavioral approach. The nomothetic approach enables the comparison of an

individual's score on an assessment measure to a normative reference group. The idiographic-behavioral approach focuses on changes over time for a single individual. Idiographic assessment provides the scientist-practitioner with useful methods for increasing the quality of the science in his or her practice (Barlow, Hayes, & Nelson, 1984).

While the integration of idiographic and nomothetic systems is strongly encouraged today, there is little evidence about how to accomplish this integration. Hersen and Bellack (1988) have advocated an "idiographic approach within the normal nomothetic system" (p. 78). Collins and Thompson (1993) suggest that an equally useful alternative is the use of "nomothetic (personality) data [integrated] within the idiographically based behavioral assessment" (p. 69).

Four broad guidelines have been recommended (cf., Collins & Thompson, 1993). First, assessments must use empirically sound measures. There are many unsound measures, frequently tied to particular theoretical constructs or part of "clinical lore." Not only must the assessment measures used have an empirical basis, the "methods must be *administered* and *interpreted* from an empirical data base" (p. 65). Second, assessments derived from specific measures must be confirmed. Most measures are open to multiple interpretations. Third, the behavior of interest must be differentiated from the assessment score. Assessment data are only useful because of their ability to *predict* behavior; however, the assessment data are not the behavior. Finally, assessment data must be collected repeatedly over time. Single measures are rarely useful in clinical settings.

#### 1.02.4.3 Clinical Interventions

Our knowledge about the scientific status of clinical interventions has grown immensely. However, the conclusions drawn from these data are not consistent. On the one hand, there is a lot known about which types of therapy are likely to produce the greatest benefit in some clinical disorders (Kazdin, 1996b); on the other, there is continued evidence that most forms of psychotherapy are useful with little to no differences between the "type" of therapy and success (Seligman, 1995). These somewhat antithetical conclusions have been derived using different scientific methodologies and will be explained in some detail.

By 1980, many psychotherapy researchers (e.g., Bergin & Lambert, 1978; Luborsky, Singer, & Luborsky, 1975; Shapiro & Shapiro, 1982; Smith & Glass, 1977; Smith, Glass, &

Miller, 1980; VandenBos & Pino, 1980; Yates & Newman, 1980) reached the general conclusion that "Psychotherapy, as a generic treatment process, [is] demonstrably more effective than no treatment" (VandenBos, 1986, p. 111). The primary reason for this has to do with what has been called "nonspecific factors" thought to be common to all forms of psychotherapy (cf., Arkowitz, 1995; Beutler, 1995; Elkin, 1995; Frank, 1995; Ilardi & Craighead, 1994; Luborsky, 1995; Weinberger, 1995). The years since 1985 have focused on comparative outcome research. That is, the emphasis was no longer on whether or not psychotherapy works, but what type of psychotherapy works with what disorders. In the mid-1980s, VandenBos, in the introduction to the special issues of the *American Psychologist* on Psychotherapy Research, appeared to encourage such research:

It now appears that single-focus "outcome" (or efficacy) research should be a "thing of the past." Future psychotherapy outcome research should, at a minimum, be "comparative" outcome research—exploring the relative advantages and disadvantages of alternative treatment strategies for patients with different specific psychological and behavioral difficulties. (p. 111)

In 1995, the Task Force on the Promotion and Dissemination of Psychological Procedures published a list which contained "Examples of empirically validated treatments" (Table 3, p. 22). The publication of this list has resulted in immense praise and criticism and, regardless of one's perspective as to the accuracy of the list, the list is important for the evaluation of the scientific status of clinical intervention in psychology.

Many psychotherapy researchers agree with the general conclusions of the Task Force. For example, Seligman (1995) states,

studies show . . . that cognitive therapy, interpersonal therapy, and medications all provide moderate relief from unipolar depressive disorder; that exposure and clomipramine both relieve the symptoms of obsessive-compulsive disorder moderately well but that exposure has more lasting benefits; that cognitive therapy works very well in panic disorder; that systematic desensitization relieves specific phobias; that "applied tension" virtually cures blood and injury phobia; that aversion therapy produces only marginal improvement with sexual offenders; that disulfiram (Antibuse) does not provide lasting relief from alcoholism, that flooding plus medication does better in the treatment of agoraphobia than either alone; and that cognitive therapy provides significant relief of bulimia, outperforming medications alone. (pp. 965–966)

Barlow (1996) adds,

I believe, along with many others, that the evidence is incontrovertible that we have effective psychological interventions for a large number (but not all) of psychological disorders. Numerous studies and subsequent meta-analyses have demonstrated that any number of specific psychotherapeutic approaches, either alone or, in some cases, in combination with pharmacological approaches, are more effective and often longer lasting than credible alternative psychological interventions. (p. 216)

Given these strong endorsements, it may at first seem confusing to learn that some of the more influential psychotherapy researchers (e.g., Garfield, 1996; Havik & VandenBos, 1996; Seligman, 1995; Shapiro, 1996) believe that these research studies are of little importance to the scientific study of psychotherapy.

One of the major criticisms of comparative outcome studies is that the results have little relevance to clinical practice (Garfield, 1996; Havik & VandenBos, 1996; Munoz, Hollon, McGrath, Rehm, & VandenBos, 1994; Seligman, 1995). Participants seen in comparative outcome studies give informed consent to be randomly assigned to different treatment conditions, whereas patients seen in clinical practice do not. Comparative outcome studies frequently use manualized treatment protocols that place guidelines on therapist behaviors and the number of treatment sessions provided, whereas therapists in clinical practice rarely adhere to the type of manualized treatments used in comparative outcome studies (Persons, 1995) and almost never routinely limit the number of sessions.

Seligman (1995) argues that “effectiveness” psychotherapy studies, not comparative outcomes studies, are the best way of “finding out what treatments actually work in the field” (p. 966). To conduct a sound effectiveness study requires allowing a great deal of flexibility. For example, Seligman identifies five “properties that . . . characterize psychotherapy as it is done in the field” (p. 966). These factors will be discussed in some detail as they are critical for evaluation of the scientific status of clinical psychology.

First, psychotherapy cannot be defined in terms of number of sessions. In the field, it continues until there is some improvement or until the patient decides he or she should quit. This is extremely important since the most elaborate effectiveness study conducted to date, the *Consumer Reports* study (1995; Seligman, 1995), found duration of therapy to be positively related to improvement with the greatest improvement reported by individuals in therapy for two or more years.

Second, psychotherapy allows for correction. That is, if a technique or approach is not working for a client, then another approach is typically tried. This issue of self-correction is similar to medical treatments that are discontinued and/or replaced if the desired short-term outcome is not forthcoming. Comparative outcome studies are rarely designed with this in mind. Blanchard and his colleagues (Blanchard et al., 1996) used a self-correcting design in the study of biofeedback and hypertension. Treatment failures were then randomly assigned to an alternative therapy. However, I am not aware of similar designs in the psychotherapy literature.

Third, in the field, patients are often engaged in “active shopping.” They learn about different forms of therapy and actively seek out someone who can provide that type or modality of therapy. In controlled outcome studies, random assignment results in a great deal of passivity on the part of the participant (Howard, Orlinsky, & Lueger, 1994).

Fourth, patients seen in the field often have more than one problem, or the problems that they present with do not fit diagnostic criteria. Many times the symptoms of their problems result in multiple diagnoses. See Lilienfeld, Waldman, and Israel (1994), Rutter (1994), Widiger and Ford-Black (1994) for a discussion of the problems with comorbid diagnosis in *DSM*. Comparative outcome studies, however, tend to focus on individuals with fairly specific inclusion criteria. While this is reasonable in terms of the specific data analytic models used in comparative outcome studies, it still makes generalization to field studies more difficult.

Finally, the end goal of field studies is “with *improvement in the general functioning* of patients, as well as amelioration of a disorder and relief of specific, presenting symptoms” (Seligman 1995, p. 967). Comparative outcome studies focus on specific symptom reduction.

#### 1.02.4.4 Scientific Status of Clinical Interventions

Clearly there is a great deal of information available regarding rule-governed aspects of psychotherapy. Elaborate manuals have been developed to help therapists administer a variety of interventions ranging from relaxation training (e.g., Bernstein & Borkovec, 1973) to cognitive therapy (e.g., Beck, Rush, Shaw & Emery, 1979). Therapist manuals are available to help in the treatment of many disorders (e.g. *Panic and anxiety*, Barlow & Craske, 1994; *Craske, Meadows, & Barlow, 1994*; *Borderline personality disorder*, Linehan, 1993).

The development of manualized therapy has had a significant impact on the science of clinical psychology (Eifert, Schulte, Zvolensky, Lejuez, & Lau, 1997). The operationalization of specific aspects of therapy has contributed significantly to clinical outcome research (Barlow & Craske, 1994; Chambless, 1996; Seligman, 1995); however, the existence of a manual does not guarantee that all therapists will be equally effective, or that they will present the material in the same manner (Chambless). Likewise, manualized therapy does not guarantee that the behaviors identified in the therapy manual are identical to those used in the practice of clinical psychology (Collins & Thompson, 1988).

Most therapy manuals provide some caution that successful implementation of manualized treatment requires skilled therapists. For example, Barlow and Craske (1994) state:

The first revolution in the development of effective psychosocial treatments during the past decade has been the manualization of these treatments. Because these are structured programs for specific disorders, they can be written in sufficient detail to allow trained therapists to administer them in roughly the same manner that they were proven effective. *This does not imply that therapeutic skills are no longer needed.* In fact, in the training tapes...there are numerous examples of the importance of highly trained psychotherapeutic skills as one proceeds with the program. (Chap. 2, p. 6, italics added)

Chambless (1996) concurs but expands on the usefulness of manualized therapy:

I agree that manuals do not completely mirror clinical practice but argue that they have much to teach us if the treatment they depict is efficacious. I find that students learn treatment approaches much more quickly from their systematic depiction in manuals...than through supervision alone. (p. 231)

Using the model of rule-governed/contingency-shaped behaviors as a guide, it is clear that the professional use of treatment manuals will foster the acquisition of rule-governed behaviors. Likewise, clinical supervised practice using efficacious treatment manuals sets the occasion for continued contingency-shaped learning. Several writers (Iwamasa & Orsillo, 1997; Persons, 1995) have suggested that incorporating efficacious treatment manuals into a clinical training program is critical. Persons suggests that the main reason most psychotherapists do not conduct therapy consistent with the outcome literature is that they have received little or no training in the use

of efficacious training manuals. Such training will enhance the scientific status of clinical interventions by directly shaping therapist behavior.

Flexibility in the use of treatment manuals is less clear. Allowing therapists to “freelance” or stray too far from the procedures outlined in the manuals may not be desired (Eifert et al., 1997). For example, Frank, Kupfer, Wagner, McEachran, and Cornes (1991) demonstrated a strong relationship between adherence and positive outcomes. More research is needed, and it is possible that some manualized treatments need to be closely adhered to, while others may allow for substantial therapist flexibility.

### 1.02.5 TRAINING SCIENCE IN CLINICAL PSYCHOLOGY: THE SCIENTIST-PRACTITIONER MODEL

The scientist-practitioner model was first articulated at the Boulder Conference (Raimy, 1950) and continues to be one of the most prominent, if not the primary, model for training clinical psychologists. Rice (1997) sees a “science-based practice” as a likely scenario for psychology as a profession, and in my opinion, the most optimistic scenario for psychology. The Gainesville Conference (Belar & Perry, 1992) articulated the components of this model quite well and a brief summary follows.

First, delegates at the Gainesville Conference “asserted that the scientist-practitioner model was essential for the ever-changing discipline of psychology” (Belar & Perry, 1992, p. 71). While there appears to be room for alternative models of training, this model is critical to the continued scientific development of clinical psychology.

Second, the hallmark of this model is the integrated training of science and practice, not the job title or position of the graduate. This difference is important. The usefulness of this training model cannot be evaluated by counting the number of graduates who publish scholarly work or who are employed as clinical scientists. Rather, training in the scientist-practitioner model should produce professionals who use the scientific knowledge base of clinical psychology in their practice and who approach the problems and questions of clinical practice in a scientific manner.

Third, the conference identified the importance of both didactic (rule-governed) and practical (contingency-shaped) training in the science of psychology and the strategies and tactics of applied work. Didactic science

training includes competence in the major scientific areas of psychology identified as the biological bases of behavior, cognitive-affective bases of behavior, social bases of behavior, and individual differences. Didactic practice training emphasizes “the application of scientific thinking and behavior to problem-solving and hypothesis-testing in practice” (p. 73). Practical training must focus on the integration of science and practice and should include both predissertation research as well as the completion of a dissertation. The doctoral dissertation should provide an opportunity for the trainee to demonstrate “competence to carry out an original, independent scientific investigation that furthers psychological knowledge” (p. 74).

Clearly, the scientist–practitioner training model articulated by Belar and Perry (1992) is consistent with the model of science discussed throughout this chapter. Scientist–practitioner training requires contemporary didactic training in the knowledge base of clinical psychology. Such training should strengthen the acquisition of rule-governed behaviors critical for the science of clinical psychology. Likewise, practical experience is needed. However, such experiences should continue to integrate science knowledge in all aspects of practice. This includes research and clinical work. Thus, research training in clinical psychology must integrate the acquired knowledge of the science of psychology, with supervised experience conducting clinical research. Likewise, clinical practice must integrate the acquired knowledge of the science of psychology, with supervised experience in clinical assessment and psychotherapy.

While the scientist–practitioner model is typically applied to clinical psychology, Rice (1997) sees a future where all professional psychology is science based:

In this scenario, all psychological practitioners, including counseling, school, and industrial/organizational (I/O) psychologists, are trained as science-based practitioners to work in a variety of settings. Most of the persons preparing for the practice attend professional schools, which are typically attached to universities and maintain close relations with academic psychology departments. These professional schools usually offer the PhD rather than the PsyD degree, though there is variation with respect to this.

The professional school curriculum is rigorously and narrowly structured during the first two years; students take course work in cognitive psychology, neuropsychology, psychometric theory, developmental and social psychology, psychopharmacology, and research methods. During the next two years, students take more specialized courses to prepare them for work in various organizational

settings. A two-year specialty internship completes the training. A research dissertation, usually of an applied nature, is required for all completing the PhD. Fewer students are admitted to these programs because of the training, which is rigorous and expensive due to the large number of full-time faculty who teach these basic courses and who engage in a range of applied psychology research ventures.

Academic psychology departments continue the tradition of preparing teachers and researchers. Teaching positions are fairly abundant in both psychology departments and professional schools. Research positions are found outside academia in commercial and governmental settings. The traditional psychology fields such as social, experimental, developmental, and I/O psychology remain, but most departments offer training in health psychology, experimental psychopathology, or both. (pp. 1177–1178)

While some will cringe at the notion that training in applied psychology should occur in a professional school, this scenario is more consistent with current training goals and in no way minimizes the importance of science training. Likewise, much of the conflict between the science and practice of psychology can be minimized through this scenario.

#### **1.02.6 SCIENCE, THE CHANGING HEALTH CARE SYSTEM, AND THE DEMAND FOR PSYCHOLOGY**

Changes in health care appear inevitable (VandenBos, 1993). Two factors will influence the final outcome of this process: (i) cost—all else being equal, less expensive forms of treatment will be preferred; and (ii) effectiveness—for problems with known effective treatment, those treatments shown to be effective will be supported (Barlow, 1994). It is critical, therefore, to develop a strong case for scientific evaluation and implementation of clinical psychology services.

Detailed guidelines are already being developed. For example, the Agency for Health Care Policy and Research, which was commissioned by the federal government to determine the effectiveness of treatments for specific disorders, has published a two-volume document which provides recommendations for treating major depressive disorders (Barlow, 1994). The summarized findings of this evaluation was that medications are the first-line treatment for almost all cases of major depressive disorder. Exceptions to medication are when the depression is mild or when the patient specifically asks for psychosocial treatments (Barlow).

While clinical psychology continues to debate the usefulness of controlled outcome studies, it

is clear that these types of studies are providing the major source for the analysis and ultimate development of public policy. Students in clinical psychology must be trained in the science of clinical psychology and the methods that currently appear most efficacious. It is expected that the specific methods will change (Iwamasa & Orsillo, 1997); however, sound professional science training will allow clinical psychology to adapt and develop.

One often overlooked aspect of clinical psychology is the extent to which practice activities are the exclusive domain of clinical psychology, or also in the domain of other health care providers. Clinical psychology as a discipline requires the doctoral degree for entry into the profession; however, many of the professional activities historically identified with clinical psychology are today provided by other professionals, often with a terminal master's degree. The professionals are often licensed by states as counselors, social workers, marriage and family therapists, and in a few states as psychologists.

Given the change in health care that emphasizes less expensive forms of treatment as preferred, it is not surprising that many state agencies and health organizations frequently employ master's training professionals to provide counseling and psychotherapy services. This change, combined with the large number of doctoral clinical psychologists graduating each year, has led to a concern with the current supply and future demand for clinical psychologists (cf., Pion, 1991; Robiner, 1991a, 1991b; VandenBos, DeLeon, & Belar, 1991). While it is not clear how "many" psychologists are needed, it is clear that psychology must identify those aspects of training that make psychologists unique from master's level practitioners. Rigorous training in science that includes rule-governed and contingency-based training may prove to be the critical difference.

Clinical scientists will be expected to show leadership in research on effective clinical assessment and intervention approaches. Clinical practitioners must incorporate the knowledge base of clinical psychology in their practice and identify strengths and weaknesses of proposed efficacious treatments. This dialog between clinical scientists and clinical practitioners is critical for the continued development of science in clinical psychology.

### 1.02.7 CONCLUSIONS

The hallmark of clinical psychology is the emphasis on science. The scientific status of clinical psychology is strong. Models of science

are consistent with the primary training model of clinical psychology (the scientist-practitioner model) which is flexible enough to allow clinical psychologists to focus on the science (clinical scientists) and practice (clinical practitioners) of clinical psychology.

Clinical assessment has a strong didactic knowledge base. While clinical psychologists are not using clinical assessment strategies as frequently as in the past, changes in the way psychological assessment is conceptualized and used appear promising. However, it is likely that clinical assessment and clinical intervention will become more integrated and the need to differentiate between assessment and intervention may no longer be critical.

Much is known about the most effective treatments for some psychological problems. While the development of effective treatments is contested by some, the proliferation of treatment manuals and potential changes in public policy should serve to strengthen the science base of clinical intervention.

In summary, clinical psychology retains a strong commitment to science and science models predominate all aspects of clinical psychology. There is room for growth and new developments in the field; however, the current status of clinical psychology will serve as a strong foundation.

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# 1.03

## Informational Sources in Clinical Psychology

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### 1.03.1 INTRODUCTION

Knowledge is of two kinds. We know a subject ourselves, or we know where we can find information upon it. (Boswell, *Life of Samuel Johnson*, 1775)

A new client presents to your clinical practice with pica. How do you determine the best course of treatment? A colleague asks you to lecture to the family therapy class on innovations in the treatment of marital discord. Where do you find a comprehensive review of the recent literature? You are interested in learning about the new research on post-traumatic stress disorder. Is the library your only source of information?

The field of clinical psychology is diverse and informational material continually becomes available in a multitude of places. Because the half-life of psychological knowledge is brief, the need for updating and accessing resources is nearly constant for both researchers and practitioners alike. The purpose of this chapter is to indicate where sources of information and the vast array of resources are available. Understanding the variety of reference tools that are useful and how to employ them effectively is critical to "exploit the richness of the materials contained within them" (Mendelsohn, 1987, p. 1). In this first section, we outline where to find psychologically relevant material and some of the "nuts and bolts" of accessing the available information. In the second section, we highlight the types of journals and books that may be most useful to the clinical psychologist. Third, we address the various systems and indexes that summarize the psychological literature. Finally, we hope to bring the reader "on-line" with information regarding the use of the Internet for the novice web surfer. It is our hope that this chapter will serve as a road map to the wide world of information available in clinical psychology.

Although it has been speculated that these days are numbered, the search for informational resources in clinical psychology still begins at the library. Those fortunate to have access to a comprehensive, research university library, with all the advantages of interlibrary loan and on-line search capabilities, will be in the best position to retrieve the psychological literature. As would be expected, the reference section of

the library is the researcher's first stop. In this area, the holdings of the library are cataloged and access to the bibliographic tools is usually available. Depending on the size and fiscal solvency of the library, the tools may include card catalogs, computer terminals connected to library-specific holdings catalogs, CD-ROM readers for systems such as PsycLIT and Educational Resources Information Center (ERIC), and a variety of journal abstracts and references. Many of the materials described in the third section of this chapter will be located in this section where they are available to users.

Regardless of how this information is obtained, call letters and numbers will be used to find the material in the library. The two systems of classification, the Library of Congress and the Dewey Decimal System, are most frequently used in libraries in the United States and psychological materials are cataloged under a variety of call letters and numbers. The search for books is relatively straightforward but may allow for some aerobic exercise benefits, depending upon the layout of the library. Journals, in contrast, are often more difficult to locate. Most libraries separate, often by several floors, the current periodicals from the bound, archived volumes. Although this would appear to present a minor inconvenience, the consistency with which this procedure is employed is often variable. Thus, a recent edition of *Journal of Consulting and Clinical Psychology* may be unbound and on the shelf with other current issues; bound and in the stacks; unbound in the stacks with some or all of the other issues in that volume; or finally, off to the bindery.

In light of the declining financial resources of university libraries and the often substantial increases in the cost for books and periodical subscriptions, libraries are becoming more selective in their holdings. In the future, the easy and immediate access that many have grown accustomed to may become atypical. Fortunately, other resources are becoming increasingly available to aid the clinical psychologist.

### 1.03.2 JOURNALS AND BOOKS

#### 1.03.2.1 Journals

The scientific-practitioner field of clinical psychology has relied on scholarly journals as

the prime source of professional information. The foundations of today's general and specialty clinical psychology journals may be found in the *American Journal of Psychology* (founded by G. Stanley Hall in 1887), the *Journal of Abnormal Psychology* (founded by Morton Prince in 1906), the *Psychological Clinic* (founded by Lightner Witmer in 1912), the *Journal of Consulting Psychology* (founded by Johnnie Symonds for the American Association of Applied Psychologists; later to become the *Journal of Consulting and Clinical Psychology*), and the *Journal of Clinical Psychology* (founded by Frederick C. Thorne in 1945) (Routh, 1996). Clinical psychology and related applied psychology specialties can now boast literally hundreds of serial publications. Similarly, there has been a significant rise over the years in the number of books serving the field. In this section, we will describe the range of journals and books with a view to the distinctive contributions each can make. The PsycINFO database (of the American Psychological Association [APA]) abstracts around 1500 journals and publications each year (a list of these is available on the APA homepage). Of course, not all of these publications contain information applicable or useful to the clinical psychologist, but many do. Consequently, sorting through the information sources remains a challenging task.

Many professional and scholarly organizations sponsor journals. For example, in addition to the major organizations of APA and American Psychological Society (APS), other groups supporting journals include the American Orthopsychiatric Association, the Society for Research in Child Development, the American Association for Applied and Preventive Psychology, the American Association on Mental Deficiency, the Association for Advancement of Behavior Therapy, and the Association for Humanistic Psychology. Many of these contract with commercial publishers or self-publish their journals.

In addition, numerous commercial publishers have also developed journal publications through contracts with scholarly editors. Some of the major publishers of privately developed journals include Plenum Publishing Corporation, Clinical Psychology Publishing Company, Lawrence Erlbaum Associates, Sage Publications, Cambridge University Press, Human Sciences Press, Elsevier Science/Pergamon Press, and Guilford Publications. These publishers also produce journals sponsored by organizations; many are noted later. All of these journals are abstracted or indexed by the major services as will be described in Section 1.03.3.

Although clinical psychology as an area of practice and research can be divided and subdivided into varieties of categories, in the following sections, we will illustrate the topical coverage of these archival and serial publications through the following subsections: psychopathology and abnormal psychology (Section 1.03.2.1.1), general clinical psychology (Section 1.03.2.1.2), clinical psychology specialties and topics (Section 1.03.2.1.3), therapeutic techniques (Section 1.03.2.1.4), and professional issues (Section 1.03.2.1.5).

#### **1.03.2.1.1 Psychopathology and abnormal psychology**

Much of the basic research forming the foundation of clinical psychology is conducted under the rubric of psychopathology or abnormal psychology. The oldest journal in this area, the *Journal of Abnormal Psychology*, published by APA, generally covers articles on the etiology of psychopathology and abnormal behavior. Similarly, the *Journal of Abnormal Child Psychology* publishes studies of behavioral pathology in children and adolescents, but also includes etiology, assessment, and some treatment aspects. *Psychological Assessment* branched from the *Journal of Consulting and Clinical Psychology* to focus on assessment theory and methodology employed in clinical psychology research and practice. The *Journal of Psychoeducational Assessment* and the *Journal of Psychopathology and Behavioral Assessment* also attend to the theoretical and methodological aspects of psychological assessment.

Other journals are published to provide scholarly and clinical coverage for a particular diagnostic category or psychological disorder. For example, *Mental Retardation*, *American Journal of Mental Deficiency*, *Journal of Autism and Developmental Disorders*, and *Research in Developmental Disabilities* provide an arena for research in mental retardation and developmental disabilities. The *Journal of Affective Disorders*, *Journal of Anxiety Disorders*, and *Anxiety, Stress, and Coping* focus on internalizing disorders, while *The Schizophrenia Bulletin* and *Schizophrenia Research* publish research dealing with the etiology, diagnosis, and treatment of serious emotional disorders. The *Journal of Substance Abuse* and *Journal of Drug and Alcohol Abuse* disperses scholarly literature on addictive behaviors. Many other journals publish applied and basic research related to specific psychological problems (e.g., *Journal of Traumatic Stress*, *Child Abuse and Neglect*, *Journal of Emotional and Behavioral Disorders*, *Journal of Family Violence*).

### 1.03.2.1.2 General clinical psychology

The field of clinical psychology is diverse in the range of problems and interventions that are within its domain. This diversity may not have been a major problem in the early years of development; many clinical psychologists considered themselves generalists with the ability to research and intervene on a wide range of problems and psychotherapeutic techniques. Increasingly, however, specialties have emerged within clinical psychology. Nonetheless, some journals remain diverse in their topical catchment and do not restrict their view on the types of research or professional articles they publish. For example, two empirical journals, *Journal of Consulting and Clinical Psychology* and *Journal of Clinical Psychology*, place no restrictions on subject population, theoretical, or methodological approaches. Similarly, two review and conceptual journals, *Clinical Psychology: Science and Practice* and *Clinical Psychology Review*, are open to any relevant content. Within their pages, these journals may intermingle articles reflecting different ages or populations, the types of problems studied, methodological approaches, and theoretical conceptualizations, although some special issues might focus on a specific topic.

### 1.03.2.1.3 Clinical psychology subspecialties and topics

Increasingly, the field has become an umbrella for research and applications targeted to special populations and different theoretical or methodological approaches. Thus, different ages or psychopathological conditions are some of the divisions used to categorize publications.

Clinical child and pediatric psychology are perhaps the oldest subspecialties in clinical psychology. Clinical psychologists have conducted research and made therapeutic applications for children and adolescents dating back to the inception of the field. Two child-oriented sections within the APA Division of Clinical Psychology (Section on Clinical Child Psychology, Society of Pediatric Psychology) sponsor scholarly journals. The *Journal of Clinical Child Psychology* publishes articles on child and adolescent psychopathology research and interventions, services and programs for children and families, and professional issues, such as training and ethics. The *Journal of Pediatric Psychology* publishes empirical, review, and case study articles related to applications of psychology to pediatric problems and issues. Another APA Division, Child, Youth, and Family Services, recently decided to sponsor a journal, *Children's Services: Social Policy,*

*Research, and Practice*, to begin publication in 1998.

Non-APA journals in this area include *Children's Health Care*, an interdisciplinary journal published by the Association for the Care of Children's Health, with editorial and article contributions by pediatric and clinical child psychologists as well as nurses, child life specialists, and pediatricians. Another interdisciplinary journal, *American Journal of Orthopsychiatry*, is published by the American Orthopsychiatric Association with contributions by psychologists, psychiatrists, and social workers. An international, interdisciplinary journal, *Clinical Child Psychology and Psychiatry*, began publication in 1996. Finally, sometimes the child and family focus gets paired with a particular theoretical approach, as in *Child and Family Behavior Therapy* and *Psychoanalysis of the Child*. Both of these journals publish articles reflecting a specific orientation and target population.

Geropsychology, at the other end of the developmental continuum, focuses attention on adult development and aging, including the development of psychological problems. As a primary resource, APA publishes the journal, *Psychology and Aging*. Other psychology and interdisciplinary publications that cover this age group for clinical psychology interests include *The Gerontologist*, *Clinical Gerontologist*, *Journal of Geropsychology*, *Journal of Aging and Health*, and *Journal of Applied Gerontology*.

Health psychology/behavioral medicine has been a fast growing field allied with clinical psychology, although it is not always conceptualized as a clinical subspecialty (Bernard & Krupat, 1994). Journals that publish articles at the interface of behavioral sciences, health, and illness include the *Journal of Pediatric Psychology*, *Health Psychology* (sponsored by the APA Division of Health Psychology), *Psychology and Health*, *Journal of Health Psychology*, and *Journal of Behavioral Medicine*.

Community psychology is often thought of as distinct from clinical psychology, however, some aspects of its publications bear on clinical psychology interests. For example, the *American Journal of Community Psychology* (sponsored by APA Division 27) and the *Journal of Community Psychology* report applied and basic research as well as evaluations of intervention programs employing clinically important techniques.

Neuropsychology and rehabilitation psychology have several publications of interest and utility to clinical psychologists in different employment settings. These range from basic research on brain and nervous system functioning, assessment and diagnosis, to treatment, rehabilitation, and prevention. Journals

in this and related fields include: *International Journal of Clinical Neuropsychology*, *Psychosocial Rehabilitation Journal*, *Rehabilitation Psychology*, and *Journal of Applied Rehabilitation Counseling*.

#### 1.03.2.1.4 Therapeutic techniques

In practice, clinical psychologists use a broad range of psychological/behavioral intervention techniques to improve the functioning of those presenting with problems. Research and practice publications in this area may describe clinical analogues, controlled experiments, and field evaluations. Some journals take a broad view of psychotherapeutic techniques appropriate for publication; others take a more focused view. *Psychotherapy: Theory, Research, and Practice* (sponsored by the APA Division of Psychotherapy) and the *American Journal of Psychotherapy* are among the more generally oriented journals.

In contrast, there are many periodicals that reflect a specific theoretical orientation. Journals that evidence psychoanalytic or psychodynamic viewpoints include *Psychoanalytic Psychology* (sponsored by the APA Division of Psychoanalysis), *Psychoanalytic Review* (sponsored by the National Psychological Association for Psychoanalysis), and *Contemporary Psychoanalysis*.

An orientation to family-based interventions and family and marital therapy is provided through publications such as *Journal of Family Psychology* (sponsored by the APA Division of Family Psychology), *Family Therapy*, *Family Process*, and *The Family Therapy Networker*.

Cognitive-behavioral interventions, as an amalgamation of cognitive therapy approaches and behaviorally-oriented treatments, are represented by such journals as *Cognitive and Behavioral Practice*, *Cognitive Therapy and Research*, and *Journal of Rational-Emotive and Cognitive Behavior Therapy*. Behavioral therapy and behavior analysis publications on clinical interventions derived from learning theory are found in a number of scholarly journals including *Journal of Applied Behavior Analysis*, *Behavior Therapy*, *Behavior Modification*, *Journal of Behavior Therapy and Experimental Psychiatry*, *Behavior Research and Therapy*, and the new, on-line journal, *Journal of Behavior Analysis and Therapy*.

Group psychotherapy has been served by several interdisciplinary publications, including the *International Journal of Group Psychotherapy*, *Small Group Behavior*, and *Journal of Child and Adolescent Group Therapy*.

Prevention of mental and physical health problems is a frequently mentioned goal of

clinical psychology interventions. Although several journals in the field publish articles on prevention activities, a primary focus on this topic is provided by such journals as *Applied and Preventive Psychology*, *Journal of Primary Prevention*, and *Journal of Prevention and Intervention in the Community*.

Finally, psychopharmacological treatments represent a substantial and growing specialty interest within clinical psychology. Although some treatment and/or psychopathology oriented journals provide coverage of these issues, the *Journal of Psychopharmacology*, *Research Communications in Psychology, Psychiatry, and Behavior*, *Journal of Child and Adolescent Psychopharmacology*, and *Experimental and Clinical Psychopharmacology* focus exclusively on medication and medication-related issues.

#### 1.03.2.1.5 Professional issues

Within the profession of clinical psychology, issues such as (i) ethics for clinicians, researchers, and educators, (ii) training for professional competence, and (iii) professional practice and functioning (including employment and reimbursement considerations) are important topics. Such professional issues are regularly covered by *Professional Psychology: Research and Practice*, *Ethics and Behavior* and *Psychology, Public Policy, and Law*. Practitioner oriented publications have provided clinicians with information on changes in public sector and private practice concerns, insurance reimbursement and managed care issues, such as in *Journal of Mental Health Administration*, *The National Psychologist*, *Professional Practice of Psychology*, *Journal of Clinical Psychology in Medical Settings*, *Psychotherapy in Private Practice*, and *Behavioral Healthcare Tomorrow*.

The range of journals for the field of clinical psychology is extensive; this sampling cannot do justice to the wealth of serial publications archivable for the clinician and researcher. Additional sources can be ascertained through the APA publication, *Journals in Psychology: A Resource Listing for Authors* (APA, 1993) and the list of journals abstracted by PsycINFO (see APA homepage on the World Wide Web).

#### 1.03.2.1.6 On-line journals

In addition to the familiar print form, some journals have also been added to the Internet. The quality of these publications varies, although they provide an additional source of information for the clinical psychologist. Table 1 lists some of the publications currently available on-line.

**Table 1** On-line journals for clinical psychology.

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<i>Homepage Association</i>
<i>URL address</i>

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<i>American Journal of Psychotherapy</i>	<a href="http://www.apj.com/">http://www.apj.com/</a>
<i>Canadian Journal of Behavioural Science</i>	<a href="http://www.cycor.ca/Psych/ac-main.html">http://www.cycor.ca/Psych/ac-main.html</a>
<i>Gestalt!</i>	<a href="http://rdz.stjohns.edu/gestalt/">http://rdz.stjohns.edu/gestalt/</a>
<i>International Journal of Psychopathology, Psychopharmacology, and Psychotherapy</i>	<a href="http://www.psycom.net/ijpppp.html">http://www.psycom.net/ijpppp.html</a>
<i>Journal of Behavior Analysis and Therapy (jBAT)</i>	<a href="http://sage.und.nodak.edu/org/jBAT/jbatinfo.html">http://sage.und.nodak.edu/org/jBAT/jbatinfo.html</a>
<i>PsychNews International</i>	<a href="http://www.cmhc.com/pni/">http://www.cmhc.com/pni/</a> or <a href="http://www.mhnet.org/pni/">http://www.mhnet.org/pni/</a>
<i>Psychology</i>	<a href="http://cogsci.ecs.soton.ac.uk/harnad/psyc.html">http://cogsci.ecs.soton.ac.uk/harnad/psyc.html</a>

### 1.03.2.2 Books

In addition to journal publications, published volumes have been the *sine qua non* for the science and clinical practice of psychology. Authored books and edited books with individually authored chapters have summarized the knowledge and progress of this field since G. Stanley Hall.

Currently, a vast number of books, from a variety of publishing companies too many to list here, serve the field. Many book series act as collections of various titles in clinical psychology, such as *Advances in Pediatric Psychology* (Guilford), *Sage Human Services Guides* (Sage), *Wiley Series in Clinical Psychology* (Wiley), *Wiley Series in Personality Processes* (Wiley), *Developmental Clinical Psychology and Psychiatry* (Sage), *Applied Clinical Psychology* (Plenum), *Clinical Child Psychology Library* (Plenum), *Issues in Clinical Child Psychology* (Plenum), and *Stress and Coping* (Plenum).

As with other forms of information, the prodigious number of published volumes makes it difficult to determine their utility. Decisions about quality are often made by considering the source, the qualifications and reputation of the author/book editors, series editor(s), and the publishing company. Fortunately, there is some additional guidance from *Contemporary Psychology*. This journal is devoted to publishing scholarly and critical reviews of new book publications. Other journals also publish a few book reviews (e.g., *Journal of Pediatric Psychology*, *Clinical Psychology Review*). Unfortunately, the time lag for the reviews is often too long to be helpful to the reader. Unlike most journals that employ peer review, books frequently do not undergo adequate scrutiny before publication. This deficit is particularly

unfortunate as descriptions of treatments of unproven quality may be widely disseminated with potential adverse impact on clients.

### 1.03.2.3 Conclusions

The nature of journals and books is changing with the rise of computer and Internet capabilities to transmit information. Although many predict the ultimate demise of the printed paper format for journals and books, others have not been so quick to accept this prediction. Given the seemingly chaotic nature of the Internet development, the need to sort through and apply some standards of accuracy and utility will remain. Thus, it is likely that the fundamental and time-tested aspects of publishing will hold up, that is, the tradition of the editor as gatekeeper, and the peer review process.

### 1.03.3 ABSTRACTING SERVICES

As was suggested in Section 1.03.2.3, the sheer number of individual references for any given topic may often be overwhelming. Fortunately, there exist several good sources of abstracts, indexes, and other retrieval mechanisms to facilitate the identification of relevant literature. Information systems such as PsycINFO (from the APA) attempt to provide the resources to access the world's literature in psychology and related disciplines. Journals, technical reports from university and government sources, dissertation abstracts, monographic series, and similar materials are the fodder for these systems. The systems do, however, vary considerably. Some abstracting products provide nonevaluative reports of any



reference while others abstract the material presented within a selected set of journals. Further, some indexes provide summaries of the literature, while others offer only bibliographic information. In this section we will describe the major systems and ways to access the information they contain. Although we have provided the most current (as of the late 1990s) information regarding abstracting sources in psychology, because of the rapidly evolving technology, databases, particularly CD-ROM systems, change frequently (Reed & Baxter, 1991). Thus, in order to minimize frustration and maximize successful searches, the reader is advised to monitor new developments in the field.

### 1.03.3.1 PsycINFO

The major source of information regarding the psychological literature is PsycINFO. In addition to maintaining the comprehensive PsycINFO database, this department of the APA produces several other resources from their database such as *Psychological Abstracts*, PsycLIT, and PsycSCAN. The PsycINFO electronic database includes all the bibliographic references in psychology since 1967, a collection of more than one million citations of psychologically relevant material including journal articles, book chapters and books, dissertations, as well as technical and governmental reports (APA, 1996). References prior to 1967 have been digitized and indexed, and will be available as an option to institutions that lease the database (Hayward, 1996). PsycINFO is updated monthly and all references are presented in English, although not all of the literature abstracted is from English-language publications.

PsycINFO is an abstracting system for psychology and related disciplines that is not bound by geographic or linguistic constraints, but rather evaluates the entire body of relevant psychological information. The database includes research reports and other scholarly works, literature reviews, surveys, case studies, theoretical papers, dissertations, books, chapters, conference and case law reports, and, if the original work was included in the database, comments and replies. Excluded are editorials, letters to the editors, news items, interviews, and poems. Unlike some other abstracting services, PsycINFO does not maintain a core list of journals but rather evaluates the psychological relevance of any individual entrant regardless of the source (e.g., journal, book chapter, government monograph, etc.). By providing a compendium of the psychological literature,

PsycINFO permits scholars to “stay current” in their field without surveying all the primary sources.

The PsycINFO database, and the other PsycINFO reference systems, supply three fundamental types of information: bibliographic information, an abstract (or summary), and standardized keywords. The database, however, may only be accessed directly through institutions that have an annual lease for its use and through commercial on-line services, such as Knight-Ridder (formerly DIALOG) and CompuServe.

Before using any PsycINFO products, an understanding of the language and logic of the search systems is necessary. As is the case with most searchable databases, Boolean logic is used. Boolean modifiers such as “AND,” “OR,” and “NOT” may be used to alter the results of a search. By using these modifiers, a large search can be narrowed to a more relevant topic, while restricted topics can be expanded. For example, a search using anxiety “AND” adolescents will generate fewer matches than anxiety “OR” adolescents. If no modifier is specified, an “OR” is assumed, which may result in fewer relevant references.

Familiarity with the *Thesaurus of Psychological Index Terms* (APA, 1997), the key to the “controlled vocabulary” used to index entries in the PsycINFO database, is essential. Because a variety of words exist to describe psychological concepts, the *Thesaurus* provides a standardized wording, thereby decreasing the frustration that may result when confronted with the idiosyncrasies of the language. By using the *Thesaurus* one is redirected from unused words to the keywords used by PsycINFO (and the corresponding five digit subject codes). For example, searching “birth parents,” an unused word, retrieved only 23 records, but by substituting the keyword recommended in the *Thesaurus*, “biological family,” 125 records were obtained.

In addition, this reference provides related terms, both broader and narrower, that may be used to further define a literature search. In general, use of the narrower descriptor is preferred because PsycINFO records are indexed with the most specific descriptors possible (APA, 1996). Now in its eighth edition, the *Thesaurus* is updated every three years to reflect changes in the vocabulary of psychology and related disciplines.

Although the *Thesaurus* strives for completeness, there may be times when searching the “free-text” fields may be more fruitful. The free-text fields are entries such as the title (TI), abstract (AB), key phrase (KP), and author (AU) that may be searched for a word or phrase. A complete list of the possible “free-text” search

fields is available in the *PsycINFO User Manual* (Walker, 1992) and through on-line "help." Searching the free-text fields may be necessary when a concept does not have a corresponding descriptor. When this occurs, searching the title, the key phrase and abstract of journal articles, may result in a successful "match." For example, if a psychologist was interested in finding articles that address the topic "corporate culture" at the FIND prompt, one would enter "corporate culture in TI (title) or corporate culture in KP (key phrase)." For books and chapters, the table of contents may also be searched.

A free-text field search would be appropriate when references with a specific word or phrase are of interest to the researcher. For example, if interested in studies evaluating the effectiveness of the antidepressant "Zoloft," a search for that specific term in the free-text fields would be made. Likewise, a hunt for a specific person or assessment device may be conducted by this method.

Finally, a free-text search may be needed when searching for studies prior to the development of a new descriptor. For example, to retrieve articles on "AIDS prevention" prior to 1994 (the year it was entered into the database as a descriptor), a free-text search would be necessary. The *Thesaurus* indicates when a particular descriptor was added and in some cases provides alternative descriptors to use when searching the older literature. For example, it suggests that to access the research on post-traumatic stress disorder published between 1973 and 1984, one may use "stress reactions" or "traumatic neurosis" as the index words.

Two other "tricks" that may be used to maximize the results of a "free-text" search are the use of the truncation symbol (\*) and the wildcard symbol (?). The truncation symbol may be used to retrieve variants of the specified word or phrase. For example, entering "sex\* in TI" retrieved 7636 records from the journal database (January 1990 to June 1996) with a broad range of related words in the titles such as sexualized, sexual harassment, sex offenders, sexually abused, and sexual orientation. Consequently, to obtain the most relevant information, it is important to restrict use of the truncation symbol by including as many of the letters in a word as possible. In the above example, "had sexu\*" been used, then fewer records would have been produced.

Use of the wildcard symbol (?) in a word will permit a letter (or letters) to replace it. If the "?" is used at the end of a word, it may be used to represent an "s" for plural forms of the word. Another use of the wildcard symbol is to obtain references with alternate spellings of a word.

Thus, to retrieve records with both American and British spellings, "color" and "colour," in the title, "colo?r in TI" must be entered.

### 1.03.3.1.1 Psychological Abstracts

The "work horse" of the PsycINFO stable is *Psychological Abstracts*, the well-used reference for nearly 70 years. This reference, published monthly, is available only in printed form and provides abstracts of journal articles, technical reports, book chapters, and books. Each volume contains approximately 47 000 records. By including coverage from 1927 to the present, scholars can learn of advances in the field as well as the historical contributions to clinical psychology. Because of the volume of psychological literature worldwide and the cost of producing a printed publication, the inclusion of foreign language entries in *Psychological Abstracts* was discontinued in 1988, although the PsycINFO database still retains these references (Reed & Baxter, 1992).

For many years, *Psychological Abstracts* was the only system available to researchers and clinicians who wished to find literature on any given topic in psychology. However, with the development of PsycINFO and related resources, gone are the days of thumbing through the cumbersome subject references to find the page numbers of corresponding abstracts. PsycLIT replaces, for many, the printed abstracts and allows the user to access the same material from a CD-ROM database. Nevertheless, *Psychological Abstracts*, available only through subscription from the APA, remains a staple reference in most libraries. Each issue features subject, book title, and author indexes, and at the end of the year cumulative indexes of authors and subjects are published in a separate volume.

### 1.03.3.1.2 PsycLIT

PsycLIT is the database with which most individuals are familiar. Although PsycLIT and PsycINFO are frequently used interchangeably, there are significant differences between the two. As previously described, PsycINFO is an on-line database; in contrast, PsycLIT is a CD-ROM database derived from the PsycINFO database, that contains references for journals, book chapters, and books only. Contained within two compact discs, this reference, updated quarterly, comprises three separate databases: (i) journal references from 1974 through 1989, (ii) journal references from 1990 to the present, and (iii) books and book chapters from 1987 to the present. Each entrant includes a bibliographic citation, an abstract,

and, for journal articles, index words. The indexing is included to allow similar articles to be selected from the database. Book and chapter records include comparable information, with the addition of the table of contents for books.

To find references on this CD-ROM version of the *Psychological Abstracts*, keywords are entered. As previously discussed, the use of Boolean modifiers is necessary. There are several support systems that make PsycLIT very user-friendly including an "on-disc" Thesaurus, a tutorial, a videotape entitled "How to Search PsycLIT on CD-ROM," several user's manuals from PsycINFO, and a toll free number to the PsycINFO Actionline ([800] 374-2722) (APA, 1996).

Similarly, the ClinPSYC CD-ROM database offers a reduced set of abstracts specifically chosen for their relevance to practitioners and interested others in clinical psychology, behavioral medicine, and related fields. Because of the reduced volume of entries, the ClinPSYC database is much less expensive and may be useful for smaller institutions and organizations.

### 1.03.3.1.3 PsycSCANs

Finally, PsycINFO publishes field-specific bibliographic and abstract journals including *Psychoanalytic Abstracts* and PsycSCANs. The increasing specificity of these publications allows individuals to keep up to date with current literature in a circumscribed area of interest. The PsycINFO database of over 1300 journals in psychology and related disciplines is searched to provide the citations and abstracts for these journals. For example, *PsycSCAN: Behavior Analysis and Therapy* provides concise summaries of journal articles on basic research and applied topics accompanied by complete citation to assist readers in locating the full-text article. PsycSCANs, published quarterly, are currently available for the following areas of interest: applied psychology, behavior analysis and therapy, clinical psychology, developmental psychology, and learning disabilities/mental retardation.

*Psychoanalytic Abstracts* and *Neuropsychology Abstracts* are two similar PsycINFO products that encompass a specific area of psychology. The quarterly *Psychoanalytic Abstracts* replaced the *PsycSCAN: Psychoanalysis* in 1993 and features abstracts of journal articles, as well as summaries of book chapters and books relevant to the specialty area of psychoanalysis. A core set of approximately 40 psychoanalytic journals, in addition to the 1300 general psychology journals, are abstracted. *Neuropsychology Abstracts* fulfills a similar function for the field of neuropsychology.

Both the Abstracts and the PsycSCANs can be very useful in that they provide a manageable number of references to peruse. Nonetheless, scholars interested in a wider scope of information may be frustrated by the circumscribed range covered by these journals. For example, clinical psychologists interested in pediatric phenomena and applications would not have access to all relevant research in this area by using the *PsycSCAN: Clinical Psychology* exclusively.

In summary, the PsycINFO products are excellent resources and allow the scholar to access a wide variety of materials through several user-friendly mechanisms. By developing several different kinds of products, PsycINFO has provided multiple levels of access to their database that vary in terms of scope and cost. Thus, while PsycLIT may be too expensive for a small group practice, the lower cost of the PsycSCANs may allow clinical psychologists entry to the research literature.

### 1.03.3.2 Educational Resources Information Center

In addition to PsycINFO, another large database that produces a variety of products that may be relevant to clinical psychologists is the ERIC. This national information system, established in 1966, provides access to the large body of education-related resources. ERIC is supported and operated by the National Institute of Education (Gay, 1992).

As with PsycINFO, ERIC offers a range of services, although a primary function is the maintenance of a national bibliographic database of over 775 periodicals in education and related fields. This database is compiled from 16 subject-specific "clearinghouses" that abstract and index the education literature. The majority of these clearinghouses, however, will not be of interest to clinical psychologists as they primarily provide teaching-related materials such as curricula and classroom resources. While there is considerable overlap with the material covered by PsycINFO, ERIC does include references for unpublished research that is not usually available from other sources, such as papers presented at professional conferences and studies conducted by school districts. The ERIC database can be accessed through the Internet (URL: <http://ericir.syr.edu>) and by CD-ROMs purchased from vendors such as Knight-Ridder, Oryx Press, and SilverPlatter Information, Inc.

In addition to the database, two other products available in both microfiche and printed versions are offered by ERIC: *Resources*

in *Education* and *Current Index to Journals in Education*. As with the *Thesaurus of Psychological Index Terms* produced by PsycINFO, ERIC has a companion guide, *Thesaurus of ERIC Descriptors* (Houston, 1990) that should be consulted before beginning a search. Although indexed similarly, the monthly *Resources in Education* includes educational research, conference proceedings, position papers, curriculum guides, books, and dissertations, while the *Current Index to Journals in Education* covers only periodicals.

Clinical psychologists interested in references for child development, education, testing and evaluation research will find references within the ERIC database. Notwithstanding, for many other clinical topics, such as psychological disorders and treatment, PsycINFO products would be a preferable resource. For example, while the search with the keyword "pica" resulted in 50 "hits" from the PsycLIT CD-ROM database, the same search performed with the ERIC CD-ROM generated no relevant references.

### 1.03.3.3 *Index Medicus*

*Index Medicus*, published by the National Institutes of Health (NIH), is an abstracting reference for all medical journals, including those areas of psychology related to the health sciences. Approximately 62 psychology and 80+ psychiatry journals are indexed, although many of these are "selectively" searched, and not all their references are included (NIH, 1996). Unlike PsycINFO or ERIC, this reference does not provide abstracts or summaries of the material, only bibliographic citations organized by subject and author.

On-line access to *Index Medicus* is available from MEDLINE, which also includes the *Index to Dental Literature* and *International Nursing Index*. MEDLINE may be accessed through the World Wide Web (<http://www.healthgate.com/> or <http://www.medscape.com/>), by institutional lease, and on CD-ROM, through vendors such as Ovid and Knight-Ridder. The *Medical Subject Headings*, the thesaurus for use with MEDLINE and *Index Medicus*, is updated and published annually.

Many topics of interest to psychologists, such as conduct disorders, "bystander intervention," and family therapy, are found on MEDLINE. Most MEDLINE searches, however, will necessitate some culling to obtain the most useful subset of references as both psychosocially oriented articles and physical medicine articles are retrieved. For psychologists interested in health and pediatric psychology, *Index Medicus* and MEDLINE are invaluable references,

providing coverage of medical journals not included in PsycINFO. For example, a search for "pica" resulted in the retrieval of 710 references, in contrast to the 50 references obtained through the PsycLIT search.

One of the major limitations of *Index Medicus*, however, is the limited information provided. Because only the title is provided in the index, there is no indication as to the content of the article, making it difficult to identify critical references from this resource. In contrast, MEDLINE searches may include abstracts when they are available, thus overcoming this deficiency.

### 1.03.3.4 Focused Abstracting Services

In addition to the three major resources previously described, PsycINFO, ERIC, and *Index Medicus*, a number of other resources provide reference sources and informational services relevant to clinical psychology. Although most of the psychological literature will be accessible through PsycINFO and the other "powerhouses," these additional resources may be of interest to clinical psychologists. In this section we will describe these products, with an eye to their unique contributions.

#### 1.03.3.4.1 Dissertation Abstracts International

There are two parts of the *Dissertation Abstracts International (DAI)*, Part A: Humanities and social sciences, and Part B: Sciences and engineering. Within this resource, dissertations are listed under subject headings, with interdisciplinary dissertations listed under more than one category. The majority of psychology dissertations will be found in Part B, although Part A contains educational psychology, tests and measurements, and gerontology (Reed & Baxter, 1991). Published monthly by the University Microfilms International at the University of Michigan, the *DAI* is available on microfilm, CD-ROM, and in print. Because dissertations often represent the "cutting edge" of research, this reference may be very useful to individuals interested in new developments in the field. In addition, this reference may be one of the few ways to access this body of scholarly works since dissertations are no longer included in PsycLIT or *Psychological Abstracts*.

#### 1.03.3.4.2 Child Development Abstracts and Bibliography

This publication of the Society for Research in Child Development (SRCD) provides abstracts and bibliographies for books and journal articles related to the growth and

development of children. Unlike the PsycINFO products, *Child Development Abstracts and Bibliography* maintains a list of journals regularly searched and solicits contributions of abstracts from journals not included in this list (SRCD, 1996). This database reference is available in print and free through the Internet (<http://www.journals.uchicago.edu/CDAB/>).

#### 1.03.3.4.3 Clinician's Research Digest

Designed specifically for clinicians, the monthly *Clinician's Research Digest (CRD)* is published by the APA. This six-page newsletter covers the highlights gleaned from the review of over 50 clinically relevant journals. Each issue includes a summary of recent research and complete bibliographic information. The *CRD* claims to be more "user friendly" than similar publications (APA, 1996), although the restricted number of articles reported and the absence of PsycINFO descriptors and keywords would appear to limit its utility. Nonetheless, for individuals seeking a brief, informal review of clinical research, the *CRD* may be a very approachable resource.

Similar reference information for clinical psychologists may be found in print publications such as the *Behavioral Medicine Abstracts* (published by the Society for Behavioral Medicine), the *Human Resources Abstracts* (published by Sage), and the *Social Work Research and Abstracts* (produced by the National Association of Social Workers). These references are limited to a specific content area and are currently not available electronically. In addition, because of their cost and redundancy with information abstracted by the other larger abstract services, they may not be carried at many libraries.

In contrast, there are several on-line databases that provide abstracts for specific content areas that are only available electronically, with no corresponding print equivalents. Among these are *Child Abuse and Neglect* (produced by the National Center on Child Abuse and Neglect), *DRUGINFO* which provides information regarding the psychological aspects of drug and alcohol abuse (produced by Drug Information Services at the University of Minnesota), the *Mental Health Abstracts* (published by IFI/Plenum Data Company), and *REHABDATA* (from the National Rehabilitation Information Center).

The utility and availability of these abstract services varies considerably. The smaller resources reference a much more circumscribed range of journals and are therefore redundant with the larger systems. Conversely, for some psychologists, the reduced number of references

may be more manageable, and consequently, useful. With regard to availability, discipline-specific libraries, such as those found at large research universities, may carry the appropriate references. Nevertheless, because of financial exigencies, many institutions have discontinued their subscriptions to these abstract services.

#### 1.03.3.5 Indexing Systems

In contrast to abstracts, indexes provide only bibliographic information, yet they may be useful reference tools for some scholars. Given the emergence of the "computer age," many of these resources are available electronically as well as in print form.

##### 1.03.3.5.1 Social Science Citation Index

This quarterly publication from the H. W. Wilson Company provides only author and subject indexes from 1400 international English-language periodicals and selective coverage of 3300 additional journals. The coverage of the *Social Science Citation Index (SSCI)* is very comprehensive; journal articles, review articles, scholarly replies to the literature, interviews, book reviews, meeting notes, corrections, obituaries, and biographies are indexed (Garfield, 1995). In addition to a print edition, the index is also available on CD-ROM and Social SCISEARCH is the corresponding on-line database from the Institute for Scientific Information. While the majority of psychology journals are covered in the *SSCI*, there is some overlap with the companion publication, the *Science Citation Index (SCI)*. Both the *SSCI* and the *SCI* are very expensive and therefore may not be available in smaller libraries.

The *SSCI* allows the identification of research that has been cited by others. If an author's work has been cited by another it will be indexed here, allowing interested individuals to find more recent work based on older research. This information may be very useful for scholars seeking to understand the historical trends in research or the impact of a specific empirical finding. A distinct advantage of these indexes is the use of the Permuterm Subject Index. This system indexes every substantive word in an article's title, thus overcoming some of the obstacles of other indexing systems that require the use of specific descriptors.

##### 1.03.3.5.2 Current Contents/Social and Behavioral Sciences

This is one of the seven multidisciplinary editions available from the Current Contents database, a product of the Institute for Scientific

Information (ISI). This database is updated weekly and is available in both print and electronic forms. For the Social and Behavioral Sciences edition, 1421 journals from psychology, psychiatry, education, public health, social work, and education, as well as other social science areas, are indexed (ISI, 1996). All forms of the publication (e.g., print, disk, CD-ROM, on-line) provide the tables of contents from all the journals reviewed, along with author and publisher addresses. The provision of addresses is particularly handy for ordering reprints. In some formats of Current Contents, abstracts and keywords are also included. As with PsycLIT, on-line access to Current Contents/Social and Behavioral Sciences is available from commercial services such as Knight-Ridder and Ovid Technologies.

Although there is considerable overlap with the PsycINFO database, Current Contents does provide a unique reference tool. For scholars interested in a wide range of psychology-related materials, such as sociology, law, and social work, Current Contents is unparalleled in its scope. The wide range of coverage is not inexpensive, however; the price of a one-year individual subscription on disk being \$895 in 1997. Moreover, because it is updated weekly, continual review of the contents is necessary to keep up with the issues, a task some individuals may find difficult.

Another drawback to Current Contents is the division of the database into multiple issues (e.g., Social and Behavioral Sciences, Life Sciences, etc.). Thus, individuals may find that one edition does not include all the articles in their area of interest. Finally, the index terms included in the back of each issue are very broad (e.g., "personality," "child") and therefore provide only minimal guidance in searching for references on specific topics.

### **1.03.3.5.3 UnCover**

Libraries have long maintained interactive systems to share their resources (e.g., through interlibrary loan, faxes) and have developed computerized systems of informational retrieval. One particularly important and useful electronic development has been the establishment of electronic databases for access to scholarly publications. As a result of the widespread use of these information sources, library groups and private companies have established document notification and delivery services. The UnCover Company (a Knight-Ridder Information Company) is one such service (<http://www.carl.org/uncover/unchome.html>; telephone: 1-800-787-7979; e-mail: [uncover@carl.org](mailto:uncover@carl.org)). It provides Internet

access to almost 17 000 journals and over seven million articles. The literature available is not just in psychology, but from the totality of science, social science, and the humanities. Journals that are related to clinical psychology (as listed in this chapter) are included in the database.

There are several ways to access the UnCover database: at libraries with an institutional lease, by telnet ([database.carl.org](http://database.carl.org)), by modem ([303] 756-3600), and through the World Wide Web at the address provided. There is no cost to search the database and individuals may search by author's name, keyword, or journal title. Journal titles allow branching to tables of contents for issues published over a period of years. The results of a search are limited to bibliographic information. Once articles are identified in the search, UnCover can be requested to fax the article to a specified fax machine. The average service charge for fax delivery of the article, including the copyright release fee, is \$10.25.

An UnCover topic keyword search for the topic of "managed care" identified 2303 entries. By adding the phrase "mental health" to "managed care," the number of entries was reduced to 115 articles in a variety of journals. All entries seemed relevant, although it is not discernible how many potential entries were missed in the search. Examination of the entries will eliminate some from interest; others may be marked for fax delivery. An UnCover keyword search for "depression" found 7687 items and "depression in the elderly" netted 247 entries. Large numbers of entries are not sorted by the service. The user must scroll through the entries to find useful references.

In addition to the search capacities, another service of this electronic data bank is UnCover Complete which allows the ordering and faxing of any articles in journals not indexed in the data bank or from pre-1989 issues. Entry of the bibliographic information obtained from other sources (e.g., PsycLIT) or reference lists of other articles is required.

A particularly useful service available from the UnCover company is UnCover Reveal. UnCover Reveal allows a subscriber to select up to 50 journals from the database for an "awareness service" where the table of contents from the selected journals are sent to the subscriber's e-mail account as the journal issues are published. Once the desired articles are identified, subscribers may then order them to be sent to their facsimile machine within 24 hours. UnCover Reveal also permits the subscriber to identify 25 keyword and/or author names to be searched weekly as new articles are entered into the database and the results

delivered to his or her e-mail address. This service is particularly useful for individuals who may not have access, either personally or through their library, to all of the journals in their area of interest or for scholars who want the most current information as soon as it becomes available. A one-year subscription to this service is \$25.00.

In general, UnCover appears to be a potentially useful database service for individuals with limited library access. The ability to order articles on-line and receive them within a brief time period is very convenient, albeit costly. The charge for this service, however, may be equivalent to interlibrary loan fees, with a more expedient delivery time.

#### **1.03.4 ELECTRONIC MEDIA: THE INTERNET**

The rise of the electronic media in its multitude of forms has created a vast array of new resources for clinical psychologists, including both researchers and practitioners. At the same time, however, the rapid rise and the enormous wealth of resources have made sifting through and evaluating the utility of those resources more difficult. In this section, we will outline the types of resources available in the electronic media, some of the information sites and their contents, how to access that information, and how to evaluate their use. We want to emphasize that, perhaps different from other referential sources of information in clinical psychology heretofore, any list of materials available through the Internet is, upon compilation, almost immediately outdated. That is, informational homepages, professional and topical newsgroups, discussion networks, Internet-based journals, newsletters, publications, and other forums are being created daily (and others cease to exist). Most importantly, we emphasize that the mere availability of abundant information does not ensure its accuracy or utility for the clinical psychologist.

Before describing some useful Internet resources, we want to clarify and define some of the terms. Although it is often described as a single entity, the Internet is the network of networks, (Kehoe, 1995). Individuals who are connected to another computer in some way, either through telephone lines and a modem, or through direct wiring, are part of the Internet and join approximately 20 million other users in 50 countries on any given day. It is through this network of computers that electronic mail (e-mail), file transfer protocols (FTP), mailing lists (listservers), on-line discussions (bulletin boards and newsgroups), and the World Wide Web (WWW) are possible.

Access to the Internet, and the many services it supports, may be made through commercial companies (e.g., America Online, Compuserve, Prodigy, and many local network providers) as well as educational institutions and governmental agencies. Generally, the type of connection to the Internet does not affect the types of services available, but will impact the speed at which information may be accessed.

In the last few years, consumer-oriented, user-friendly, services and tools have developed to make it quite easy for the novice computer user (a "newby" in Internet lingo) to gain access to the multitude of informational sites (as well as entertainment and other diversions) on the Internet. The software for sending out electronic mail or searching the Internet for information via the larger commercial companies are fairly easy to use (although the financial charges for use and time can accumulate fairly rapidly). For those accessing the Internet through other means (e.g., regional or local networks, or .edu or .gov computer accounts), special e-mail software and/or use of a "web browser" may be provided. Web browsers are generally point-and-click tools for searching and accessing information; we have found the easiest ones to use are Netscape and MS Internet Explorer. These programs are free for academic and research users and provide a hypertext interface to the World Wide Web. Easily installed, they can be quickly activated for conducting searches and acquiring information. These browser programs generally provide a range of options for accessing information. We will describe some of the informational search options later in this section.

##### **1.03.4.1 Electronic Mail**

Although directly e-mailing other professionals is a useful way of requesting, obtaining, and exchanging information for the clinical psychologist, we will not deal with this medium in detail. Electronic mail addresses for individuals are now frequently included in membership directories for professional organizations such as the APA and the APS.

##### **1.03.4.2 Listservers**

Collections of e-mail addresses of those people who have actively subscribed to a central computer list are called listservers (or listservs). These groups are typically centered around a common interest or activity such as members of the Division of Clinical Psychology, or researchers and practitioners with interests in traumatic stress. Members of the listserver may

post inquiries for information, posit a thought, initiate dialogues and discussions over professional issues, and provide information via e-mail to the central computer and all members of the group receive copies of the message in their e-mailboxes. Some listserver groups are moderated by a professional; others are freewheeling and supervised only by peer pressure.

As an example of how a listserver group might assist the clinical psychologist, an inquiry to a clinical psychology network from a practitioner asked for valid and reliable ways to assess a particular disorder suspected in a client. Replies from the group included several suggestions of how to handle the case, references to written material, and summaries or abstracts of articles on the topic. Often the replies illuminate and educate, although sometimes the discussion turns into an argument or meaningless chatter. (A word of warning here: There are those who enter a newsgroup apparently to stir up trouble deliberately by posting erroneous information or making statements calculated to provoke the other members of the newsgroup.) To enroll in a listserver usually requires only that one sends a "subscribe" message by e-mail, although a few others require checking against a master list or review of credentials. Some listservers of relevance to clinical psychology are listed in Table 2.

#### **1.03.4.3 Usenet Newsgroups**

Usenet newsgroups differ from e-mail list-servers in that messages or articles are not transferred directly to a user, but are posted in a central location accessible by individuals' computers through the Internet. Usenet newsgroups typically form around a topic or interest. Members of the newsgroup post messages on the "bulletin board" of the group, and other members may react by posting replies or new positions/statements to the bulletin board for all to see or send personal replies to those who have posted before. There is no Usenet organization or authority, although some newsgroups are "moderated" by an individual who reads and approves a message before posting it in the newsgroup board. Supportive contacts with others facing a particular problem are often facilitated in these groups.

The lengthy list of the thousands of Usenet newsgroups can be accessed through Netscape (<http://sunsite.unc.edu/usenet-i/hier-s/top.html>). Each newsgroup address is preceded by a category prefix (e.g., sci., soc., alt., etc.) that represents a broad topic area (e.g., science, social, alternative, etc.). Subsequent words serve to add specificity to the particular newsgroup.

For example, many of the groups interested in psychology begin with the "alt.psychology" followed by another, more specific term such as "personality." The DejaNews research service (<http://www.dejanews.com>) may be used to locate newsgroups addressing specific topics. Some examples of Usenet newsgroups that may be of interest to clinical psychologists include:

alt.psychology	(.adlerian, .jung, .help)
alt.support	(.attn-deficit, .depression, .schizophrenia)
alt.recover	(.compulsive eating, .sexual addiction, .aa)
sci.psychology	(.theory, .psychotherapy, .personality)
alt.sexual.abuse.recovery	
soc.support.loneliness	

#### **1.03.4.4 File Transfer Protocols**

Through file transfer protocols (FTP), professionals may obtain and exchange information in computer files from one system to another. A variety of information resources including journals, books, and other documents can be downloaded to the clinical psychologist's computer through this mechanism. Searches for information obtainable through FTP may be made through the "Gopher" system which searches for documents and retrieves them or a Wide-Area Information System (WAIS).

#### **1.03.4.5 World Wide Web**

The collective term for the global network of interlinked computers is the Web or World Wide Web (WWW). The vast array of information contained in all those computers is available for access through special, easy to use tools. The Web is both a "treasure trove" and a "chaos morass." Indeed, a new user accessing the WWW for the first time will be amazed and perplexed at the variety and range of offerings. After initial shock and fascination in "surfing" around the Net, the Internet user will approach the Web and Internet activities with a more calculated examination of what is useful to him or her.

All Web pages are found by their addresses known as uniform resource locators (URLs). These typically start with <http://www> and the entire address must be exactly entered. WWW homepages often contain sound, color, and graphics. Consequently, many pages may take some time to download into one's computer, thus a fast modem is recommended (2400 baud).

Individuals, organizations, and companies all have a presence on the Web. Many individuals



**Table 2** Listservers for clinical psychology

<i>Organization</i>	<i>E-mail address</i>
Behavior Analysis	behvioraanalsys@mankato.msus.edu behav-an@listlistserv.nodak.edu
Division 12 Net	(for members of the Division of Clinical Psychology, APA) div12@listserv.nodak.edu
Society for a Science of Clinical Psychology	(for members of the Section III of the Division 12, APA) sscpnet@bailey.psych.nwu.edu
Group-Psychotherapy	Majordomo@freud.apa.org (in body of message, type: SUBSCRIBE GROUP-PSYCHOTHERAPY)
Society for Community and Research Action	(Division 27 of APA) SCRA-L@UICVM.CC.UIC.EDU
PsyUSA: Practice-Oriented discussions	(managed by John Roraback: jmroraback@aol.com) PsyNetUSA@aol.com
Autinet (Autism)	autinet-request@iol.ie LISTSERV@sjuvvm.STJOHNS.EDU
Behavioral Disorders in Children	LISTSERV@ASUVM.INRE.ASU.EDU (in body of message type: Subscribe BEHAVIOR username)
Dual Diagnosis	(for professionals with interests in persons with dual diagnoses of mental disorders, drug and alcohol addiction) MIDAA-dual-diagnosis@idealist.com
NetPsy: Internet Psychotherapy	(issues related to psychotherapy services provided via Internet) listserv@sjuvvm.stjohns.edu
PsychNews International	LISTSERV@LISTSERV.NODAK.EDU (in body of message type: SUBSCRIBE PSYCHNEWS Firstname Lastname)
GrantsNet	listserv@list.nih.gov (in the body of the message type: subscribe GNET-L FirstName LastName)

have set up their own homepages containing information about themselves and their interests, businesses, productions, and professional activities. Specific to topics within psychology, professionals and lay people with common interests have established homepages with information about biopsychosocial phenomena (e.g., on dual diagnoses, depression, anxiety disorders, aggression, drug and alcohol addiction). Table 3 provides the Internet addresses for special interest groups.

Similarly, companies offering services or information have also created homepages with book or product catalogs. (We do suggest, until better safeguards are in place, placing any orders for materials through the mail or telephone for credit card number security.) Many colleges and universities also have

homepages with links to other sites within their institution, including descriptions of course offerings, faculty, their expertise and research, and other useful information. Other Web sites contain encyclopedic-type information (e.g., ARGUS/University of Michigan Clearinghouse and The Electric Library). Most importantly, governmental agencies, scholars and practitioners, and scientific and professional organizations have created sites of information containing a great variety of resources in their homepages (and interlinked them with others of similar topics). Table 4 provides the Internet addresses for several organization homepages. These are some illustrative samples, not a comprehensive list, of organizations related to clinical psychology accessible on the Net.

**Table 3** Special interest and discussion groups on the Web.*Homepage association**URL address**The Obsessive-Compulsive Foundation (Resource Center)*<http://www.ocdresource.com>*Society for Research in Adolescence*

(special interest group in peer relations)

<http://www.darling.hhdev.psy.edu/peesig.htm>*Group Psychotherapy*<http://freud.tau.ac.il/~haimw/group2.html>*Behavior OnLine*

(discussions and interviews with practitioners and researchers)

<http://www.behavior.net/>*Dual Diagnosis*

(for professionals with interests in persons with dual diagnoses of mental disorders, drug and alcohol addiction)

<http://www.erols.com/ksciacca/>*The Psychoanalytic Connection*<http://marge.infohouse.com/psacnct/>**1.03.4.5.1 Accessing the World Wide Web**

If the reader is not familiar with accessing the Web, we suggest consulting the variety of books on surfing the Net, although the best way to learn the Net resources, capabilities, and limitations is to take some time to explore it for oneself. We will give here only a brief introduction to accessing the WWW with a focus on clinical psychology resources.

Most novices (and likely more experts) access the Internet for information using one of the popular Web browsers (e.g., Netscape, MS Internet Explorer). There are numerous Web indexes available providing some organization (e.g., the Yahoo index site itself lists and links to over 400 index sites). Programs (called crawlers, robots, spiders, and search engines) allow the user to search through the Web and collect addresses of the information sought through a keyword search; the user can then access the identified sites through pointing-and-clicking (e.g., through Webcrawler, Lycos). These search programs are easily accessed through the Netscape or Mosaic programs. The newest search vehicle is AltaVista, reported to index and search over 30 million documents on the Web out of 225 000 Web servers around the world (Heacock, 1996).

The various sites are, thus, interlinked and permit exploration and searching. The search capacities of the programs or search engines allow the user to enter a keyword or phrase for a search through all the Internet for the concept or exact word/phrase. Often a large number of hits on the topical term will be found. In a search via Yahoo (through Netscape 2.0), we obtained 24 hits or sites with information on the topic of

“clinical psychology.” Searches can be fine-tuned, specified, or narrowed by the inclusion of additional concepts central to the individual’s desired search (e.g., add “psychopathology” or “elderly”).

To demonstrate the wealth of information available, we conducted a search via the bigger programs in Netscape on the keyword of “depression” and to refine the concept, added the phrase “in the elderly.” The results of the various searches include:

*Depression Depression in the elderly*

Yahoo	82 matches	0 matches
Magellan	6640 “results”	12 651 “results”
Excite	over 100 documents	over 100 documents
Infoseek	5681 “best” (searched through 51 191 696 unique URL sites)	13 061 “best”
Altavista	7000 matches	7000 matches

The browser programs organize the site hits using an algorithm, and the results are often ranked by “relevance” or confidence in the “fit to the concept.” Given the ambiguity of the term “depression” and the range of topics in “elderly,” these searches generated quite a few unrelated information sites for such things as “depression era artists,” “depression glass,” “The Great Depression,” and data on a tropical depression off Acapulco. Fortunately, many documents or pages were found for psychological depression. These sites included health center information, on-line depression screening tests, governmental information pages, support groups, professional organizations, consensus

**Table 4** Organization homepages relevant to clinical psychology.

<i>Homepage Association</i>	<i>URL address</i>
<i>Psychology organizations</i>	
American Psychological Association	<a href="http://www.apa.org">http://www.apa.org</a>
American Psychological Society	<a href="http://www.hanover.edu/psych/APS/aps.html">http://www.hanover.edu/psych/APS/aps.html</a>
British Psychological Society	<a href="http://www.bps.org.uk/">http://www.bps.org.uk/</a>
Canadian Psychological Association	<a href="http://www.cpa.ca/">http://www.cpa.ca/</a>
Society for a Science of Clinical Psychology	<a href="http://www.sscp.psych.ndsu.nodak.edu">http://www.sscp.psych.ndsu.nodak.edu</a>
<i>Clinical child/pediatric psychology organizations</i>	
Society of Pediatric Psychology	<a href="http://129.171.43.143/SPP/index.html">http://129.171.43.143/SPP/index.html</a>
University of Minnesota: Pediatric Centers, Policies, and Societies	<a href="http://www.peds.umn.edu/centers">http://www.peds.umn.edu/centers</a>
Children's Defense Fund	<a href="http://www.tmn.com/cdf/index.html">http://www.tmn.com/cdf/index.html</a>
<i>Legal organizations</i>	
ABA Network: American Bar Association	<a href="http://www.abanet.org/home.html">http://www.abanet.org/home.html</a>
LAWlink: ABA Legal Research Selected Starting Points	<a href="http://www.abanet.org/lawlink/home.html">http://www.abanet.org/lawlink/home.html</a>
The ABA Center on Children and the Law	<a href="http://www.abanet.org/child/">http://www.abanet.org/child/</a>
<i>Governmental information sites</i>	
National Institute of Mental Health	<a href="gopher://gopher.nimh.nih.gov">gopher://gopher.nimh.nih.gov</a>
GrantsNet: Department of Health and Human Services	<a href="gopher://gopher.os.dhhs.gov:70/1/Topics/grantsnet">gopher://gopher.os.dhhs.gov:70/1/Topics/grantsnet</a>
FedWorld	<a href="http://www.fedworld.gov">http://www.fedworld.gov</a>
National Institute on Drug Abuse	<a href="http://www.nida.nih.gov">http://www.nida.nih.gov</a>

conference statements on treatment, a number of testimonials (e.g., autobiographies, poetry, photos) of people describing their own depressive episodes and treatment, and a large number of adverts offering psychological services. Some of the information offered treatment approaches or information of questionable accuracy, efficacy, and taste (and relatedly, ethics).

Once the search service (e.g., Lycos, Altavista) presents the results, the user points-and-clicks on each interesting entry and that site is accessed. Because one site is usually linked to several others of similar topics, one may then follow from one piece of information to another. Alternatively, one may return to the list of search results to explore the next entry. Information in the sites can be read at the time, downloaded for later use, or printed into hardcopy. In our search, many of the same sites were uncovered by the various services (but

some search by different mechanisms or in different sources). Going through the multitude of sites identified in the search on "depression" and "depression in the elderly" (or whatever keyword) is eye-straining, if not mind-boggling.

In other keyword searches for clinical psychology topics, we found a similar bewildering, yet potentially helpful, array of information. A search on "panic disorders" turned up the NIH consensus statement and the NIMH patient information pamphlet. A search on "traumatic stress" obtained the homepage for the VAMC in Phoenix with diagnostic and treatment information. "Attention Deficit Disorders" generated a large number of sites on treatments, professionals offering diagnosis and therapy, educational and behavioral assessment, and support services. It is unclear how all of these reflect on the call from some professionals in clinical psychology for validated

assessment and treatment information, but the information is all “out there” for public consumption.

#### **1.03.4.5.2 Collections of resources on the World Wide Web**

Professional groups and individual psychologists have developed a number of WWW sites in which many other homepages are listed for the users to follow up. (Special icons or indicators are used, such as push-pins, highlighting, underlining, or different colored words, to note that a linkage to another set of information or page may be accessed by pointing at the icon and clicking.) A central site listing homepages is a useful place to start in finding information related to psychology in general and clinical psychology, in particular. Following from one site to another is the best way to explore the Web. Once the user has identified some sites as more useful than others, the URLs can be retained as bookmarks for easy access. Table 5 provides the Internet addresses for several resource links. Each of the sites listed includes links to many other sites related to clinical psychology.

Using the Web browser (and various search engines such as Yahoo, Lycos, Magellan, WebCrawler, AltaVista), one can also enter the name of an organization or group having related interests on a topic and find its resource pages of position papers, scientific or professional articles, and other informative documents. The URLs for the APA links to pages of its Public Policy Office and Practice Directorate, while the APS has several psychology links.

Other organizations' homepages, such as the American Bar Association (ABA), maintain interesting and useful Internet resource linkages as well. The ABA entity, for example, links 2200 sites including the Commission on Mental and Physical Disability Law, the Center on Children and the Law, and the Commission on Domestic Violence, as well as numerous publications and position papers. Similarly, the American Medical Association (<http://www.ama-assn.org/>) maintains links to the *Journal of the AMA* and other medical information sites. Other useful homepages include the American Academy of Pediatrics (<http://www.aap.org/>), the American Psychiatric Association (<http://www.psych.org/>), and the American Academy of Child and Adolescent Psychiatry (<http://www.psych.med.umich.edu/web/aacap/>).

Mental health and human services organizations such as The National Alliance for the Mentally Ill (<http://www.cais.com/vikings/nami.index.html>), the National Mental Health

Association (<http://www.worldcorp.com/dc-online/nmha>), and HandsNet (30 000 human service and public interest organizations: <http://www.igc.apc.org/handsnet>) also maintain informational sites.

Governmental agencies are often a font of information through Web homepages (URLs) or Gopher sites. For example, the Center for Substance Abuse Prevention (in the Department of Health and Human Services) maintains the informational service of the National Clearinghouse for Alcohol and Drug Information accessible through the Web (<http://www.health.org/>). Service and research grant information from the Federal government is obtainable through a Gopher search (gopher://gopher.os.dhhs.gov:70/1/Topics/grantsnet). The National Technical Information Services, also known as FedWorld, provides electronic access for health-related information (<http://www.fedworld.gov>), while information about US House and Senate legislation is accessible through the Library of Congress (<http://thomas.loc.gov/>).

#### **1.03.4.6 Limitations**

The rapid rise of the Internet led to great expectations and excitement over its motherlode of information. Increasingly, however, the Internet has been criticized for a number of limitations. A primary criticism is that there is no overview or organization of the material placed on the Web, for example, and that almost anything can be and is out on the Net for public and professional access. In its very foundation and its chaotic development, “management” has been avoided. While this may be seen as an asset, the explosion of information and the huge number of sites has made it difficult to make sense of it all. In contrast to scientific and professional publications that typically have editors and editorial boards for evaluating the credibility of the information prior to publication, most of the material on the Web is not subject to any overview, except in certain instances (such as for some Internet journals).

As an example of how bad information can be disseminated widely, we have received e-mail messages via a large listserver group with repeated claims that there are federal laws requiring that a report of child abuse be made anytime a child client presents with a change in behavior. (This claim is untrue, and several attempts to correct this information were met with harsh and personal attacks from the vociferous poster of the misinformation.) Similarly, Web sites may offer psychotherapy through the Internet or services and products of

**Table 5** Resource links for clinical psychology.

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<i>Homepage association</i>
<i>URL address</i>

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<i>Psychology resources</i>
Psychology Web Pages
<a href="http://www-psy.ucsd.edu/otherpsy.html">http://www-psy.ucsd.edu/otherpsy.html</a>
Psych Web
<a href="http://www.gasou.edu/psychweb">http://www.gasou.edu/psychweb</a>
Psyc Site
<a href="http://www.unipissing.ca/psyc/psycsite.htm">http://www.unipissing.ca/psyc/psycsite.htm</a>
Hanover College Psychology Department
<a href="http://psych.hanover.edu">http://psych.hanover.edu</a>
Mental Health Net
<a href="http://www.cmhc.com">http://www.cmhc.com</a>
PsychScapes Worldwide
<a href="http://www.mental-health.com">http://www.mental-health.com</a>
<i>Informational and related resource links</i>
Clearinghouse for subject-oriented Internet resource guides
<a href="http://www.lib.umich.edu/chhome.html">http://www.lib.umich.edu/chhome.html</a>
THOMAS: Legislative information on the Internet
<a href="http://www.thomas.loc.gov/">http://www.thomas.loc.gov/</a>
Neuroscience mailing lists
<a href="http://www.lm.com/~nab/neurolist.html">http://www.lm.com/~nab/neurolist.html</a>
MedWeb
<a href="http://www.emory.edu/WHSC/medweb.ejs.html">http://www.emory.edu/WHSC/medweb.ejs.html</a>

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uncertain ethical or probative value. Indeed, there is no requirement that any Web site contain truthful or useful information. Thus, in our view, the warning of *caveat emptor* (buyer beware) extends to the free access to the Web information sites.

How does one sort through this range of information? As with other professional literature, we suggest the reader consider the source and apply one's professional training of evaluation. Heacock (1996) suggests that many people "have been led to believe that the Internet (and more specifically, the World Wide Web) is some kind of exhaustive library of human knowledge, that anything you want to know is out there, and that there is some simple, logical way to find it. Nothing could be further from the truth" (p. 2D). Thus, Web searching may result in an overabundance of undigested and untested information. The old saying about the "need to separate the wheat from the chaff" is quite applicable here.

### 1.03.5 EVALUATING THE RESOURCES

Evaluating the vast array of reference materials for clinical psychology may be overwhelming, perhaps similar to "being asked to climb Mount Everest without a Sherpa guide" (Rosnow & Rosnow, 1995, p. 19). It is our conclusion that most of the informational

resources available to clinical psychologists are good. The difficulty is, then, in assessing the quality of unknown information and locating appropriate references.

With respect to quality, the peer-review process provides one mechanism by which the caliber of research literature may be kept high. "Refereed" journals, where others knowledgeable about the topic review each article prior to publication, are common, although not universal. Unfortunately, this system also results in many potentially important research reports not being published as the average rejection rate for journals is about 75% (Salkind, 1994). In contrast, most books do not undergo a similar pre-publication evaluation. Thus, it behooves the scholar to keep a skeptical eye open when evaluating the utility of any given resource, be it journal, book, or Usenet newsgroup.

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# 1.04

## Contributions of Behavioral Genetics Research to Clinical Psychology

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## 1.04.1 INTRODUCTION

There have been numerous significant advances in our understanding of the nature of mental disorder in recent years, and considerable progress derives from the contributions made by behavioral genetic research. Research findings from behavioral genetics have substantially altered how we understand: the risks for major mental illnesses such as schizophrenia, depression, and bipolar illness; personality disorders and dimensions; disorders of childhood and adolescence; stability and change in psychopathology; and the nature of psychosocial risks for psychopathology. No longer a peripheral issue, behavioral genetics has made lasting contributions to the field of clinical psychology. This is perhaps most obviously indicated by the inclusion of a chapter on behavioral genetics in the “Foundations” volume.

Writing a foundations chapter in behavioral genetics is complicated for many reasons. First of all, the field of behavioral genetics is, by design, multidisciplinary and complex. At a very basic level, behavioral genetics provides a conceptual and methodological bridge between the biological and the behavioral sciences. Conceptually, behavioral genetics hypothesizes test whether individual differences in a behavioral phenotype can be attributed to genetic and environmental influences. Methodologically, behavioral genetics is the research tool that

specifies the samples and methods necessary to answer conceptual questions. A second difficulty in writing this chapter is that there are already a number of excellent texts that introduce many of the key methodological and conceptual issues. In particular, the third edition of *Behavioral genetics* (Plomin, DeFries, McClearn, & Rutter, 1997) introduces many central concepts and methods in behavioral genetics research and reviews of many of the more challenging findings. In addition, other well-received sources focus on more specific areas within behavioral genetics, such as the role of genetic factors in major mental illness (Gottesman, 1991) and childhood psychopathology (McGuffin, Owen, O'Donovan, Thapar, & Gottesman, 1994; Rutter et al., 1990). Still other volumes provide an overview of the recent and ongoing questions of behavioral genetics in psychology (Loehlin, 1992; Plomin, 1994; Plomin & McClearn, 1993). Technical texts on statistical genetics (e.g., Neale & Cardon, 1992) and molecular genetics are also available. In this chapter, we incorporate lessons from each of these perspectives, provide an update on more recent findings with major implications for research on psychopathology, and mold it in a form that is pertinent to clinical psychology.

An additional major challenge for a review chapter is the amazing speed with which the field of behavioral genetics is progressing. Research reviews on the genetics of one area or another



published just a few years ago are already outdated. Nowhere is this more apparent than in the field of molecular genetics and the interface between molecular genetics and psychiatry. Accordingly, our primary focus is on the conceptual underpinnings of behavioral genetics. An effort is made to review key findings, but we avoid an encyclopedic approach in which the genetics of all disorders is discussed. Chapters that focus on specific disorders in this series will address genetic influences.

A further orienting remark concerns the perspectives from which we review the relevance of behavioral genetics for clinical psychology. We view behavioral genetics research as relevant not only to our understanding of genetic influences on development, but also to our understanding of psychosocial factors. A theme emphasized throughout the chapter is that behavioral genetic studies provide one of the best handles on environmental influences. A second perspective is that we adopt a developmental approach. The reasons for this are rooted not only in our background as developmental psychopathologists, but also in the fact that empirical studies (behavioral genetic studies included among them) repeatedly emphasize that much can be learned about the behavioral phenotype, risk and protective factors, and mechanisms underlying the development of disorder when clinical phenomena are observed longitudinally.

This chapter is divided into six sections. First, in order to place the current set of findings and methods in context, we briefly highlight the history of behavioral genetics research. Classic papers and exemplary studies that challenged basic notions of child and adult development are emphasized. The central theme in this section is that the pendulum that has swung back and forth between nature and nurture—the terms coined by Francis Galton over a century ago—appears to have settled to a happy equilibrium. Second, we review some of the core conceptual and methodological issues in behavioral genetics. Some of the central issues are included here for readers who may not have come across these ideas previously. In particular, we seek to clarify some of the popular controversies and misunderstandings and differentiate those debates that have been largely resolved from those that require additional attention. Third, we examine how behavioral genetics research has contributed to nosological debate and a number of related issues (e.g., comorbidity) and whether psychopathology is qualitatively different from the normal range.

Fourth, the ways in which behavioral genetics research has contributed to our understanding of development, and to developmental psycho-

pathology in particular, are delineated. The penultimate section introduces the burgeoning field of molecular genetics and its links with clinical psychology and psychiatry. Such is the influence and momentum of molecular genetics that a chapter on the foundations of behavioral genetics and clinical psychology would surely be incomplete without a general discussion of the methods and emergent findings. Finally, the last section discusses some of the implications of behavioral genetics methods and findings for clinical psychology.

## **1.04.2 HISTORICAL CONTEXTS AND CHANGING CONCEPTUALIZATIONS OF BEHAVIORAL GENETICS AND PSYCHOLOGY**

### **1.04.2.1 The Changing Dynamics of Behavioral Genetics Research**

The current prevalence of behavioral genetics research in a very wide array of journals is impressive. Indeed, it is difficult to avoid reading behavioral genetics studies in mainstream clinical, personality and developmental psychology journals such as *Journal of Abnormal Psychology*, *Journal of Personality and Social Psychology*, *Developmental Psychology*, or in journals of disciplines that have traditionally been eager to criticize and slow to adopt this approach (e.g., *Journal of Educational Psychology*). The current state of affairs belies the difficult history of behavioral genetics research as applied to psychology and psychiatry.

Curiously, historical assessments of the receptiveness of the general and scientific audience to genetics research in psychology reveal not only outright dismissal, but also over-eager acceptance. The pendulum metaphor as applied to the last half-century, with different swings punctuated by key studies, has been discussed (Plomin & McClearn, 1993). In addition, Kimble (1993) provides an overview of some of the key epistemological issues underlying the history of nature–nurture in psychology. In recent years a critical mass of findings has accumulated to permit a reasoned debate of the conceptual and methodological implications of behavioral genetics research. Many of these debates have been published (e.g., Detterman, 1996; Plomin & Bergeman, 1991, and commentaries; Scarr, 1992). It is indeed encouraging that behavioral genetics research has attracted attention and spurred discussions from psychologists from diverse perspectives (Plomin & McClearn, 1993).

Fortunately, the most recent iteration in the nature–nurture debate has moved beyond

discussing *whether* genetic influences are significant in behavioral development. Instead, questions now focus on *how* genetic factors exert their influence. The question of “how” to integrate genetic and environmental factors in developmental theory is not new. Anastasi’s (1958) recently exhumed paper raised this issue most directly, and subsequent papers also emphasized the need for a détente or rapprochement between competing genetic and environmental models of development and psychopathology (e.g., Gottesman, 1974; Scarr & McCartney, 1983; Turkheimer & Gottesman, 1991). However, it is only relatively recently that we have been able to come to grips with the empirical analyses needed to answer this question. Some of the important inroads are discussed in Section 1.04.5.

#### **1.04.2.2 Key Studies in Behavioral Genetics Research**

Central to the pendulum metaphor noted above are key papers, conferences or books that substantially changed the way in which the behavioral genetics link to clinical psychology was understood. A full discussion of this issue is not within the scope of this chapter, but it is instructive to cite a few examples.

##### **1.04.2.2.1 Genetics and major mental illness**

Although genetic influence on schizophrenia (and most major mental disorders) has been widely accepted for many years, this was not always the case. The critical studies to demonstrate the role of genetic factors appeared in the late 1960s and early 1970s (see Gottesman, 1991). As Gottesman (1991) notes, researchers studying schizophrenia were “converted” by the remarkable findings reported by Heston (1966) and others in a ground-breaking conference in 1968.

##### **1.04.2.2.2 Genetics and development**

Several key papers ushered behavioral genetics research into “mainstream” psychology and psychopathology research. Behavioral genetics had received some attention in developmental psychology up to the early 1980s, but the influence was largely limited to certain areas such as temperament and cognitive abilities. A special issue of *Child Development* in 1983 (Plomin, 1983) was devoted to a relatively wide range of topics in which behavioral genetics findings offered important insights into developmental processes underlying change and stability and models that sought to integrate genetic and environmental factors from childhood through adolescence. Subsequent pub-

lications dealt more extensively with how development could be studied from a behavioral genetic perspective (e.g., Plomin, 1986).

##### **1.04.2.2.3 Genetics and the environment: sibling differences in adjustment**

One of the most significant advances in behavioral genetics research concerns not genetics but the environment. Few findings illustrate this point better than the remarkable finding that simply growing up together in the same home does not make siblings (very) similar. Correlations between siblings on a range of personality and other indicators in individual differences research (with the notable exception of cognition) are remarkably weak, and usually less than 0.3. Leaving aside methodological issues such as measurement error, this low correlation suggests that siblings are quite different, and this has led to the search for factors that make siblings different from one another, that is “nonshared environment.” A target article and attached commentaries to Plomin and Daniels’s (1987) paper published a decade ago still provides a useful introduction to this issue, and continues to be widely cited in research on family influences, sibling relations and children’s psychosocial development.

It is now accepted that nonshared environment, and particularly differential parental treatment, has provided critical insight into the socialization influences within families. Parents do treat their children differently, because of age differences, triangulated family alliances (Vogel & Bell, 1960), or other reasons. Phenotypically, this differential treatment of sibling children has been linked to problems in adjustment. Not surprisingly, a child who has a more negative relationship with his or her parent compared with a sibling, is more likely to have higher levels of behavioral and emotional problems (Dunn, Stocker, & Plomin, 1991; Plomin & Daniels, 1987; Rodgers, Rowe, & Li, 1994). The extent to which the effects of differential treatment are moderated by siblings’ perceptions and family context has also been examined (McHale & Pawletko, 1992; Quittner & Oipari, 1994).

Although not usually explicitly tested, the prevailing assumption in social development research was that there was no need to include more than one child per family. Implicitly, the hypothesis was that one child was an adequate “proxy” for processes affecting other children in the family. For example, in research on marital conflict and children’s disruptive behavior only one child per family needed to be studied because the focus was on the between-family effects—the association between individual differences in

marital conflict and individual differences in children's disruptive behavior across families. However, the implication of nonshared environment is that simply "sharing" parental conflict does not make siblings similar to one another. In addition to encouraging a search for familial and nonfamilial influences that would make siblings different from one another, the possibility was raised that siblings adjusted differently to the same stressor. Several studies now support the role of nonshared environment (Plomin, 1994), and there is a suggestion that siblings are differentially affected by family stresses hitherto thought to be "shared" (O'Connor, Hetherington, & Reiss, in press). Accordingly, it is no longer sufficient to examine just one child per family; assessing the impact of how siblings are similarly or differentially affected by stresses can inform our understanding of how psychosocial risks operate.

#### ***1.04.2.2.4 Genetics and the environment: environments are not independent of genetic "effects"***

A second major insight concerned the way in which genetic influences were conceptualized: so-called environmental influences are not independent of genetics (Plomin & Bergeman, 1991). That is, environments do not just "happen." Instead, the way in which individuals actively select and respond to their environment is correlated with personality, temperamental, and intellectual abilities that are genetically influenced. In other words, environments are not randomly distributed but correlated with genetic factors (Rutter, 1991).

The ways in which genetic influences shape how individuals approach their environments has been discussed at a conceptual level (Plomin, DeFries, & Loehlin, 1977; Scarr & McCartney, 1983), but there were relatively few empirical examples that directly assessed the hypothesis (Lytten, 1977; Plomin & Rowe, 1979) until the 1980s (Plomin & Bergeman, 1991; Rowe, 1981). Numerous studies published subsequently provide support for their hypothesis (Kendler, 1995, 1996; Plomin, Reiss, Hetherington, & Howe, 1994). Lessons about how genetic factors influence experience are discussed in detail in Section 1.04.5.

#### ***1.04.2.2.5 Progress in animal research***

The history of behavioral genetics research needs also to include advances made in research using animal models, in which both genotype and environment can be manipulated. The development of animal models has long been considered an important step in studying

disorders and diseases such as depression, Alzheimer's disease, alcoholism, and schizophrenia. Genetic research using animal models found evidence for genetic influence in areas such as learning, behavioral effects of drugs, and social behavior, long before similar conclusions were reached in human research (Plomin et al., 1997; Scott, 1977). Animal model research is currently leading the way towards the integration of molecular genetics and psychology, especially in the area of psychopharmacology (Crabbe, Belknap, & Buck, 1994; McClearn et al., 1997), but also in other areas such as fearfulness (Flint et al., 1995).

#### ***1.04.2.2.6 Psychometric developments in behavioral genetics***

A final area that deserves historical attention is the psychometric progression underlying behavioral genetics research. A brief overview of the key papers and iterations is provided here because behavioral genetics is intensely quantitative, and improved ability to test genetic hypotheses came about only following quantitative advances.

Partialling variance in a phenotypic trait into genetic and environmental factors, the hallmark of behavioral genetics research and an analytic approach that leads to statements regarding heritability, was laid out by Fisher over a half century ago (see Jinks & Fulker, 1970). Falconer (1960) compiled available evidence and distilled the basic elements of research design that formed the basis for quantitative genetics (see Loehlin, 1992). Subsequent contributions that had a major impact on behavioral genetics research were both computational and conceptual in origin. Most notably, Jinks and Fulker (1970) provided the quantitative foundations for contemporary approaches to regression analysis and model-fitting. More recently, DeFries and Fulker (1985) developed a way of examining whether genetic influences are greater for extreme (clinical) groups than for groups within the normal range.

Additionally, because behavioral genetics analyses are computationally intense, the development of software packages has also had much to do with the accessibility of behavioral genetics research approaches (Joreskog & Sorbom, 1989). Moreover, criticisms of the basic assumptions of the twin and other research designs needed to be empirically assessed, such as influences of assortative mating, the equal environments assumption in twin studies, selective placement in adoption studies, and differentiating nonshared environment from error of measurement (McArdle & Goldsmith, 1990).

Conceptual questions regarding longitudinal analyses, analyses of latent traits, and multivariate genetic analyses were also asked, and similarly required new analytic models. Fortunately, the methods to model these sources of influence are now readily available in Neale and Cardon's (1992) comprehensive book.

Finally, molecular genetic research on complex dimensions and disorders required new analytic paradigms from single-gene approaches (Plomin, Owen, & McGuffin, 1994). The essence of these methods is to identify genes in multiple-gene systems that are of modest effect size, which have been called quantitative trait loci (QTL) or susceptibility genes. These advances include QTL linkage approaches that attempt to locate general regions of a chromosome where a QTL resides (Fulker & Cherny, 1996) as well as association analyses that correlate candidate genes with complex traits (e.g., Risch & Merikangas, 1996).

#### **1.04.2.3 Controversies Old and New**

Misunderstandings of what research findings from behavioral genetics do and do not imply have led to several controversies, some of which continue to linger. Rutter (1991) outlines many of the misconceptualizations of behavioral genetics research in psychology and psychiatry. One of the most frequent, and perhaps most important, areas of misunderstanding concerns what behavior genetic research examines: individual differences. That is, why do individuals vary, for instance, in their likelihood of approaching and conversing with strangers, performing well on ability tests, or experiencing depressive symptoms.

The magnitude of genetic influence on individual differences does not readily translate into a second paradigm in psychological research: group differences, or why two groups of individuals may differ in, for example, their susceptibility to alcoholism. Despite efforts to clarify the marked contrasts between these approaches, examples of misunderstanding of the distinction between research on individual differences and group differences continue to appear in the scientific and popular press. A notable misunderstanding of genetic methods is made in the attempts to link ethnic differences on ability tests and the findings that scores of ability tests are partly genetically influenced to conclude that group differences in scores are genetically determined. Although our focus is on an individual differences approach to development and psychopathology, it is not the only way that genetic influences operate. Several authors have attempted to integrate

research on individual differences with models of species-typical behavior and processes (Buss, 1991).

A second but related source of confusion is heritability, the degree of genetic influence on individual differences. Heritability is a population parameter derived from a particular sample at a particular time. Although estimating heritability is often a "bottom line" of the majority of behavioral genetics research, there is no "true" heritability estimate, just as there is no "true" estimate of the effects of any descriptive statistic such as the correlation between life events and depression. Like any other statistic, heritability can vary depending on sample characteristics, methodology, and historical context.

A further fundamental aspect of understanding heritability is that genetic influences are not fixed, nor is it the case that strongly heritable forms of psychopathology will not respond to environmental input. Thus, there is no necessary relationship between genetic influence and psychosocial susceptibility. Numerous examples of secular changes in aspects of development and psychopathology help illustrate this point (e.g., see Rutter & Smith, 1995).

#### **1.04.2.4 Summary**

Not surprisingly, controversy remains regarding the conceptualization of genetic influences in the development of psychopathology. Fortunately, however, there is now good reason to suspect that the dialogue between behavioral geneticists, developmental psychologists, and clinical psychologists and psychiatrists is increasing and has reached a critical mass (Reiss, 1995). The kinds of conceptual questions now asked in behavioral genetics research are more sophisticated than those asked a decade ago, and there is far greater interest in combining analytic strategies to test competing theories of psychopathological processes (Rutter et al., 1997). Moreover, there are a number of investigations currently underway that are likely to provide important clues of how genetic factors are involved in the development of psychopathology (Reiss et al., 1995). In the remainder of the chapter we examine these advances and highlight new research directions.

### **1.04.3 A SELECTIVE OVERVIEW OF METHODOLOGICAL ISSUES IN BEHAVIORAL GENETICS RESEARCH**

As in every other branch of psychological research, behavioral genetics investigations are

limited by methods and design. The mechanics of the designs used in behavioral genetics research have been discussed widely (Loehlin, 1992; Plomin et al., 1997; Rutter et al., 1990), and will therefore receive only limited attention here. Rather than discuss the actual methods in detail, we instead emphasize the hitherto largely underappreciated relative strengths and weaknesses of certain designs in light of current models of genetic and environmental influences on psychopathology.

#### 1.04.3.1 A Précis of Quantitative Genetics

The first point to make is that genetic influences, or the role of individual differences in genotype, are estimated based on phenotypic or observed patterns of similarity in genetically informative designs that vary genetic or environmental relatedness. Given the frequent misinterpretation of behavioral genetics findings, we briefly discuss how empirical findings allow us to make inferences regarding the role of genes and the environment.

Behavioral genetics decomposes individual differences in the variation of behavior into genetic, shared environment, and nonshared environmental sources of influence. Genetic influence refers to inherited genetic differences that contribute to observed (phenotypic) differences among individuals. For identical twins, these genetic factors contribute entirely to their phenotypic similarity, while for first-degree relatives (parents, offspring, and siblings), who are 50% similar genetically, additive genetic factors contribute to phenotypic similarity and differences in equal measure.

One way to determine genetic influence is to compare identical (monozygotic or MZ) and fraternal (dizygotic or DZ) twins. If identical twins are more similar than nonidentical twins, it follows that they may be more similar because they are more similar genetically. The extent to which genetic influences are important can be quantified because we know that MZ twins share all of their genes and DZ twins are, on average, 50% similar genetically. Doubling the difference between MZ and DZ correlations estimates heritability, or  $h^2$ , the proportion of phenotypic variance that is explained by genotypic variance. Comparing adopted-apart MZ and DZ twins (that is, pairs that were raised in different homes) provides a rare but even more powerful way of estimating genetic influences, that “controls” for shared family environment.

Shared environmental influences can also be straightforwardly assessed. The magnitude of shared environment is most easily seen by

comparing unrelated siblings, as in the adoption design. The adoption design is particularly powerful because it “controls” for genetic similarity (i.e., adopted siblings are genetically unrelated). Because unrelated siblings do not share genes, any similarity must be attributed to common environment. Finally, the magnitude of nonshared environment is most easily seen in the differences within pairs of identical twins. Because MZ twins are genetically identical, any differences must be attributed to different experiences (and measurement error).

As a general point, it is noteworthy that whereas it is possible to examine the magnitude of the three parameters assessed in behavioral genetics research (genetic, shared and nonshared environment), different designs have different strengths and weaknesses and are differentially affected by, for instance, gene–gene interactions (which are included in the similarity of MZ twins because they are genetically identical for all genes) and gene–environment correlations (which are differentially assessed in adoption and twin studies). This helps explain why different results are sometimes obtained from twin and adoption designs and why no design should be taken as the absolute estimate of these influences.

#### 1.04.3.2 An Overview of Research Designs to Detect Genetic and Environmental Influence

##### 1.04.3.2.1 Twin studies

The most frequent form of behavioral genetics design is the twin study. Historically, twin studies have provided critical initial findings that have fundamentally altered debates regarding genetic influence on schizophrenia (Gottesman, 1991) and autism (Folstein & Rutter, 1977), among other disorders. To date, twin studies provide data on a wide range of psychiatric and psychological disorders and dimensions virtually across the lifespan (Bouchard & Propping, 1993; Eaves et al., 1997; McGuffin et al., 1994). As noted above, studying twins is perhaps the best way to assess the role of nonshared environment independent of genetic effects. Pike, McGuire, Reiss, Hetherington, and Plomin (1996) exploited this issue directly in their search of nonshared environmental influences that underlie MZ differences in behavioral adjustment in adolescence. They found that differential parental negativity and warmth could explain differences between MZ twins in rate of problem behavior.

Further examples of exploiting discordant MZ twin pairs has provided important insights into which biological substrates associated with

psychopathology might (or might not) be under genetic control. For example, Torrey, Bowler, Taylor, and Gottesman (1994) reported that identical twins discordant for schizophrenia also differed in brain structures implicated in its neuropathology. This finding suggests that genetic influences on schizophrenia are unlikely to be mediated through genetic effects on brain pathology.

#### **1.04.3.2.2 Adoption studies**

Adoption studies have an equally revered place in behavioral genetics research on psychopathology. For instance, it was the finding that children of schizophrenic mothers who were adopted away (and living with nonschizophrenic families) were at increased risk of developing schizophrenia that led to a major shift in how the causes of schizophrenia was conceptualized (Heston 1966; see Gottesman, 1991). Furthermore, adoption studies based on registries in Denmark and Sweden have proved indispensable in our understanding of genetic influences on alcoholism and antisocial behavior, among other disorders (Cloninger, Bohman, & Sigvardsson, 1981). Adoption designs are also well suited to assess genotype–environment correlations and interactions, a topic discussed below.

#### **1.04.3.2.3 Family history studies**

Family history designs, which also have a long history in psychiatric research, examine the extent to which family members, usually first degree relatives of a proband, or the “target” patient, are affected by a disorder. The central drawback of the family history method is that it is impossible to explain why, in terms of genetic and environmental influences, family members may be similarly affected. Depression, for instance, may run in families because family members share genes or because they share the psychosocial stresses associated with depression (low socioeconomic status, family conflict). Nonetheless, family history studies have made several important contributions to our understanding of genetic influences on psychiatric conditions, for example in the area of heterogeneity (see Coryell, 1997) and age of onset (e.g., Weissman et al., 1984) among others. A final advantage of the family history method is that it allows for the test of genetic hypothesis without requiring that investigators ascertain a sample of twins or adoptive families. It is therefore possible that a genetic component could be added to existing studies of environmental risk for psychopathology. Psychopathology in first-degree relatives can be used as a proxy for

genetic risk (although, of course, it also indexes environmental risk), and it would then be possible to assess the extent to which there is overlap between genetic and environmental risk factors for psychopathology.

#### **1.04.3.3 Alternative Research Designs**

The designs noted above are the most common, but no means the only way of assessing genetic influence. A recent addition to the armory of behavioral genetics designs is the half-sibling or stepsibling design. The viability of the stepsibling model follows from two major demographic changes affecting families since the mid 1970s. First, the number of adoptions has steadily decreased, and it is especially difficult to ascertain representative samples of adoptive children and adoptive families. The second demographic trend is that the number of divorces and remarriages and, consequently, the number of half- and unrelated siblings in reconstituted families has increased dramatically (see Reiss et al., 1994). The stepsibling design therefore has the unique advantage of uniting the diverse perspectives of behavioral geneticists and family researchers.

From a quantitative genetic perspective, the half- and stepsibling design is as informative as a twin or adoption design because it is possible to estimate the degree to which sibling similarity is explained by known genetic similarity (full siblings are 50% similar genetically, half-siblings are 25% similar genetically). There is evidence that this approach may be a reasonable addition to twin and adoption studies. The most encouraging evidence comes from research on intelligence. When similarity of relatives’ intellectual ability is assessed across numerous types of consanguinity, results from half-siblings and cousins complements and does not detract from the overall pattern of results (Bouchard & McGue, 1981). Qualified encouragement for the half/unrelated sibling design comes from the Nonshared Environment and Adolescent Development project (NEAD) (Reiss et al., 1994), the first study to combine the twin and stepsiblings in the same design (Neiderhiser, et al., in press). Other studies employing the half-sibling design have endorsed this strategy for other behaviors (e.g., Van den Oord & Rowe, 1997).

The stepsibling design provides a further parallel to the adoption design in that it is possible to ascertain the magnitude of genetic effects by comparing biological parent–child correlations with stepparent–child correlations. In quantitative genetic terms, the adoptive and stepparent–offspring correlations

are identical in one respect, i.e., the correlation can be explained only by shared environmental influence.

There are, of course, a number of limitations that complicate interpretation of the stepsibling model results, including potential assortative mating differences in first and second marriages, variation in length of time sharing a household, and elevated stress in stepfamilies. However, there are clear benefits of this strategy, such as examining how environmental factors (e.g., family context) modify genetic influences.

The final point regarding research design is that studies of biological families can tell us surprisingly little about mechanisms and processes in development (Scarr, 1992). The reason is that, as reviewed in Section 1.04.5, findings from biological families cannot distinguish between genetic and environmental processes, nor can they identify the ways in which these influences correlate and interact. Thus, behavioral geneticists and psychosocially minded investigators need to be equally concerned about the use of genetically informative designs to answer questions of causality (Plomin, 1983).

#### 1.04.3.4 Methodological Criticisms of Behavioral Genetics Methods

Criticisms have been raised, and are continuously raised, about the methods used to make conclusions about the impact of genetic factors on development. We briefly review these criticisms and outline the implications for clinical research.

One criticism of the twin method concerns the "equal environments" assumption, that is MZ twins are assumed to experience environments that are no more similar than DZ twins. We now know that MZ twins do in fact receive a more similar familial and extrafamilial environment than DZ twins (Plomin, 1994). In this sense, then, there is not equal similarity in the environments experienced by MZ and DZ twins. But the issue is not whether this is the case, but whether parental differences in treatment are the cause or the effect of twins' differences in behavior (Lytton, 1977). Only if the former is true is the equal environments assumption violated.

A separate issue is whether there are features about being a twin that are fundamentally different from being a singleton. If so, then again we would be concerned about generalizing findings from twin studies. Relatively few factors distinguish twins from nontwins. For example, there is a suggestion that twins may be more likely to exhibit language difficulties and

experience perinatal complications (Rutter & Redshaw, 1991), and these factors may have implications for the development of psychopathology. Some studies also suggest that twins appear to be at slightly elevated risk for conduct or other behavioral problems compared with singletons, but other studies suggest the opposite (see Simonoff, 1992). Additionally, mothers of twins are more likely to experience depression than parents of closely spaced children (Thorpe, Golding, MacGillivray, & Greenwood, 1991), and there may be twin-specific processes that increase the risk for antisocial behavior, such as imitation (Carey, 1992). This is an important finding given the association between maternal depression and children's adjustment problems. To be sure, the assumption that findings from twins can be generalized to the larger population should not be accepted uncritically, and further research is warranted (Rutter, Simonoff, & Silberg, 1993), but there are good reasons for accepting twin study results in research on psychopathology. A more recent methodological issue in twin studies is chorionic status, as MZ twins can be either monozygotic or dizygotic but DZ twins are always dizygotic.

A number of criticisms have been raised regarding adoption study design as well. Perhaps the most important of these concerns the association between the biological and adoptive family characteristics, or selective placement. Selective placement in which children from high (or low) genetic risk are placed into families at high (or low) environmental risk confounds these sources of risk that adoption designs are designed to disentangle.

Assortative mating, the correlation between spouses, is an issue relevant to both twin and adoption studies. The net effect of assortative mating for genetic studies is that it will deflate heritability estimates in twin studies (because DZ twin correlations will be increased but MZ correlations will be unaffected) and increase parent-offspring correlations between one biological parent and offspring. Although assortative mating is not usually found for personality (see Loehlin, 1992), it is common for educational attainment. For psychopathology, there is an elevated concordance between husbands' and wives' levels of psychopathology for depression and antisocial behavior (e.g., Krueger, Moffitt, Caspi, & Bleske, in press), but it is important to consider that assortative mating does not necessarily imply assortment on the same form of psychopathology (see Mednick, 1978).

A further methodological concern is that different research designs often produce different estimates for genetic and environmental

influences. Of course, there is no reason to expect that different designs would necessarily produce the same results. Nonetheless, attempts to include data from adoption and twin studies are relatively rare (Bouchard & McGue, 1981; Miles & Carey, 1997). Where differences are found among research designs, the general pattern is for twin designs to produce the largest genetic effects. Of course, it may be that the greater genetic effects derived from twin studies is a real finding, attributed to non-additive genetic influences and epistasis that are best assessed by studying twins. That there is variation among different genetically-informative designs, and that this variation may be both substantive and methodological in nature, cautions against over-reliance on a single research design and the need to incorporate multiple designs in the same research program.

#### 1.04.3.5 Methodological Recommendations

Just as behavioral genetics research findings provide clues to the role of both genetic and environmental factors, findings from these studies also suggest methodological recommendations for future studies of mechanisms in development and psychopathology.

##### 1.04.3.5.1 Multiple measures

Methodological problems such as rater bias and other forms of measurement error in behavioral genetics research complicate the interpretation of findings. This is particularly true in research on children and adolescents, in which it is customary to gather information from multiple sources. Were multiple sources of data routinely collected in research on adult psychopathology, the same problems would inevitably result. Rater effects have predictable influences on the relative magnitude of genetic and shared and nonshared environmental estimates. Specifically, compared with child self-report data, there is a general tendency for parent ratings to indicate more shared environment and less nonshared environmental effects; the differences in the magnitude of the genetic parameter among raters is somewhat less predictable. In a recent meta-analysis of behavioral genetics studies of aggression, Miles and Carey (1997) found that methodological differences across studies was as important a contributor to the overall pattern of results—almost as large as the genetic parameters! In other words, there is almost as much variation attributed to methods as to genetic influences (although this was true more for the shared environmental parameter than the genetic

parameter). A somewhat exaggerated example of rater effects is given by Thapar and McGuffin (1995). They reported that the heritability of anxiety symptoms in children was 59% according to parent reports but 0% according to children's self-reports! The low agreement invariably found among different reporters in research on psychopathology in childhood is not just an issue of rater bias, because data from different reporters may lead to different substantive findings.

However, even when multiple sources of data are simultaneously assessed—an attempt to analyze an error-free latent construct defined by what is common among raters—the results are not necessarily any clearer. An example of this approach is provided by Simonoff, Pickles, Hewitt, and Silberg (1995), who examined adolescent self-reports and mother and father reports of adolescent behavioral problems. In addition to assessing the genetic–environmental pattern for the common variance among reporters (i.e., the latent trait), these investigators also examined the genetic–environmental pattern *specific* to each rater. Thus, these authors hypothesized that genetic (and environmental) influences on children's behavioral problems could be detected by mothers but not by children or fathers. It is not clear, however, how to interpret mother-specific genetic effects on children's behavioral problems. To date, no satisfactory way of handling method effects has been proposed. What is clear is that single-reporter designs may suggest misleading substantive conclusions, particularly in research on childhood psychopathology. Consequently, multiple-reporter designs should be considered and results based on different designs compared.

##### 1.04.3.5.2 Specification of individual-specific environmental risks

A wealth of research demonstrates that environmental risks that are shared by siblings, or what is termed “shared environment”, play little role in the development of psychopathology. The one robust exception to this rule is conduct disorder and aggression, for which there is substantial evidence that simply growing up in the same home (and the concomitant sharing of psychosocial risks such as poverty and neighborhood violence) makes siblings similar beyond the effects of genetics (McGuffin et al., 1994; O'Connor, McGuire, Reiss, Hetherington, & Plomin, 1998; Plomin et al., 1997). In general, however, environmental influences that are salient for development appear to make siblings different from one another.



A central implication of this finding for research is that risk needs to be conceptualized as individual-specific. That is, there is a need to assess the actual impact of the putative risk factor on the individual's construction of events. For example, rather than define parenting risk in terms of global parent attitudes, it is important to assess the parent's attitudes towards the specific child, how the parent acts toward the specific child, how parenting to the target child differs from parenting experienced by siblings. The bottom line of much of the research on nonshared environment is that risk processes occur on an individual-by-individual basis and not on a family-wide level (Plomin, 1995). A corollary is that it is not sufficient to equate exposure to risk with the effect of the risk.

#### 1.04.3.6 Summary

A number of strategies are available to estimating the effects of genes and environment in behavioral development. Previous behavioral genetics research has underscored the methodological strengths and weaknesses of each approach and has illustrated a number of lessons for researchers seeking to integrate genetic and environmental mechanisms in the development of psychopathology.

### 1.04.4 THE CONTRIBUTIONS OF BEHAVIORAL GENETICS TO RESEARCH ON CLINICAL PHENOTYPES

Having discussed the historical change in behavioral genetics research and some of the central methodological issues in interpreting behavioral genetics findings, we now turn to an overview of the ways in which behavioral genetics research has contributed to our understanding of clinical phenotypes. Our review focuses on five areas: etiology, the continuity/discontinuity debate, comorbidity, heterogeneity, and stability and change.

#### 1.04.4.1 A Selective Review of Genetic and Environmental Etiology in Psychopathology

Genetic influences appear to play an important role in the development of most forms of psychopathology. For example, research consistently shows higher concordance of schizophrenia among MZ than among DZ twins and elevated rates of psychopathology among children adopted away from disordered biological parents, to name just two examples.

As we discuss in more detail below, the major questions for genetic research in psychopathology are no longer whether or not (or how much) a disorder is genetic, but what features of a disorder are genetic (e.g., onset and course, heterogeneous subtypes, response to treatment), how does genetic research inform nosology and treatment, what is the nature of genetic risks (e.g., do they influence disorder, core symptoms, or other risks), and how do genetic and environmental risks jointly lead to maladjustment. These questions shape the discussion in this and the following section of this chapter.

Rather than discuss the forms of psychopathology that show genetic influence (this would take an inordinate length of space, and is covered in chapters on specific manifestations of psychopathology), we highlight the more recent and more challenging findings. Several texts and papers describe in detail genetic influences on a range of disorders in childhood and adolescence (for depression, Tsuang & Faraone, 1990; for schizophrenia, Gottesman, 1991; Karayiorgou & Gogos, 1997; for aggression, Bohman, 1996). General overviews are provided by Plomin et al. (1997), and more specific volumes are also available (Gershon & Cloninger, 1994). What follows is an overview of the key conceptual questions for understanding psychopathology for which behavioral genetics research provides unique insights.

It may not be a surprise to learn that many forms of psychopathology are genetically influenced, but it may be surprising to learn how early in development behavioral problems can be attributed to genetic etiology. Several studies identify relatively robust genetic influence on externalizing and internalizing symptoms from age 2–3 (e.g., Van den Oord, Verhulst, & Boomsma, 1996). It is not yet known how (or if) genetic influence on behavioral problems can be differentiated from the genetic influences on temperament at this age, to what extent the results may reflect strong parental reporter effects (i.e., self-reported symptoms of psychopathology are not reliable before late childhood), or the degree to which the picture might be different if extreme cases were included (the above studies were based on normal risk groups and included few “cases”). Nonetheless, the replicated findings that early behavior problems show genetic influence challenges the notion that early forms of problem behavior are entirely determined by family environmental factors. These reports provide a good example of how research that simply shows genetic influence can challenge prevailing views of the nature of psychopathology.

Nor is it the case that genetic influences disappear in older age. Perhaps the best example is provided by research on depression. McGue and Christensen's (1997) study of Danish twins 75 years or older indicated strong genetic influence on both affective and somatic symptoms. These findings are noteworthy because they challenge the notion that somatic symptoms of depression index physical impairments in old age rather than depression *per se*. These findings also contradict the hypothesis that—for a variety of reasons, including the loss of friends and loved ones from death, increasing social isolation, and difficulty adjusting to physical limitations—depression in older age is more psychosocially determined.

#### 1.04.4.2 Continua and Categories in Models of Psychopathology

Although diagnostic or categorical approaches have dominated phenotypic research on psychopathology, there is increasing interest in comparing dimensional and categorical models of disorder. Continuous phenotypic dimensions or syndromes have been demonstrated for a wide variety of medical disorders in addition to psychiatric forms of disorder, including personality disorders (Livesley, Jang, Jackson, & Vernon, 1993; Nigg & Goldsmith, 1994) and depression (Kendler, Neale, Kessler, Heath, & Eaves, 1992a).

The debate about dimensions vs. diagnoses has attracted the attention of genetic investigators in recent years because at the core of this debate lies a fundamental question of etiology: do the same genetic and environmental factors that operate at the extremes also affect the entire continuum in the population? Given the novel findings that behavioral genetics research is contributing to this debate and the important clinical implications derived from these results, we provide a detailed overview of the conceptual and methodological issues in this area.

##### 1.04.4.2.1 Distinguishing between the underlying assumptions and quantitative implications of categorical and dimensional models

The dimensional method assesses quantitative variation throughout a continuous distribution of symptoms. By definition, individual differences one standard deviation below the mean are just as informative as 1, 2, or 3 standard deviations above the mean. The diagnostic approach, on the other hand, emphasizes average differences between an "extreme" diagnosed group and a nondisordered group.

The two approaches require different statistical methods. A quantitative genetic analysis of individual differences in dimensions of psychopathology, which is by far the more straightforward of the two, is based on correlations. In the example of a twin design, one would hypothesize that identical twins would be more similar than fraternal twins. Alternatively, the diagnostic approach is based on twin siblings' concordance for disorder. The genetic effect size or heritability is not estimated from concordances alone, but also takes into consideration base rates in the population. The concordance is translated into *liability* correlations that assume a continuous distribution of risk. This procedure assumes, paradoxically, a continuum of liability despite the categorical measurement. This liability-threshold model assumes that the genetic and environmental contributions to the disorder are continuously distributed, but that disorder only occurs when a certain threshold of liability is exceeded.

An additional statistical approach, referred to as DF extremes analysis (from DeFries & Fulker, 1985), integrates quantitative genetic research on dimensions and disorders. This approach requires continuous measurement of behaviors that are relevant to the disorder of interest among the cotwins of selected, or diagnosed, probands. Familial resemblance is estimated as the extent to which the mean cotwins' score on this quantitative measure regresses back to the population mean. If the cotwin mean regresses all the way back to the population mean, the *group* correlation is 0; if the cotwin mean is the same as the proband mean, the group correlation is 1. Group correlations for identical and fraternal twins can then be compared to estimate *group heritability*, or the degree to which genetic influences are associated with being in the extreme group.

There is a fundamental difference between the DF extremes method and the liability-threshold method already described. In contrast to the liability-threshold model, group heritability is not based on the assumption that the disorder is manifested only after a certain threshold is reached. On the contrary, disorder-relevant symptoms are assumed to increase continuously from the normal to the abnormal "levels" of the disorder. Group heritability assesses the contribution of genetic and common environmental factors to the average quantitative difference between the selected probands and the population.

Although the liability-threshold and DF extremes models are based on different underlying assumptions, they do not necessarily yield different empirical results. Indeed, if the

assumption of the liability-threshold model is correct (i.e., there is an underlying continuum of liability for the disorder), and if the quantitative dimension measured in the DF extremes model assesses the actual continuum underlying the disorder, then the heritability of liability in the liability-threshold model will be very similar to the group heritability in the DF model.

Determining whether genetic influences are more salient at the extreme is complex. For instance, most approaches require extremely large samples (of mild and severe impairment) in order that there is sufficient power to detect even large differences. Additionally, these approaches determine only if the magnitude of genetic influence varies across different levels of impairment; it is not possible to determine from the quantitative methods whether *different* genes are involved in the normal and more extreme levels of impairment; molecular genetic studies are needed for this.

To date, there is only limited evidence that directly addresses the question of whether genetic influence is different at the extremes; most reports do not find differences. For example, using DF analyses, Deater-Deckard, Reiss, Hetherington, and Plomin (1996) found that genetic influence at the extremes was similar to genetic influence on normal variation in parent- or adolescent-reported behavior problems. Gjone et al. (Gjone, Stevenson, & Sundet, 1996; Gjone, Stevenson, Sundet, & Eilertsen, 1996) reported some evidence for greater heritability on more extreme levels of behavioral problems, but this effect depended on how the scale scores were analyzed. Other studies have compared heritability across different definitions of disorder. Kendler et al. (1992a) reported similar heritabilities across different definitions of depression in women, and Slutske et al. (1997) reported identical genetic and environmental influences using broad and more severe definitions of conduct disorder in their study based on retrospective reports from the Australian twin registry. In general, then, the genetic evidence available so far indicates that there is a continuity of genetic influence between the normal and the abnormal (Plomin, Nitz, & Rowe, 1990).

#### **1.04.4.2.2 Are shared environmental influences more salient at the extremes?**

A recurring theme throughout this chapter is that genetic studies provide unique clues to understanding the mechanisms through which environmental risks operate. This is also the case in the context of the debates on whether genetic influences affect the continuum of psychopathology. Using the same approaches

defined above (e.g., DF analysis), it is possible to assess the extent to which shared environmental influences are greater in extreme, diagnosed groups compared with nondiagnosed, less impaired groups. As in the case of research on genetic influences at the extremes, there are few studies that examine whether environmental effects differ at the extremes compared with the normal range, and available data suggest that the differences are minimal (Deater-Deckard et al., 1996; Eaves et al., 1993).

#### **1.04.4.2.3 Continuum, boundaries, and spectra**

A further nosological question related to the debate of categorical and continuous measurements concerns the boundaries around a disorder (this also relates closely to the topic of heterogeneity, which is reviewed next). Behavioral genetics research findings indicate that even with major mental disorders, the phenotype extends more broadly than the traditional diagnostic boundaries. For example, it is clear that first-degree relatives of schizophrenic individuals are not only more likely to exhibit schizophrenia but also schizophrenic-related symptoms such as neurological impairments and schizoid symptoms (Kendler, Gruenberg, & Kinney, 1994). A parallel pattern is found among first-degree relatives of autistic individuals, who exhibit elevated levels of pervasive developmental disorder (Bailey, Phillips, & Rutter, 1996; Bolton et al., 1994; Rutter, Bailey, Simonoff, & Pickles, in press).

An additional example, which complicates matters even further, is that the genetically influenced liability to psychopathology may not concern the disorder *per se*, but associated personality features—as in the case of neuroticism and depression (Kendler, Neale, Kessler, Heath, & Eaves, 1993). In this instance, it is not clear whether associated personality features constitute: a core, less acute condition of the disorder; a distinct feature that increases the risk for the disorder; or a consequence of experiencing a disorder. Genetic research findings may play an important role in nosology, but it is equally true that confusion regarding the clinical phenotypes may lead to misinterpretations of behavioral genetics findings.

#### **1.04.4.3 Comorbidity of Psychopathologies**

One of the most robust findings in psychiatric research is that disorders covary at rates far higher than would be expected by chance. This is illustrated by numerous epidemiological reports, such as the National Comorbidity Study (NCS) (Kessler et al., 1994). It is generally

found that a minority of individuals account for the majority of disorders. The degree of overlap between and among dimensions of psychopathology is found regardless of whether diagnostic or dimensional indicators are examined. Several explanations have been proposed to explain remarkably elevated rates of cooccurring disorders or dimensions (Caron & Rutter, 1991). Higher than expected rates of comorbidity or symptom cooccurrence may be explained by: diagnostic overlap of individual symptoms (i.e., a particular symptom is included under more than one diagnosis); the possibility that one disorder may be a risk factor for developing a second disorder; different disorders may be overlapping because of common psychosocial risks; diagnostic schemas may favor multiple diagnoses (*DSM-IV*) over identifying a central diagnosis (*ICD-10*). Other explanations, however, are also possible.

The degree to which two (or more) disorders or dimensions may be correlated because of a shared genetic liability has been examined only recently. Virtually all available evidence indicates that genetic influences are not diagnosis- or dimension-specific. Rather, the nearly universal phenotypic correlations between different dimensions of psychopathology can be explained, in part, by shared genetic factors, called genetic correlations.

A genetic correlation underlying two (or more) syndromes is possible if each syndrome is genetically influenced and the syndromes are correlated phenotypically. The quantitative basis for analyzing genetic correlations is described in Neale and Cardon (1992). In simple terms, genetic influence on the correlation between two disorders is based on the cross-twin cross-correlation. In the case of antisocial and depressive symptoms, genetic influence on the correlation between these two dimensions is suggested if the correlation between twin A's antisocial behavior and twin B's depressive symptoms is greater in MZ than in DZ pairs (or other genetically informative comparison).

In a series of studies on adult female twins in the Virginia twin registry, Kendler and colleagues reported a significant genetic correlation between depression and Generalized Anxiety Disorder (Kendler, Neale, Kessler, Heath, & Eaves, 1992d), alcoholism (Kendler, Heath, Neale, Kessler, & Eaves, 1993), and smoking (Kendler, Neale, MacLean, et al., 1993). In the case of major depression and generalized anxiety disorder, Kendler and colleagues reported a genetic correlation of 1.0 between major depression and generalized anxiety disorder. In other words, the same genes make one vulnerable to depressive and anxious symptoms. The finding of a genetic correlation of 1 is,

however, an exception to the general rule that the genetic correlation between correlated dimensions is usually more moderate.

Research findings from psychopathology in childhood and adolescence lead to similar conclusions. For example, the robust phenotypic correlation between depressive symptoms and antisocial behavior in children and adolescents is well known, and there is increasing evidence that both dimensions are under some genetic influences (for depression, see Silberg et al., 1994; for antisocial behavior, see Gottesman & Goldsmith, 1994). A genetic correlation between these dimensions was recently found in a large national study of 720 families using a multi-method measurement design (O'Connor, McGuire et al., in press). Approximately 50% of the phenotypic correlation between these dimensions could be explained by shared genetic influences. A follow-up study conducted approximately two years later found roughly the same results (O'Connor, Neiderhiser, Reiss, Hetherington, & Plomin, 1998). It is important to note that the study also found genetic influences unique to each dimension. Interestingly, the same pattern of genetic and environmental influences was found for boys and girls despite marked mean differences in these dimensions of maladjustment. Data from a large study of juvenile twins in Virginia examined the genetic correlation underlying hyperactivity and oppositional/conduct problems (Silberg et al., 1996). Results of that study indicated that the correlation between these two dimensions was explained largely from the genetic influences common to both syndromes, although the pattern varied slightly by age and gender. Family history data provide an additional source of support for genetic factors underlying different disorders. In this approach, evidence for genetic influences underlying cooccurring disorders is suggested if first-degree relatives of depressed probands are at elevated risk for depression, and for a second disorder, e.g., alcoholism, compared with first-degree relatives of nondepressed probands. Such data are taken as evidence for cosegregation of depression and alcoholism. Cosegregation among disorders is relatively common (see, e.g., duFort, Kovess, & Boivin, 1994; Feng & Baker, 1994; Mednick, 1978) and has also been demonstrated in research on child probands (Puig-Antich et al., 1989). It is important to note, however, that some studies that suggest cosegregation of disorders do not always control for the fact that probands may exhibit multiple disorders.

However, just as there is mounting evidence of nonspecificity of genetic risks for psychopathology, there is also evidence, in some cases,

for specificity. Most of the available evidence suggesting specificity of genetic risks derives from family history studies. Among the better known examples is the finding that first-degree relatives of schizophrenic individuals are at greater risk for schizophrenia and schizophrenia-related conditions but not at greater risk for bipolar disorder. This supports the hypothesis, which is also suggested from available molecular genetic studies (see below), that schizophrenia and bipolar disorder are not influenced by the same genes (see, e.g., Kendler et al., 1994; Tsuang & Faraone, 1990). Similar methods also provide convincing evidence that the genetic influences underlying liability to bipolar disorder are not linked to those associated with unipolar depression (see Tsuang & Faraone, 1990; Weissman et al., 1984), suggesting that the same genetic influences are not shared by all affective disorders.

Unfortunately, too few studies have systematically examined cooccurrence among multiple (i.e., more than two) disorders, so it is not yet known whether the same genetic influences may help explain that, among affected individuals, the average number of diagnoses per individual is greater than two (Kessler et al., 1994).

#### **1.04.4.3.1 Comorbidity of psychopathologies: environmental evidence**

There is little specificity in the relationship between environmental risks such as interpersonal stress or absence of social support and particular manifestations of psychopathology (Wrate, Rothery, McCabe, Aspin, & Bryce, 1995). Behavioral genetic research has contributed to this line of research by also demonstrating that, controlling for genetic factors, there is evidence that two (or more) disorders or dimensions may cooccur because of common shared and nonshared environmental risks. In the study examining antisocial behavior and depression noted above (O'Connor, McGuire, et al., 1998), there was a small but significant effect of shared environment that contributed to siblings' similarity in both syndromes. That is, by virtue of sharing the same household, siblings share familial experiences that function to make them similar to each other in their levels of both behavioral dimensions (cf. Downey & Coyne, 1990). Of course, studies that rely on only model-fitting cannot identify which shared environmental factors underlie the correlation between behavioral dimensions, but the key advantage is that they are able to identify environmental influence *independent* of genetics.

Similarly, individual-specific environmental risk factors will contribute to the covariation between disorders in multivariate behavioral

genetics analyses to the extent that siblings who differ in their experience of environmental risks also differ on both dimensions of psychopathology. An example is provided by the study of antisocial behavior and depressive symptoms in adolescence noted above (O'Connor, McGuire, et al., 1998).

#### **1.04.4.4 Genetic Heterogeneity**

The concept of heterogeneity of disorder is a focus of increasing clinical research attention. Heterogeneity is an important topic to study because a clear understanding of the diverse ways in which a disorder may manifest is central to optimally effective treatment and prevention. Notwithstanding the significance of the concept, there has been little progress in how it should be defined. Recent papers on the topic define heterogeneity in diverse ways including presumed etiology, biological correlates, response to treatment, longitudinal course, and association with other disorders. These different definitions likely lead to different conclusions regarding heterogeneity. Moreover, there are few attempts to examine multiple alternative definitions within the same study.

Some of the most important data that focuses on heterogeneity are based on the work of Winokur, Coryell, and colleagues. The "Iowa 500" family study has provided evidence that familial influences are associated with certain subtypes of depressive disorder (see Coryell, 1997 and references therein). Unfortunately, there have been few attempts to replicate these subtypes, and it is not known whether psychopharmacological treatments are differentially effective for different subtypes (but see Sigvardsson, Bohman, & Cloninger, 1996, for the case of alcoholism). A further example of using genetic data to identify subgroups of affected individuals is provided by DiLalla & Gottesman (1989), who hypothesized that whereas adolescence-limited and late-onset delinquency may derive from largely psychosocial factors, antisocial behavior exhibited by individuals who exhibit a stable antisocial trajectory may be genetically mediated (Moffitt, 1993).

Behavioral genetics methods can be used to assess whether different symptom clusters of the same disorder are influenced by the same genes, and whether subgroups of disordered individuals can be identified by degree of genetic risk. In a recent study of depression in Danish twins 75 years or older, McGue & Christensen (1997) tested the hypothesis that two factors of depression in the elderly, somatic and affective symptoms, were influenced by the same genetic

factors. They found that despite being clearly distinguishable at a phenotypic level, the two factors of depression were nonetheless explained by the same genetic factors. In other words, there were no genes that were specific to somatic symptoms or affective symptoms. In this way, these authors provide a model for further research to examine whether the heterogeneous manifestations of a dimension of psychopathology have different genetic and environmental origins.

An opposite example is provided by reports on aggression and delinquency, two aspects of conduct disorder. These two behavioral syndromes are distinct, but nonetheless are highly correlated. However, despite the considerable overlap, two studies reported that aggression is strongly genetically influenced but delinquency is under minimal influence (Edelbrock, Rende, Plomin, & Thompson, 1995; Eley, Lichenstein, & Stevenson, *in press*).

#### ***1.04.4.4.1 Age of onset and other features***

Behavioral genetics methods have also been used to examine whether certain features of a disorder are under genetic control. Family history data have suggested that younger age of onset is a proxy for stronger genetic influence (Todd, Neuman, Geller, Fox, & Hickok, 1993; Weissman et al., 1984). In general, probands with an earlier age of onset tend to have more first-degree relatives with a similar disorder than those with a late onset. However, although many studies have found that age of onset is correlated with genetic risk, not all studies replicate this pattern, or they report that the pattern holds for adolescent but not childhood onset probands (*cf.* Harrington et al., 1993).

More convincing support implicating genetic influences on the age of onset is reported by Kendler, Neale, Kessler, Heath and Eaves (1992). In their large sample of adult female twins, they were able to identify a large number of identical and fraternal twins concordant for major depression. When they examined how these concordant, affected cotwins manifested depression, they found that depressed identical twins were no more similar in the likelihood of exhibiting particular symptoms of depression. However, concordant depressed identical twins were more similar than concordant fraternal twins in the age of onset of disorder. A conclusion drawn from these data is that genetic influences underlie the likelihood of experiencing a disorder and perhaps some features such as age of onset, but genetic influences appear not to directly influence the particular pattern of symptoms the disorder takes.

#### **1.04.4.5 Genetic Influence on Change and Stability of Psychopathology**

A critical developmental question that awaits firm answers is the extent to which genetic influences mediate the marked stability of disorder from childhood through adulthood. A related question is whether genetic influences on psychopathology change with age, pubertal status, or other markers of development. In other words, to what extent are genetic influences on psychopathology in adolescence and adulthood different from those detected in childhood. It is not that an individuals' genes change, but that genes can have different effects at different points in development.

Consider, for example, the finding that bipolar and schizophrenic disorders are rare before puberty, and may first emerge during adulthood and even into older age. These disorders are known to be under genetic influence, so it seems likely that the genetic mechanisms are not apparent in early childhood (although there is little research addressing this issue) but come "on line" only later in development. Further examples of genetic influences explaining discontinuity or marked change in development include such disorders such as Huntington's disease and life-course changes such as pubertal development. This is an important lesson for molecular genetic studies (see below) that seek to identify the presence of a single or a set of genes associated with the presence of a disorder. The developmental genetic perspective suggests that individuals may have the genetic risk but may not exhibit signs of the disorder until late in development.

The analytic models used to assess genetic influences underlying stability and change in psychopathology are similar to those used to examine genetic correlations underlying comorbid disorders (Plomin, 1986). However, although the analytic models are straightforward, there are few examples of longitudinal behavioral genetics analyses of psychopathology (McGue, Bacon, & Lykken, 1993).

Two studies found that both genetic and environmental factors contribute to stability in adolescent adjustment. For example, Van den Oord and Rowe (1997) reported that genetic and shared environmental influences contribute to the stability of antisocial, anxious/depressed, and hyperactive symptoms. O'Connor, Neiderhiser et al. (1998) found that genetic influences explained approximately half of the stability in antisocial and depressive symptoms, shared environmental factors explained stability of antisocial but not depressive symptoms, and nonshared influences also contributed to stability for both syndromes.

Genetic influences have also been reported in research on adult psychopathology. Data from the Virginia sample studied by Kendler and colleagues suggests that, for example, stability of depressive symptoms is mediated genetically (Kendler, Neale, et al., 1993). Data from a twin registry composed of individuals who served in the military during the Vietnam War indicated that phenotypic stability between juvenile and adult forms of antisocial behavior was due in roughly equal measure to genetic, shared environment, and nonshared environment (Lyons et al., 1995). Although the data are derived from retrospective reports, this is one of the few studies that has examined the well-known phenotypic connection between conduct problems in adolescence and antisocial behavior in adulthood from a genetic perspective.

Family history data are also consistent with the hypothesis that genetic influences underlie stability in psychopathology. As noted, genetic mediation of stability is suggested by parent-offspring concordance rather than age-to-age genetic correlations. In one sense the family history design is a strict test of genetic stability because it requires that the genes associated with the disorder in childhood or adolescence are the same as those associated with its manifestation in adulthood, and that the phenotypic expression of the disorder is similar. Examples of analyses of this type for depression are given by Harrington et al. (1993).

#### 1.04.4.6 Summary

In this section we examined five distinct ways in which behavioral genetics research has informed and challenged our notion of the clinical phenotype. In particular, we provided evidence that genetic etiology for psychopathology is apparent, and in some cases pervasive, throughout the life-span. Second, consistent with the phenotypic research findings, behavioral genetics studies support a continuous model of psychopathology. There is remarkably little evidence that individuals exhibiting clinical levels of disorder are qualitatively distinct from individuals expressing milder forms, in terms of genetic and environmental risks. Third, behavioral genetics findings have helped to explain the pervasive cooccurrence of disorders in childhood through adulthood. The nosological implications of these findings require further attention. Fourth, the concepts of heterogeneity and disorder spectrum have also been shaped by behavioral genetics findings. Finally, although the evidence is limited, there appears to be growing support for the impact of genetic influences on the stability of psychopathology.

These findings raise important questions regarding the turning “on and off” of genes and gene mechanisms in development.

In the next section we expand these lessons by examining what behavioral genetics research suggests about the processes associated with the development of psychopathology.

### 1.04.5 MODELS OF GENETIC AND ENVIRONMENTAL RISK MECHANISMS

The range of behaviors apparently under genetic control is remarkable. It is now well accepted that intelligence, personality and psychopathology are influenced by genetic factors, but recent studies also suggest genetic influence on divorce (McGue & Lykken, 1992), life events (Plomin, Lichtenstein, Pedersen, McClearn, & Nesselroade, 1990), sexual orientation in males (Bailey & Pillard, 1991) and females (Bailey, Pillard, Neale, & Agyei, 1993), social support (Bergeman, Plomin, Pederson, McClearn, & Nesselroade, 1990; Kendler et al., 1995), peer affiliation (Manke, McGuire, Reiss, Hetherington, & Plomin, 1995), personal devotion to religion (Kendler, Gardner & Prescott, 1997), smoking (Kendler, Neale, MacLean, et al., 1993), television viewing (Plomin et al., 1990), behavior in problem-solving interactions (O'Connor, Hetherington, Reiss, & Plomin, 1995), work behavior and a remarkably wide range of attitudes and interests (Plomin et al., 1997), among others. Indeed, some authors have commented that the null hypothesis of no genetic influence is no longer justifiable (see commentaries on Plomin & Bergeman, 1991).

These findings lead to the inevitable conclusion, already stated in the chapter, that the main thrust of research in behavioral genetics is no longer whether a behavior or trait is genetically influenced. Instead, the focus of behavioral genetics research in psychopathology is how to conceptualize the mechanisms through which genetic factors impart their influence, and how genetic and psychosocial risks jointly operate to lead to maladjustment.

#### 1.04.5.1 Conceptualizing Genetic Influence

Many of the models cited for understanding how genes and environment operate together have little relevance to psychopathology. An example of this is PKU, a disorder in which affected individuals are unable to synthesize phenylalanine. This condition, which leads to mental impairment, can be effectively treated if dietary restrictions are followed. Although PKU is a good example of how genetic causes

can have environmental cures, it is an inadequate model for understanding genes and environment as applied to psychopathology. In this example, neither the genetic model (single gene) nor the environmental model (environmental risk exposure leads directly and unfailingly to the manifestation of the disorder) parallel what is known about psychopathology.

#### 1.04.5.2 Conceptualizing the Environment

Much of the controversy regarding family influence from behavioral genetics research concerns the finding that shared environmental parameters are usually modest or even zero. Does this finding suggest that there is no effect of the family environment? Absolutely not. Instead, what this suggests is that simply growing up in the same home appears to matter little in shaping children's similarity in behavioral traits. It is worth restating here that estimates of shared familial environment are based on *effects* rather than *measures*. Accordingly, shared environmental influences arise only to the extent to which environments have functionally equivalent effects on siblings.

As noted previously, one of the most important lessons for environmental research has been the importance of nonshared environment. In order to identify environmental influences that play salient roles in development and psychopathology it is critical to assess risks specific to the individual because risks shared by family members (e.g., socioeconomic disadvantage, maternal depression) may nonetheless have nonshared effects.

#### 1.04.5.3 Genotype–Environment (G–E) Correlations

A major advance in integrating genetics and the environment in research has been the discovery of genotype–environment correlations. Some of the lessons learned from these efforts are described below.

##### 1.04.5.3.1 Passive genotype–environment correlation

Genotype–environment correlations come in a variety of forms (Plomin et al., 1977; Scarr & McCartney, 1983). The most obvious example is passive genotype–environment correlation. Passive correlations arise because related parents and children share both genes and environments. That is, children share half of their parents' genes and are exposed to environments provided by them. The correlation is "passive"

in that children themselves play no direct role in creating the overlap of genetic and environmental factors. The only way of directly estimating the influence of passive genotype–environment correlations is to compare parents and their offspring in biological and adoptive families.

Passive genotype–environment correlations make a number of studies of children of psychiatric parents difficult to interpret because these children are not only more likely to inherit genetic risks but also to be exposed to dysfunctional environments that psychiatric parents are disproportionately likely to provide (Rutter & Quinton, 1984). Studying the offspring of parents diagnosed with depression, alcoholism, or other disorders was a mainstay of high-risk research, but the presence of passive genotype–environment correlations makes interpretations of the mechanisms leading to behavior problems difficult to determine.

##### 1.04.5.3.2 Evocative and active genotype–environment correlations

The principle that individuals are active participants in and create and select their own environments is widely acknowledged. Individual differences in relationship styles, hyperactivity, conduct disorder, and sociability are sustained through the dynamic interchange between personal characteristics and social experiences (Caspi & Moffitt, 1995; Rutter, Champion, Quinton, Maughan, & Pickles, 1995). There are many illustrations of this principle from phenotypic research, including the finding that aggressive children evoke negative responses from strangers (Anderson, Lytton, & Romney, 1986) and aggressive individuals are more likely to select aggressive partners (Krueger et al., in press; see Caspi & Moffitt, 1995).

Even more striking is the finding that the way in which individuals evoke, select, and react to their environments is not independent of genetic influence. In this way, there is a correlation between the genetic predispositions of individuals and the environments they experience (Plomin, 1994). Comparisons of identical and nonidentical twins (and biological and adopted siblings) consistently demonstrate that the former are more similar than the latter in, for example, their selection of peer groups (Manke et al., 1995) and manner of interacting with others, notably parents (O'Connor et al., 1995). These findings illustrate how there may be genetic influences on environmental measures (Plomin & Bergeman, 1991).

Two recent adoption studies provide parallel examples of genotype–environment evocative



correlation as regards antisocial behavior. In a high-risk adoption study, Ge et al. (1996) reported that adopted adolescents at genetic risk for antisocial behavior (by virtue of having a biological parent with diagnosed disorder) were more likely to evoke aggressive, hostile parenting and less likely to evoke supportive parenting from their adoptive mothers and fathers compared with adoptees not at genetic risk. The study also found that the more negative parenting of at-risk adoptees was mediated by their own disruptive, aggressive behavior toward the parent.

A similar study based on the Colorado Adoption Project (CAP) also found that individuals with a biological parent with a history of antisocial behavior were more likely to evoke negative, coercive parenting from their adoptive parent relative to adoptees not at genetic risk for antisocial behavior (O'Connor, Deater-Deckard, Fulker, Rutter, & Plomin, *in press*). The CAP study found that evocative genotype–environment correlations were modest in magnitude from late childhood to early adolescence. As in the Ge et al. (1996) study, O'Connor, Deater-Deckard et al. (*in press*) found that the effect was mediated by children's aggressive behavior, but the mediation effect was far less dramatic in the latter study. Both studies provide compelling evidence that genetic influences are not limited to trait-like behaviors, but are equally relevant to socialization processes and family relations.

#### ***1.04.5.3.3 Hypothesized age changes in genotype–environment correlations***

Scarr and McCartney (1983) advanced the provocative hypothesis that genotype–environment correlations increase with age and change in type. Specifically, they suggested that passive genotype–environment correlations would predominate in infancy and early childhood and that, with increasing age and ability to select one's own environments, there would be a gradual shift and evocative/active genotype–environment correlations would predominate.

There is a natural developmental appeal in this hypothesis, but few data are available to test the idea. What data are available are not consistent with the proposal. For example, rather than wane from infancy to adolescence, a recent adoption study highlighted that passive genotype–environment correlations remain strong in adolescence (McGue, Sharma, & Benson, 1996). Additionally, Lytton (1977), among others, has found that evocative genotype–environment correlations are robust in early childhood. Moreover, one of the few studies of evocative genotype–environment

correlations found no sign of developmental change from late childhood through early adolescence (O'Connor, Deater-Deckard et al., *in press*).

Of course, the hypothesis that there are developmental changes in the type and magnitude of genotype–environment correlations is different from the more straightforward hypothesis that there are age-based changes in the effects of genetic influences and, alternatively, shared and environmental influences. As noted, there is now considerable evidence that genetic influences seem to increase with age and that shared environmental influences decrease steadily from early childhood through adulthood (McCartney, Harris & Bernier, 1990).

One implication of genotype–environment correlations is that the psychosocial processes associated with the development of psychopathology may not be entirely environmentally mediated. Consider the process of coercive parenting and child disruptive behavior outlined by Patterson (1982). It is possible to view both sides of this transactional process as genetically influenced. Children's aggressive behavior is partly genetically influenced; aggressive antisocial parenting may itself be associated with genes affecting the parent's antisocial tendencies or may be evoked by genetically influenced antisocial behavior in the children.

Consistent with the processes suggested above, a handful of studies demonstrate that relations between parenting and child adjustment—the hallmark of developmental research on psychopathology—are mediated by genetic influences. For example, in a twin-sibling study, Pike et al. (1996) attributed the association between mothers' and fathers' negative parenting and adolescent symptoms of depression and antisocial behavior largely to genetic factors. That is, the correlation between, for example, coercive, hostile parental behavior and children's externalizing symptoms arises because genetically influenced aggressive and externalizing behaviors evoke negative parenting.

#### **1.04.5.4 Genotype–Environment Interactions**

It is possible to identify several types of genotype–environment interactions. The most common way in which genotype–environment interactions are conceptualized is that there are genetic influences on the sensitivity to environmental risks (Kendler, 1995). Stated differently, individuals at genetic risk are more likely than individuals not at genetic risk to react maladaptively to psychosocial stresses.

This hypothesis is a simple extension of a basic tenet in developmental psychopathology, namely, that there are individual differences in vulnerability to psychopathology. The extension is that a source of these individual differences in vulnerability is genetics.

Despite the general acknowledgement that there are likely to be genotype–environment interactions in development, there are remarkably few examples. Wahlsten (1990; see also commentaries) suggests that the reason may be partly methodological. He rightly points out that many human studies lack the power to detect interaction effects after the main effects are removed. This is particularly relevant to studies that compare correlations, such as between MZ and DZ twins, and particularly if comparisons are made between patterns under high and low stress. Wahlsten's assumption is that genotype–environment interactions are common but go undetected. One reason for believing this argument is that genotype–environment interactions are numerous in animal research in which it is relatively easy to control and constrain both genetic factors (through breeding or using identified strains) and environmental conditions (through experimental manipulation that would not be ethical in human research).

There are several additional reasons why detecting genotype–environment interactions has been difficult. First, there are few examples of behavioral genetics analyses across a wide range of stress conditions. Most authors assume that genotype–environment interactions would be most easily detected in the context of extreme adversity because genetic effects are thought to be stronger in high-risk environments than in normal and low-risk environments—although one could make the opposite argument that high-risk environments might overwhelm individual differences in vulnerability (e.g. Scarr, 1992).

These challenges notwithstanding, there are a number of illustrations of genotype–environment interactions for a range of psychopathological conditions. The Swedish adoption study reported by Bohman (1996) provides one of the best examples. Bohman (1996) reported that children with an antisocial biological parent who were also raised in their biological family (i.e., high genetic risk and environmental risk) were considerably more likely to be antisocial than children of antisocial biological parents who were raised by adoptive parents (i.e., high genetic risk but low-risk environment), children exposed to environmental risk in the absence of genetic risk, and children from the normal population. The implication of the pattern of findings is that children at biological risk are

relatively more vulnerable to environmental adversity than children not at genetic risk.

In a separate adoption study of antisocial behavior, Cadoret, Yates, Troughton, Woodworth, & Stewart (1995) reported an association between adverse adoptive home environment and adoptee aggressive, antisocial outcome only in the presence of genetic risk (see also Cloninger, Sigvardsson, Bohman, & von Knorring, 1982). In their study of depression in adult female twins, Kendler et al. (1995) found that the difference in the likelihood of experiencing depressive symptoms between those at high and low genetic risk was substantially magnified following severe life events. Cadoret et al. (1996) also reported genotype–environment interaction for depression in females (but not males), but in that study genetic diathesis was defined not by depression but by alcoholism. Nonetheless, the interaction is the same: environmental adversity is associated with increased likelihood of depression only in the presence of genetic risk.

Finally, Wahlberg et al. (1997) reported a genotype–environment interaction for schizophrenia-related symptoms. Although neither genetic risk (having a biological parent with schizophrenia) nor environmental risk (defined as communicative deviance in the adoptive family) alone resulted in thought disorder among adoptees, the joint effect of both variables was associated with a substantial increase in thought disorder (see also Tienari et al., 1994; see True et al., 1994, for an example as applied to post-traumatic stress disorder).

A slightly different model of genotype–environment interaction was reported by Slutske et al. (1997). These authors found that genetic influence on conduct disorder based on retrospective reports in adulthood existed only if the twins had the same friends; this was true only for boys. In this instance, it is not genetic control over sensitivity to environmental stress, but rather similarity in environmental circumstances that accentuated genetic influences.

#### **1.04.5.5 Summary**

Several developmental models need to be considered in elucidating genetic influences in the development of psychopathology (e.g., Bronfenbrenner & Ceci, 1993; Gottesman & Goldsmith, 1994; Kendler & Eaves, 1986; Plomin, 1994; Reiss, 1995; Rutter et al., 1997). Among the possibilities are: (i) genetic and environmental effects operating in an additive manner; (ii) genetically influenced characteristics altering the exposure to environmental risks, or genotype–environment correlations; (iii)

genetic influences mediating the association between putative risk and adjustment; and (iv) genetic influences controlling the sensitivity to environmental risks, or genotype–environment interactions. It is important to note that additive, correlated, mediational, and interactive genotype–environment models are not mutually exclusive.

Further improvements and refinements of our understanding of these mechanisms will depend on future research in which genetic risks, like environmental risks, are specified. This, of course, requires the contributions of molecular genetics research.

#### **1.04.6 MOLECULAR GENETIC RESEARCH ON PERSONALITY AND PSYCHOPATHOLOGY**

Behavioral genetics refers to the genetic analysis of behavior. Quantitative genetic analyses such as twin and adoption studies are best known, but molecular genetic analyses that attempt to identify specific genes and their functions are becoming increasingly important. Although complex traits like behavior are likely to be influenced by multiple genes, identifying some of these genes will provide the opportunity for more focused analyses of questions raised by quantitative genetics using direct assessment of measured genotypes (Plomin & Rutter, in press).

##### **1.04.6.1 Methods and Questions in Molecular Genetics Research on Psychopathology**

A detailed discussion of the relative merits of association and linkage studies and the statistical methods devised to detect quantitative trait loci, among other statistical procedures, is beyond the scope of this chapter (see Baron; 1997; Plomin et al., 1997). Suffice it to say here that just as nonbehavioral genetics investigators are increasingly familiar with the quantitative models and assumptions in that field, the methods and assumptions of molecular genetics will also become increasingly familiar.

Although we avoid a detailed methodological overview, several points deserve attention. First, it is important to recall that molecular genetics studies of psychopathology should not be equated with simplistic reductionistic models. There are a number of reasons why this is the case. The most obvious is that most authors would agree that specific genes will account for a very small percentage of the total variance in psychopathology. Moreover, simply identifying the genes provides no answers to the processes through which genetic and environmental

influences lead to psychopathology. Accordingly, results from molecular genetics studies will likely heighten the need for intensive research on environmental processes. There are, to date, few attempts to assess both genetic and psychosocial sources of risk in these studies, and this remains an important direction for future research.

It is important to distinguish research that attempts to identify genetic loci associated with disorder from research that uses previously identified loci (Plomin & Rutter, in press). Although it is difficult and expensive to identify genes associated with behavior, it is relatively easy and inexpensive to use previously identified genes as measured genetic risk factors in any type of research on description, prediction, prevention, and intervention.

##### **1.04.6.2 Contributions of Molecular Genetics Research to Models of Psychopathology**

Despite a decade of attempts to identify genes for schizophrenia and bipolar disorder (e.g., Peltonen, 1995), success in identifying genes seems to be coming more quickly for personality dimensions (Cloninger, 1997; Ebstein, Nemanov, Klotz, Gritsenko, & Belmaker, 1997) and more common disorders such as reading disability (Cardon et al., 1994; Grigorenko et al., 1996; Schulte-Körne et al., 1997), attention deficit hyperactivity disorder (ADHD) (LaHoste et al., 1996; Palmer et al., 1997; Sunohara et al., 1997) and opioid dependence (Ebstein & Belmaker, 1997). For schizophrenia, there have been several reports suggesting genes on chromosomes 3, 5, 6, 8, and 22 (see Schwab et al., 1997).

A further example of how molecular genetics has contributed to clinical psychology and psychiatry is Alzheimer's disease. A familial component of Alzheimer's disease, at least in a minority of cases, had been suspected for several years, and genetic influence was implicated in twin studies (Breitner et al., 1993). Molecular genetic studies indicated that late-onset Alzheimer's disease (onset after age 65) that shows a familial pattern is linked to a gene on chromosome 19 (apolipoprotein E4 or Apo-E4) (Corder et al., 1993). Apo-E4 operates as a susceptibility gene, quadrupling risk for Alzheimer's disease, but it is neither necessary nor sufficient for the development of the disorder. In addition, a much rarer form of Alzheimer's disease, early onset, can be caused by a gene on chromosome 14 (Sherrington et al., 1995); other even rarer single genes for early-onset Alzheimer's disease have been found.

### 1.04.6.3 Summary and Future Directions

The end goal of the “gene chase” in behavioral science (McCleary et al., 1991) is not simply to detect an association between a specific locus (or loci) and behavior. Neither a significant association between an allele and disorder nor a significant heritability estimate indicate how genetic factors operate in development. Once genes are identified, we need to explore the biological and social pathways through which genetic vulnerabilities, expressed through specified biochemical markers, respond to social and psychological events to produce psychopathology. The identification of specific genetic vulnerability markers may lead to advances such as the identification of which individuals may be more sensitive to certain psychosocial stresses and a better understanding of how genetic vulnerabilities covary with psychosocial adversity, as well as progress in defining genetic heterogeneity and genetic correlation between disorders.

### 1.04.7 CLINICAL PSYCHOLOGY IMPLICATIONS OF BEHAVIORAL GENETICS RESEARCH FINDINGS

There have already been major clinical implications for medical treatment, but what about clinical psychology? In many cases the clinical implications of behavioral genetics findings for psychology are not yet well understood. This is partly because the field is still relatively young as regards psychopathology and because the mechanisms of genetic influences have not yet been clearly delineated.

A further reason why lessons from behavioral genetics studies have only slowly begun to shape clinical practice in psychology is that there continue to be misplaced biases against behavioral genetics findings. This appears to be so despite excellent papers dispelling various myths (Rutter & Plomin, 1997). As the section on genotype–environment models emphasized, there is no “zero-sum” relationship between the role of genetics and the role of environmental influences (which include clinical interventions). Indeed, it is precisely because genetic influences do not operate in isolation from psychosocial stresses that the finding of genetic influences should encourage the development of effective prevention and intervention models.

#### 1.04.7.1 Clarifying Genetic Risks in Assessment

It is already common practice to collect information on family psychiatric history. Whether there is a history of depression or substance use in the family may, most would

agree, contribute to understanding the difficulties experienced by the patient and the likelihood of an individual developing a similar disorder. Whether such information would alter how a patient’s diagnosis is conceptualized is a slightly more complicated matter. Although behavioral genetics research findings have made important contributions to diagnostic debates, the translation of these findings into clinical practice as applied to an individual is not direct and often fraught with difficulty.

Consequently, it may be that knowledge of genetic risks (e.g., based on family history) contributes most directly not to the conceptualizing diagnosis, but rather to conceptualizing the sources of risk. Research on ADHD provides a good example. ADHD can be managed by psychostimulant medication and parenting practices (Barkley, 1990). The finding that first-degree relatives, notably parents, of ADHD probands are also at elevated risk of ADHD (Biederman et al., 1992) suggests that it would make good clinical sense to assess symptoms of poor concentration and inattention in the parents, even if they are not the identified patient. Assessing ADHD symptoms in the parents may lead to relief in the parent(s) and facilitate parent-training approaches and other forms of parent–child and family treatment.

#### 1.04.7.2 Genetic and Environmental Considerations for Treatment and Prevention

Perhaps the most important lesson for treatment is that there is no necessary connection between causes and cures—specifically, between genetic influence and type of intervention. That is, psychopathology that is strongly influenced by genetic factors may nevertheless be affected by intervention. Thus, finding genetic effects in no way contradicts the benefits of, e.g., behavioral interventions to improve the parenting of antisocial children (Patterson, 1982; Webster-Stratton, Hollinsworth, Kolpacoff, 1989). In this context it is also interesting to note the high rate of schizophrenia in people from the Caribbean living in the UK but not in those living in the Caribbean area (Van Os, Castle, Takei, Der, & Murray, 1996). In addition, it is worth remembering that the factors influencing individual differences in psychopathology are not necessarily the same as those underlying differences in the rate of psychopathology between groups or over time. Consider, for example, the marked secular changes in crime and suicide in young people over the last 50 years and the reported increase

in rates of depression and other mental disorders (Rutter & Smith, 1995).

Although somewhat speculative, it is possible to derive treatment and prevention implications from research on genotype–environment correlations and interactions. To take one example, antisocial children often have a “double” risk: inheriting genetic risk from parents and being exposed to coercive interactions and poor monitoring (that may be a result of the parents’ antisocial behavior). Consequently, it is possible to conceptualize parent–child interventions as interfering with an otherwise “naturally occurring” passive and evocative genotype–environment correlation. A complementary explanation is also possible based on genotype–environment interactions, namely, intervention and prevention efforts reduce genetic risk by decreasing environmental risks to which these individuals might be particularly susceptible. The number of studies designed to prevent the development of disorder in children of depressed parents is a second example (e.g., Beardslee, Wright, Salt, & Drezner, 1997).

More clinical research is needed that specifically identifies genetic risk status and elucidates treatment processes. In fact, clinical research is one of the best lines of investigation to document genotype–environment interactions and correlations, basic notions of how genes and environment jointly contribute to the development of psychopathology. Moreover, intervention research that explicitly incorporates genetic risk status and manipulates psychosocial experiences may help to identify subgroups of individuals who respond best to particular treatment approaches. This could be accomplished, for instance, by including genetic risk status (based on presence of disorder in first-degree relatives or perhaps even genetic markers) as a predictor of treatment response.

#### 1.04.7.3 Summary

Necessarily, the clinical implications of available behavioral genetics findings are, at this time, largely speculative. Although it is possible to derive some general lessons regarding treatment and prevention from nonclinical studies, there is an unfortunate absence of research that directly fuses psychological intervention and genetic risk models. What available findings do suggest is that the mechanisms of intervention may be far more complex than initially proposed. Psychological interventions may be effective partly because they alter genetically-based susceptibility to environmental risk or eliminate otherwise pervasive genotype–environment correlations that in-

crease the likelihood of psychopathology. The second point which we wish to emphasize here is that intervention and prevention studies are powerful designs in which to test some of the most critical but yet unanswered questions concerning the mechanisms leading to psychopathology.

#### 1.04.8 GENERAL SUMMARY AND CONCLUSION

The remarkable progress in our understanding of genetic influences on a wide range of medical conditions and behavior has been noted by numerous commentators in the scientific and popular press. Fortunately, previous conceptions of genetic influence as immutable and contrary to psychological interventions and socialization theories are now dispelled. Moreover, contemporary concerns regarding ethical and moral issues for genetic research are based, rather, on informed dialogue (e.g., see Gottesman & Bertelson, 1996). No longer a topic of largely political weight, the so-called nature–nurture debate has spawned a major research paradigm that has yielded some of the most important clues to our understanding of the development, etiology, and conceptualization of psychopathology.

We reiterate three general conceptual advances derived from behavioral genetics research. Perhaps the most important is that the conceptualization of genetic and environmental factors has advanced beyond the question of whether genetic influence is relevant (cf. Rutter et al., 1997). Questions that need to be addressed focus instead on how genetic vulnerabilities lead to maladjustment and how to incorporate genetic vulnerability into the already well-established models of risk and resilience. A second, fundamental conclusion is that although authors differ in their optimism regarding how genetic studies can shed light on environmental mechanisms of risk, all would agree that studying psychosocial risks must cooccur with analyses of biological and genetic factors (Reiss, Plomin, & Hetherington, 1993). This is as true for molecular genetics as it is for twin and adoption designs. Finally, one of the most important directions of behavioral genetics research has been to encourage molecular geneticists to search for specific genes that may help answer questions about how genetic influences create vulnerability to psychopathologies. Clearly, progress in understanding genetic and environmental sources of risk for psychopathology thus appears to be following a path of dialectic integration rather than pendulum-like swings that characterized research just a decade ago.

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# 1.05

## Psychobiology

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## 1.05.1 PSYCHOBIOLOGY AND CLINICAL PSYCHOLOGY

*When I carefully consider the curious habits of dogs I am compelled to conclude that man is the superior animal. When I consider the curious habits of man I confess, my friend, I am puzzled. (Ezra Pound)*

### 1.05.1.1 Terminology

Psychobiology describes the relationship between psychological and behavioral processes and the underlying physiological mechanisms. It is not only brain functions that are the scope of psychobiology but also the interplay of all bodily activities with behavior and cognition. The term biological psychology is often used synonymously. While physiological psychology and behavioral neuroscience deal with the CNS (central nervous system)–behavior interaction, psychobiology encompasses both peripheral–physiological and CNS functions. Neuropsychology applies a comparable strategy but uses naturally occurring lesions in humans.

### 1.05.1.2 The Three-systems Approach

All disorders of behavior and thought have a physiological basis, and virtually all physical disorders are strongly related to deviant behavior. Therefore, any separation between mind and body, or brain and behavior, is unjustified. For each disorder, the specific psychological and physical variance must be specified. Any principal distinction between the two levels of observation compromises progress toward the understanding of the etiology of diseases. The psychobiology of disease is an integral part of clinical psychology; any attempt to ignore its significance will lead to misleading conclusions.

Behavior in general can be measured in humans at the level of *verbal reports*, often called subjective level, the level of *physiological responses* (including hormonal, endocrine, and immunological), and the level of *overt (motor) responses*. The three response levels are usually elicited by stimuli in the environment. Their impact on the three systems is defined by their

*meaning or significance*. As we shall see in Section 1.05.11.3, the construction of meaning is based on a well-described associative learning mechanism in the CNS. The creation of meaning consists of a relatively fast *automatic* process (for simple stimuli it lasts up to 200 ms) which in most instances does not need conscious, *controlled* information processing.

One of the main reasons which makes the *simultaneous* observation and measurement of all three response levels mandatory in clinical psychology is their generally *low correlation* in a given stimulus context. Exceptions, such as extremely intensive emotions (e.g., terror), are rare. The problem becomes particularly obvious if we refer from a given response channel (e.g., an angry outburst) to an *underlying construct* such as an emotion. The different measures of the given response would be expected to be highly correlated with each other because they would be indicators of the same construct.

However, as shown by Öhman (1987), it is quite complicated to decide what a “high correlation” means in this instance. Since behavior, physiology, and verbal reports represent very diverse data domains, most of the variance in each of the measures would be attributable to factors that would not contribute to the variance in the others. For example, a peripheral–physiological measure such as heart rate is primarily constrained by the design and demands of the cardiovascular system, and the variance left for psychological factors is necessarily small. Similarly, verbal reports are shaped by cultural conventions including linguistic limitations which may not affect heart rate at all. Since a high correlation between two measures requires that their variance is determined by the same sources in approximately the same proportions, high correlations between, for example, heart rate and verbal reports would be unlikely. In fact, using the theorems of factor analysis for a theoretical treatment of this problem, Öhman has shown that the maximal between-response channel correlation one could realistically expect in the present case would be 0.30 rather than 0.70. Indeed, even within one of the response domains, for example, within autonomic responses, covariations between response measures and various emotional stimuli

often barely reach statistical significance, typically ranging between 0.2 and 0.3 (Fahrenberg, Walschburger, & Foerster, 1979). Given this limitation on the expected covariation, one would be likely to encounter frequent cases where, for example, behavior and physiology would provide discordant bases for inferring an emotion (for a more extended discussion of the covariation problem see Birbaumer & Öhman, 1993).

In stressing the necessity of physiological definitions and measurements in clinical psychology, particularly neurophysiological and neurochemical studies, we emphasize that physiology cannot substitute for, but rather is dependent on, the other levels. Even with perfect knowledge about the neurophysiological mechanisms, the definition of a disordered behavior requires, in addition, the contextual—situational, the subjective—verbal, and the behavioral components in order to be complete.

The covariation or a lack of covariation between the three response levels frequently becomes the main source of variance in the etiology or maintenance of a disorder. In panic disorder, for example, the aberrant subjective perception and evaluation of cardiovascular responses constitutes the main etiological factor involved (lack of correlation between response levels). On the other hand, in phobias, Lang, Melamed, and Hart (1970) have convincingly demonstrated that a high correlation between the physiological response (cardiovascular in this case), expressive motor behavior (avoidance), and verbal response (fear) provides the best predictor for fast extinction of the response. In some forms of schizophrenia, the discordance and variability between some of the response systems is often regarded as the core of the disorder (see Section 1.05.11.5).

Since physiological mechanisms play such a pivotal role in the etiology of most disorders of behavior, their *assessment* is necessary for planning and documenting *psychological treatment*. Most treatment outcome studies in clinical psychology and psychiatry are satisfied with the documentation of changes in a verbal report or, in the more advanced publications, include systematic behavioral observations. Psychophysiological measurements are more expensive and time-consuming and require appropriate training. Therefore, many clinical psychologists and psychiatrists avoid their application. As will become apparent in the following sections, a lack of acceptance and credibility of psychological treatments and high rates of relapse related to insufficient treatment outcome measures constitute the negative consequences of neglecting the psychobiological response system.

## 1.05.2 GENETICS AND CLINICAL PSYCHOLOGY

The knowledge of the principles of genetics is critical for the clinical psychologist for several reasons. The genetic make-up of an individual not only determines the limits of systematic behavior modification and treatment but in many disorders allows the guidance of interventional strategies toward the plastic gene-behavior chains and the investment of modification efforts in already fixed and rigid behavioral expressions of genetic activity.

Modern genetics teaches us that the genetic apparatus is a highly flexible system which allows environmental (learning) factors to influence its functions at most if not all levels of synthesis and expression. A nature—nurture polarization, still popular among behavioral and molecular scientists and the public, is *incompatible* with our present state of knowledge. Clinical psychology should incorporate these new scientific advances to target interventions according to the existing biological limits and possibilities. A detailed discussion of genetic foundations of clinical psychology can be found in Chapter 1.04, this volume.

## 1.05.3 PSYCHONEUROENDOCRINOLOGY AND PSYCHONEUROIMMUNOLOGY

### 1.05.3.1 Introduction and Definitions

Psychoneuroendocrinology and psychoneuroimmunology deal with the study of the relationship between the hormonal and immune system and behavior, particularly in humans. Behavioral endocrinology and behavioral immunology use mostly animals for the same purpose. Several excellent texts are available for an in-depth review of both fields (cf., Ader, Felten, & Cohen, 1990; Becker, Breedlove, & Crews, 1992; Nelson, 1994). Here we provide a short overview of the main topics of both fields without a repetition of basic immunology or endocrinology. Both can be found in the textbooks on physiology and the above-mentioned introductions.

There are intricate relationships between the nervous system, the immune system, and the endocrine system. Behavior (on all three levels: motor, cognitive, and physiological) is not directly influenced by the two systems but hormones and immune factors enter the nervous system and change behavior through that system. It is important to note that there are reciprocal interactions between the nervous system and behavior: behavior is not only affected by the hormonal and immune system but together with environmental stimuli and the

consequences of a particular behavior, behavior itself affects again the two “slow” bodily systems.

Hormones or immune factors can have *organizing* or *activating* effects. An organizing effect would, for example, be the specific formation of hypothalamic nuclei during intrauterine development which later in life cause heterosexual or homosexual behavior (see Section 1.05.9.3.6). An activating effect consists, for example, of the unspecific secretion of ACTH after stressful stimuli. Hormones influence sensory systems, the CNS, as well as the effectors. The hormonal and the immune system are phylogenetically very old systems (from insect to humans with similar structure and function) and act relatively slowly on behaviorally relevant structures. Slow means that the direct actions of the nervous system on muscles are usually much faster (in the millisecond range), whereas the “wet” bloodstream-dependent hormones and immune structures exert their effects within seconds, hours, days, or months. Both fields become increasingly important for clinical psychology and behavior modification: many clinical psychological and psychiatric disorders are caused or at least in some aspects influenced by endocrine and/or immunological agents. Additionally, the tailoring of psychological intervention strategies to endocrine and immunological changes in patients is extremely important, as can be seen from the intricate interplay of circadian hormonal rhythms and depression, sex hormones and sexual deviations, and in the modification of antisocial behavior, treatment of obesity and eating disorders, and stress-related disorders.

### 1.05.3.2 Hormones, Bodily Rhythms, and Behavior

Nearly all hormones are synthesized and/or secreted in a rhythmic manner, triggered by endogenous oscillators in the CNS which can be *entrained* to exogenous (mainly light) or psychological (mostly social) stimuli within certain limits.

### 1.05.3.3 Hormones and Emotions

The hormonal reaction to separation and loss constitutes the most intensively studied emotional reaction because of its obvious relevance to the pharmacotherapy of depression. Disruption of the circadian rhythm of GH, ACTH, and cortisol is accompanied by an initial increase in some endogenous opioids in the periventricular gray and a depletion of noradrenaline (NA) and serotonin (5-HT). A productive research direction to understand antidepressive agents might

be the insight into the inverse of separation, namely social attachment and love.

Attachment and love certainly contain strong elements of psychological dependency and in the case of separation, withdrawal reactions in the form of sadness or jealousy. Therefore, the considerations in Section 1.05.9.4 concerning drug dependency and the dopamine system are also relevant for love and attachment. All substances which stimulate the limbic dopamine system (opiates, benzodiazepines, and alcohol) reduce the symptoms of separation withdrawal. Particularly effective are direct injections of opiates in the ventral tegmental dopamine system; injection of naloxone, an opiate antagonist, produces cries of separation in rats and monkeys (Wise, 1988).

Affiliative behavior is, however, *heterogeneous* (pair bonding, parental care, mutual defense, sexual interaction, and altruistic-helpful behavior), each behavior will correlate with a specific neurohormonal profile (*response stereotypy*). Situational factors such as touch, smell, eye contact, and temperature play a central role in the formation of those emotion-specific hormonal profiles (*stimulus stereotypy*). The lack of physical contact between many developing mammals including humans and a caregiver prevents slow wave sleep and GH secretion and causes psychosocial dwarfism and autistic social behavior. For example, tactile stimulation in rat pups causes synthesis of the enzyme ornithine decarboxylase (ODC); separation stops the synthesis and brush strokes analogous to the mother's licking restore ODC production. ODC is an enzyme essential for tissue growth and development.

The peptide hormone oxytocin was for a long time known to be the central hormone in the initiation of milk ejection and uterine contractions for viviparity. However, extrahypothalamic hypophyseal oxytocin plays a totally different role from hypothalamic and brain stem-produced oxytocin. The latter is universally important for initiation of maternal behavior, sexual attraction, grooming, and monogamy. It seems as if a certain critical level of oxytocin has to be present in the hypothalamus in order to make social interaction rewarding (Schulkin, 1993). Central  $\beta$ -endorphin and oxytocin both act in an agonistic fashion on the central dopamine psychomotor reward system (see Section 1.05.9.4.2). The behavioral effects of oxytocin and endorphins depend more upon the location of their synthesis in the brain and less on their biochemical or physiological actions on nerve cells or glands. Both are strongly influenced by the circadian rhythm and suppressed during negative emotions such as stress, anxiety, and depression. Soft tactile stimulation

on nipples and genitals in social interaction increase their production. Stress, anxiety, and depression are discussed in Section 1.05.10.

#### 1.05.3.4 Relationships Between Nervous and Immune System

The nervous system has various direct and indirect (mostly) endocrine connections with different (but not all) parts of the immune system and vice versa: immune factors regulate and modify several neuronal responses mainly through endocrine effects on the CNS but also within the nervous system through direct activation of immune cells by psychological and physical stimuli.

Several substances in the nervous system (NS) constitute signals for the immune system: these are the main neurotransmitters such as NA, acetylcholine (ACh), and excitatory amino acids, such as glutamate. Neuropeptides modulate immune function through neurotransmitters or directly. Most important are substance P, VIP (vasoactive intestinal peptide), and endogenous opioids. Endocrine growth factors and cytokines (interleukines and interferons) also act as neuroactive messengers.

The immune system, on the other hand, sends signals to the nerve cells through cytokines, neuropeptides, and neurotrophins (such as GH). Immune cells possess receptors for neurotransmitters and peptides, whereas nerve cells are affected mostly indirectly through receptors for neuropeptides, histamine, and neurotrophins. Nerve cell metabolism is, however, regulated by glia cells which contain cytokine receptors in addition to all other types of membrane receptors (cf., Schedlowski & Tewes, 1996).

#### 1.05.3.5 Stress and the Immune System

There is agreement that some but not all types of stressors have an immunosuppressive effect which may later contribute to initiation or maintenance of illness and "sickness behavior" during infections or other immune disorders. Depending upon the type of stress, very different and highly specific responses of immune cells result. The main effects of stress on the immune system are mediated by the ACTH-glucocorticoid release of the anterior pituitary-adreno-cortical axis. Glucocorticoids have immunosuppressive effects on some but not all cytokines (i.e., cytotoxic T lymphocytes, CD8<sup>+</sup>). The immunosuppressive effect may be desirable or adverse for the organism. Glucocorticoids show their immunosuppressive effect mainly on nonactivated immune cells. If they have already been activated by antigen presentation, their effect is negligible. Therefore, in

humans, stress increases the probability for newly acquired infections but rarely affects already active immune diseases.

*Learned helplessness* stress (see Section 1.05.10.3.2) dramatically reduces CD4<sup>+</sup> T helper cells through glucocorticoid increase and leaves the organism more vulnerable to new infections. This may explain why in older or already weak populations, loss of the partner results in a significant reduction of survival because of an increased susceptibility to disease (cf., Schedlowski & Tewes, 1996).

On the other hand, an increased plasma level of glucocorticoids after physical stress, for example, blood poisoning, *protects* the organism. Adrenalectomized animals, who are deficient in glucocorticoid release, die from severe stress, whereas intact animals survive severe stress because an excessive immune reaction to external toxins is prevented by the release of glucocorticoids. In addition, some cytokines stimulate the adreno-cortical-pituitary axis and suppress the growth of tumor cells with reactive stimulation of ACTH and cortisol. Therefore, use of the term, "stress-induced immunosuppression" as an indicator of an adverse effect on health and homeostasis is too general to be useful. It has to be specified which psychological variable leads to which specific immunological change. These changes can be protective or harmful for the organism.

Several studies have shown that *behavioral treatment and social support* have a protective effect on various immune parameters and diseases. In human immunodeficiency virus (HIV)-infected persons, the destruction of CD4<sup>+</sup> helper cells and other lymphocytes can be significantly retarded. Survival rates of aging populations can be prolonged for years with appropriate behavioral and social interventions targeted towards improving self-control and self-efficacy. These studies document an increase in lymphocytes and long-term reduction of cortisol levels in the intervention groups (Schedlowski & Tewes, 1996).

#### 1.05.3.6 Learning and the Immune System

Early in the twentieth century, several publications reported the classical conditioning of immune reactions. Most, if not all, of the reports remained unnoticed until 1975, when Ader and Cohen published their seminal paper on classically conditioned immune suppression by using a conditioned taste aversion paradigm in rats (cf., Ader & Cohen, 1985; Ader et al., 1990). The unconditioned stimulus (US) cyclophosphamide (CY) causes severe gastrointestinal disorders and suppression of a host of immune factors. The rats in the experimental group



received the neutral saccharin as a conditioned stimulus (CS) and shortly afterwards an injection of CY as US. Three days after the conditioning trial only the CS was given and sheep erythrocytes were injected as an antigen-stimulating agent. After nine days the animals were killed and the amount of antibodies against the antigen was counted. A profound reduction of proliferation of T lymphocytes and natural killer (NK) cells was found. The same effect can be induced by the operant procedure of learned helplessness (see Section 1.05.10.3.2): animals in a yoked group that could not escape repetitive aversive shocks showed lasting immune suppression in comparison with the escape group with the same number of shocks presented. Adrenalectomy prevented the effect, demonstrating that the pituitary–adreno–cortical axis is involved in the operant effect but not in the classical conditioning effect; the latter remains after adrenalectomy.

Not only immunosuppression but also activation of immune cells can be classically conditioned, for example, by use of the injection of the cytokine agonist ovalbumine protein as a US. Clinically relevant for human transplantation surgery are experiments demonstrating that skin transplants are much less sensitive to *graft* vs. *host reactions* if the immunosuppressive CS is presented again after transplantation (cf., Ader et al., 1990).

Conditioned immunosuppression and immunoinduction has many potential clinical applications. In autoimmune diseases such as lupus erythematoses, the weekly presentation of an immunosuppressive CS significantly reduced mortality in rats. In humans, conditioned nausea during radiation therapy for cancer can be successfully eliminated by a systematic desensitization and extinction procedure (Heninger, 1995). Moderate physical (pleasurable) exercise leads to a dramatic increase in lymphocytes and NK cells; the antimicrobial activity of macrophages is strengthened and depressive mood, which strongly exacerbates immune suppression, is reduced. Since cytokines such as interleukin-1 themselves produce changes in corticosteroid release and monoamine turnover in the brain, the immune system provides negative and positive feedback loops on emotional brain systems.

#### 1.05.4 NEUROCHEMISTRY AND NEUROPHARMACOLOGY

##### 1.05.4.1 Psychopharmacology and Clinical Psychology

Neurochemistry, psychopharmacology, and the scientific disciplines studying the molecular

mechanisms of neurons and synapses are the most rapid growing fields in the biobehavioral sciences. The enormous interest and financial investment is primed by the factual and anticipated economic benefits of drug treatments and the relative ease and time-saving application for clinical problems. A representative overview of the rapid growth is provided by the 2000-page volume *Psychopharmacology. The fourth generation of progress*, edited by Bloom and Kupfer (1994). An introduction to basic neurochemistry can be found in Siegel, Agranoff, Albers, and Molinoff (1994).

The relationship between psychopharmacology and clinical psychology is characterized by an attitude of mutual exclusiveness on both sides. At best, biological psychiatrists or psychopharmacologists construe a psychological treatment as an adjunct to the “real” pharmacological treatment. Clinical psychologists, on the other hand, sometimes see the biologically oriented treatments as “unfair competition” to their time-consuming and often nonspecific socially oriented treatment approaches. This is a very unproductive state of affairs since the potential profit of integration could be substantial for both disciplines. If we conceptualize the brain–behavior–cognition relation as a unitarian identity principle or at least as a tightly coupled parallelism, as most neopositivistic natural scientists would prefer, some strategic consequences for the application of psychological, neurochemical, and neurophysiological modifications are obvious. Psychobiologists should, for example, obtain the legal right to prescribe some types of medication.

Psychological or behavioral treatments are often effective for “purely” physical diseases without a psychological etiology, while physical or pharmacological therapies are frequently indicated for complex psychological problems with no or less clear-cut pathophysiology. Behavioral medicine provides many examples of the physical effectiveness of behavioral treatments, while psychiatrists often treat psychological problems (i.e., marital conflict, addictions) effectively with drugs. For example, relapse after drug abuse can be prevented by drugs chemically antagonistic to the one abused (see Section 1.05.9.4, despite the conditioned origin of relapse).

The construction of psychological intervention strategies can and should be modeled after well-known and effective neurophysiological or neurochemical treatment modalities while avoiding the inescapable negative side effects of medical procedures. Along the same lines, drugs or medical strategies should be matched to specific behavioral and psychopathological mechanisms of a particular disorder.

The biological boundaries inherent in some disturbed behaviors or diseases can only be transgressed with physical approaches, while the social boundaries of disorders (e.g., the addictions) construct the limits of biological intervention.

Presently we are more distant than ever from an interdisciplinary interaction and respectful understanding and incorporation of the other discipline in one's own views. The widening gap between biological and social or psychological sciences has many reasons; since human beings have only very limited information-processing capacities, the overflow of scientific information in both fields causes an increasing protective tendency to exclude and ignore deviant and new principles. The narrower and the more rigid the individual's intellect, the more readily other positions are discarded. A still prevalent dichotomy of the mind-brain relationship, particularly in the psychological realm, undermines any fruitful interaction with the questionable argument of the infinite complexity and the intellectual inaccessibility of "subject-object" relations (cf., Lycan, 1996, for a philosophical analysis).

#### 1.05.4.2 Mechanisms of Action of Psychoactive Substances

##### 1.05.4.2.1 Neuronal mechanisms

Behavior and mental activity are represented in the CNS as *cell assemblies* which were formed by associative strengthening of synapses in simultaneously activated neurons (Hebb, 1949). Mental processes are coded as multidimensional *vectors* of excitatory electrical activity in strongly coupled assemblies or so-called "*syn-fire chains*" (Abeles, 1991). These electrical patterns of synchronous activity tend to *oscillate* in various rhythms because the plastic cell assemblies and brain regions all have recurrent networks forming a virtually endless reticulum of feedback and feedforward connections. The structural, physiological, and molecular processes which determine the electrical activity of nerve cells and neuronal assemblies constitute the biophysical basis of behavioral pharmacology, which attempts to influence the electrical vectors through modification of the neurochemical determinants at the cellular and synaptic levels.

The electrical properties of a cell depend primarily on the structure of its membranes. Those in turn are built by the proteins which are synthesized by the genetic apparatus. Transcription and translation can occur "spontaneously" as part of the intracellular metabolisms or are induced and modified at various

steps by incoming external synaptic or hormonal signals.

Plasticity and behavior change therefore depend ultimately upon synaptic changes which alter themselves to dendritic and somatic extra- and intracellular responsivity. During the resting state the membrane receptors are inactivated and an imbalance of electrically charged ions is maintained so that the intracellular membrane is more negatively charged ( $\approx 70 \mu\text{V}$ ) relative to its external part. With the arrival of an action potential which consists of a rapidly progressing wave of depolarization, the voltage gated  $\text{Ca}^{2+}$  channels are opened and  $\text{Ca}^{2+}$  enters the synaptic terminal.  $\text{Ca}^{2+}$  prompts the binding of synaptic vesicles containing the transmitter substance onto the presynaptic membrane terminal which causes their release into the synaptic cleft. The transmitter molecules fuse (bind) with specific receptor molecules at the postsynaptic membrane if they match allosterically the shape of the transmitter. In the case of a match between receptor and transmitter, the postsynaptic membrane channels open and positively charged ions (mostly  $\text{Na}^+$ ) are allowed to follow their electrochemical gradient and other forces quickly (less than milliseconds) and *depolarize* the postsynaptic membrane. Depolarization consists of a change in electrical polarization from an electrically positive charged extracellular membrane to a negatively charged potential ( $20\text{--}100 \mu\text{V}$ ). Only if many depolarizations at multiple synaptic sites in one cell occur simultaneously will an action potential be created at the axon hillock and information transmitted to other cells.

##### 1.05.4.2.2 Receptors for neurochemicals

Receptors are the constituents of a cell that have the ability to recognize a drug, a hormone, or a transmitter. Membrane-bound receptors such as those for transmitters cause rapid ion flows as described above and activate the so-called *second messenger systems*. This rapid mechanism of opening membrane channels for depolarization happens within one to several milliseconds and is called *classical neurotransmission*.

Steroid hormones enter the cell membrane and bind to steroid receptors which enter the membrane of the nucleus and act directly on DNA-RNA transcription. The second messengers, among the most important  $\text{Ca}^{2+}$ , cyclic adenosine monophosphate (cAMP), and inositoltriphosphate ( $\text{IP}_3$ ), cause a cascade of intracellular chemical reactions which ends with the *phosphorylation* of a protein by the protein kinases. Phosphorylation consists of the binding of a phosphate molecule with a protein that

activates its enzymatic properties, and only through this mechanism do proteins become functional and modify a cellular response.

*Neuromodulation* is slower and uses the binding of an agonist to a nonchannel receptor. Instead of involving an ion channel, these receptors are proteins that change their cytoplasmatic (intracellular) “tail” in order to bind with a so-called G-protein (a guanine nucleotide). The activated G-protein remains in an activated state for a much longer period (10–100 ms) than ion channels, again binding to adenylyl cyclase which activates the second messenger cAMP, repeating the above-described intracellular cascade. cAMP then enters the nucleus and selectively increases the expression of genes. The later phosphorylated ion channel or G-protein-coupled receptor may change the excitability of the membrane for a particular neurotransmitter.

#### 1.05.4.3 Transmitter Systems

The description of behaviorally relevant transmitter and neuromodulator systems followed the discovery and isolation of four psychoactive drugs in the 1950s: reserpine and chlorpromazine in the treatment of schizophrenia, and iproniazide and imipramine for depression. Binding arrays of receptors and cloning of receptors finally elucidated their distribution and action in the CNS.

Three main classes of neurotransmitter systems and some new transmitters can be differentiated: amino acids, amines, and peptides. New transmitter systems whose behavioral functions are less researched include arachidonic acid, nitric oxide (NO), proto-oncogenes, and purinoceptors.

##### 1.05.4.3.1 Amino acids

Excitatory amino acids, particularly L-glutamate, constitute the main excitatory transmitters in the CNS. There is virtually no behavioral function, including memory, learning, and higher cognitive functions, that does not involve glutamate. Three classes of glutamate receptors have been distinguished: *N*-methyl-D-aspartate (NMDA), kainic acid (*K*A), and  $\alpha$ -amino-3-hydroxy-5-methyl-4-isoxazole propionic acid (AMPA) receptor. The functional physiological range of glutamate and glutamate receptors is extremely narrow and even slight deviations cause a host of neurological and psychological disorders through *excitotoxicity*. Excitotoxicity of glutamate seems to be involved particularly in schizophrenia and Alzheimer's disease. The three receptor types are distributed at different

locations within the brain. They consist of several families of receptor subunits, each with different functional properties. The highest concentrations are found in the cortex and hippocampus, which indicates their role in excitatory cell-assembly formation for associative memory.

##### 1.05.4.3.2 Amines

The catecholamines (adrenaline, noradrenaline, dopamine, tyrosine, and others), serotonin, and histamine are the most important CNS amines. Each of them matches several receptors at the pre- and postsynaptic membranes. Their overall quantity in the CNS is low (approximately 3–5%); their functional role in behavior, however, is enormous. Dopamine of the mesolimbic system constitutes the main transmitter system in the organization of positive reinforcement and *incentive* motivation. Its overactivity, probably caused by dysregulation of cortical and hippocampal glutamate systems, is involved in the etiology and maintenance of schizophrenia; the blockade of dopamine receptors through neuroleptics reduces active hallucinatory symptoms but at the same time causes *anhedonia* and—through reduction of dopamine in the motor nigrostriatal system—*Parkinson's disease* and tardive dyskinesia. Overactivity of both the central and peripheral noradrenaline system is related to *anxiety* states and underavailability of NA to *depression*.

The distribution of *serotonin* (5-hydroxytryptamine, 5-HT) is also widespread in the brain. Tryptamine and melatonin are chemically related hormones. Again, despite its small quantity in the CNS, the significance of serotonin for behavior, mood, and biological rhythms is significant: *eating, sleep, sexual behavior, circadian rhythm, mood, and aggression* are more or less regulated by the diverse 5-HT transmitter-receptor systems.

##### 1.05.4.3.3 Opioids

Opium has been known to humans for thousands of years as an analgesic and a euphoria- and obstipation-inducing substance. However, only during the past years has its full importance and structure been elucidated. The fact that our peripheral and central nervous system contain receptors for plant opioid alkaloids was one of the most significant discoveries of pharmacology. In the CNS, opioid receptors are found as in the periphery, centrally, close to pain structures such as the periaqueductal gray, thalamus, subcortically near reflex centers, and in the hypothalamic-pituitary system. The main subtypes of *mu*, *delta*, and *kappa* receptors are differentially

distributed in the CNS and subserve different behavioral functions: all three are involved in pain modulation, others are critical for salt–water balance, cardiovascular functions, sexual behavior, grooming, memory, and the immune system. The addictive properties of opioids are probably related to their affinity with the positive reinforcement system (see Section 1.05.9.4) and with dopamine.

#### 1.05.4.3.4 *Neuropeptides*

Short-chain amino acids are called peptides and were originally believed to serve mainly hormonal-neuromodulatory roles. We know today that they can function as neuromodulators, hormones, and neurotransmitters depending on their location, molecular substructure, and receptor types. For most of them their role in behavioral functions is unknown. For some, a well-defined psychological “profile” has been developed such as ACTH and stress, oxytocin and the formation of social bonds, substance P and pain, growth hormone and development, sleep and immune function, and the above-described opioids, enkephalins, and endorphins and their role in pain modulation.

#### 1.05.4.3.5 *Acetylcholine*

Acetylcholine (ACh) is not only the transmitter of the somatic motor system and the parasympathetic system but also a central nervous system agent of great importance. Its functional effects depend on pre- and post-synaptic receptors, nicotinic and muscarinic, which can be classified into several subtypes. Most ACh receptors in the CNS cause excitatory activity (depolarization) at the postsynaptic membranes.

ACh has a modulatory role in the CNS in that its fiber system lacks specific point-to-point projection but exerts a more diffuse, unspecific function on its target cells. Therefore, ACh is usually identified as the main transmitter in arousal and attention. Most neurons originate in the basal forebrain of the *nucleus basalis Meynert* which projects to limbic areas (amygdala) and the entire cerebral cortex. Many intracortical connections are cholinergic and depolarize large groups of assemblies as a threshold regulation mechanism in learning and attention (Birbaumer, Elbert, Canavan, & Rockstroh, 1990; Mesulam, 1995). The *unspecific activation systems* (ascending reticular activating system, ARAS) of the midbrain reticular formation and the thalamus and parts of the basal ganglia are all cholinergic. Learning increases ACh activity, leads to an enlargement of cortical spines on plastic neurons, and

induces sprouting in dendritic growth. In Alzheimer’s disease, most basal forebrain neurons die. Electroencephalogram (EEG) desynchronization and negative slow brain potentials depend on an intact ACh system. Cholinomimetic drugs may prove useful for many psychological disorders; to date their side effects are substantial.

### 1.05.5 PSYCHOPHYSIOLOGY

#### 1.05.5.1 Introduction

The interest of humankind in body–soul or body–behavior relationships probably dates back to Stone-age populations about 250 000 years ago, where trepanations of the skull were obviously performed to modify supposed head diseases. Egyptian and Greek pre-Socratic philosophy and medicine correctly attributed mental functions to the brain and emotional ones to “humors” (Hippocrates) and autonomic functions, particularly the heart. However, Aristotle’s (384–322 BC) view of the heart as the seat of nervous and mental control was maintained until early Renaissance around 1450. Only Arab and Chinese traditions provided exceptions to the strict Aristotelian–Christian rule during the early Middle Ages, particularly in southern Italy and Spain (e.g., Avicenna, whose real name was Ibn Sina).

Psychophysiology together with the rest of medicine had to wait until the mid-nineteenth century for its first experimental investigations, which were largely dependent upon the construction of electrophysiological recording devices after Galvani’s first demonstration of bioelectricity in 1791. The first animal EEG was recorded in 1875 by Richard Caton, and the first human EEG in 1924 by Hans Berger (for a history see Clarke & O’Malley, 1995).

The modern development of psychophysiology is tightly coupled to the construction and availability of the *polygraph*, where several physiological measures can be recorded simultaneously. The foundation of the Society for Psychophysiological Research in 1961, which grew from a small US-only society into a large international organization, marked the worldwide establishment of relatively cheap and universal recording devices.

#### 1.05.5.2 Concepts in Psychophysiology

##### 1.05.5.2.1 *The three-systems approach*

In order to understand human behavior, in most instances physiological, psychological (cognitive subjective), and motor-behavioral variables have to be assessed in their particular environments. Origin and consequences of this

three-systems approach to human behavior as a “whole” were extensively described earlier in this chapter.

#### 1.05.5.2.2 *The law of initial values*

The law of initial values (LIV) states that the higher the initial value of a particular physiological function, the smaller the increase of this physiological measure in response to a particular stimulus or psychological event. Conversely, the higher the initial, baseline value, the larger the decrease to a stimulus or event which usually produces a decrease in the particular physiological system.

The LIV was found to be roughly true for cardiovascular variables, pupil dilation, vascular variables, but not for skin conductance or skin temperature. In cortical blood flow such as measured by PET or fMRI (position emission tomography or functional magnetic resonance imaging, see below) and with electrocortical measures, the validity of the LIV remains uncertain.

#### 1.05.5.2.3 *Activation, arousal, and directional fractionation*

Every behavior, sensory experience, and physiological response varies on a continuum of intensity. However the relationships between the intensity of a particular variable and other variables are rarely linear. The term “activation” is usually applied to the more long-lasting *tonic* (hours, days, months) changes in intensity, mainly within the sleep–wakefulness continuum, while “arousal” signifies the rather short-lasting *phasic* (milliseconds to minutes) changes; the border between both is, however, not fixed. Arousal therefore “rides” on top of a particular activation level being subject to the LIV.

Early in the twentieth century, Yerkes and Dodson independently discovered an inverted U-shaped relationship between muscle tension (activation) and performance in various tasks; later called the Yerkes–Dodson Law. After discovery of the ascending reticular activation system ARAS by Lindsley, Magoun, and Moruzzi (cf., Birbaumer & Schmidt, 1996), a neurophysiological basis of the inverted U-function seems to be at hand, because many neurons in this system showed firing rates compatible with the “law.” In addition, moderate electrical stimulation of the ARAS improves performance, but vigorous or rhythmic low-frequency electric stimulation has deleterious effects on behavioral output in animals (Andreassi, 1995). In sum, most studies confirmed the inverted U-shaped curve; how-

ever, for some physiological variables and for some types of performance the “law” does not apply.

In order to explain those instances, Lacey and Lacey (1970) formulated their concept of *directional fractionation* of physiological and motor responses. According to this concept, the different physiological response systems are activated or simultaneously deactivated depending upon the physiological and behavioral requirements of the particular situation. Directional fractionation is partially overlapping with another term coined by Lacey and Lacey, stimulus–response (SR) specificity.

#### 1.05.5.2.4 *Stimulus–response specificity*

Different stimulus situations may call for different response requirements, causing different and situation-specific physiological adaptations. Psychophysiology provided many examples for the correctness of this statement, despite the fact that most situation-specific adaptations occur in body systems that are difficult to record from the intact human without invasive recording, such as colon movements in anticipation of food.

One of the best-studied examples of stimulus-specificity is heart rate slowing in nonthreatening sensory information *intake* and heart rate increase to potentially aversive information *rejection*, the first demonstration of this effect dating back to Lacey and Lacey (cf., Dworkin et al., 1994). The physiological mechanism for this phenomenon was elucidated and confirmed in a series of laboratory studies by Rau and Birbaumer (1993). Increase in pressure of the carotid sinus and other large arteries causes the baroreceptors to fire as a response to blood pressure increase. The afferent fibers to the brain stem end in inhibitory centers of the reticular formation, causing heart rate deceleration and short-lasting cortical inhibition. The neuronal disfacilitation is strong enough to block the impact of aversive overarousing stimulation but improves some form of low-intensity stimulus processing. Cortical slow waves, for example, are dramatically reduced by direct stimulation of carotid baroreceptors through a neck cuff stimulation device (Rau & Birbaumer, 1993). The stress- and pain-reducing impact of phasic blood pressure increase is “used” nonconsciously by genetically prone persons as a stress coping strategy; the rewarding consequences of this strategy increase blood pressure up to the point of essential hypertension.

Another example of situation- or stimulus-specificity are changes in muscle tension: electromyographic recording of facial muscles or micromovements of peripheral muscles

(Birbaumer & Öhman, 1993; Clynes, 1989) can reliably differentiate between the basic emotions (sadness, pleasure, interest, rage) even in the absence of visible changes in facial expressions. Also, imagery of emotional scenes evokes the same peripheral but partially different CNS changes (Schupp, Lutzenberger, Birbaumer, Braun, & Miltner, 1994) as actual perception of these events. These stimulus-specific physiologies interact and overlap with individual response stereotypies.

#### **1.05.5.2.5 Individual response stereotypies**

These stereotypies characterize tonic, in most instances long-lasting "preferences" of a person to react and, in the case of pathology, overreact with a preferred bodily response system in different situations. Individual response idiosyncrasies originate from genetic, constitutional, or learned causes. The concept of response specificity, as it is also called, is extremely important for the explanation of psychosomatic, psychiatric, and somatic disorders.

Apart from the development of essential hypertension, individual response stereotypies were found to be responsible for musculoskeletal *chronic pain* related to the lower back, face, and head (Birbaumer, Flor, Lutzenberger, & Elbert, 1995). While in healthy subjects individual response stereotypies are unreliable and short-lasting, they cause pathology in subjects with a high genetic risk and they are triggered under conditions of long-lasting (particularly instrumental or classical conditioning) circumstances such as intensive stress in vulnerable periods of ontogenetic development.

#### **1.05.5.2.6 Orienting, habituation, and defense**

Pavlov was the first to examine these prepared response patterns to novel, old, or potentially harmful stimuli in dogs. The principles he described for their occurrence and psychophysiological meaning are as valid today as they were 100 years ago (Pavlov, 1926). Habituation to repeated stimulus presentation is regarded as the most primitive form of learning, learning to cease responding to biologically irrelevant stimuli. In mollusks and other simple animals, the molecular basis of habituation involves the gradual reduction of intracellular cascades which eventually lead to a decreased binding of  $\text{Ca}^{2+}$  on to presynaptic terminals. The reduced release of transmitters as a consequence weakens the strength of synaptic connections between previously functionally connected cells (cf., Birbaumer & Schmidt, 1996, for a review).

In higher animals and humans, an orienting or defensive response depends on a short-term memory comparison process between actually present and stored similar stimulus configurations. In the case of a mismatch of biologically relevant stimuli, orienting occurs; in cases of potentially threatening stimuli, a slowly habituating defensive "reflex" is recorded. In the CNS, the amplitude of a positive evoked brain potential 300–500 ms after stimulus presentation, the so-called P300, is a good indicator of that mismatch process. In the periphery, heart rate decrease is mostly related to orienting, while heart rate increase is related to defensive responses. Very short-latency muscular reflexes, such as the *startle reflex*, measurable as an eye blink 40 ms after an intensive stimulus, act as an "interrupt" to threatening stimuli and are part of a whole-body escape response disposition (cf., Birbaumer & Öhman, 1993). Modulation of the startle reflex amplitude through emotionally positive or negative background material is one of the most reliable indicators of the emotional condition of the CNS state: startle inhibition through emotionally positive and startle potentiation through emotionally negative material is easy to measure and provides an excellent diagnostic tool in psychopathology (Lang, Bradley, & Cuthbert, 1990).

#### **1.05.5.2.7 Social psychophysiology**

All human behavior and subjective experience evolved out of social interaction and communication. The laboratory situation therefore creates an artificial isolation of the individual which may produce response patterns not representative of "real" life. The observation and measurement of motor and verbal behavior, cognition, and emotion simultaneously with the physiology is the most promising but methodologically difficult area of psychophysiology. During the last 20 years, a considerable amount of data has emerged which has shed light on the enormous impact of social factors on physiological response systems (Cacioppo & Petty, 1983; Hauser, 1996). Flor, Elbert, et al. (1995) recently demonstrated a dramatic effect of the presence of a solicitous spouse on the processing of painful stimuli in chronic back pain patients. The mere physical presence of the partner not only induced a more than threefold increase in pain sensitivity but brain electrical potentials to painful stimuli were increased at a very early stage (100 ms) of information processing in patients and high-risk groups. Social factors such as reinforcement history with a spouse obviously modify CNS stimulus processing,

already at an early preconscious level not accessible to conscious voluntary control.

### 1.05.5.3 Methods in Psychophysiology

#### 1.05.5.3.1 Electroencephalography

EEG frequency spectra, averaged evoked responses combined with computer-aided bioelectric imaging of many electrodes on the scalp, are still the most inexpensive CNS measures with an excellent time resolution. In addition, source localization of early evoked potentials allows in some cases a precise anatomical localization of the cortical origins of these potentials. Figure 1 shows localization of electrical sources of tactile evoked primary brain potentials components with a latency of 40–70 ms at the postcentral gyrus in patients with phantom limb pain due to amputation of one upper limb. The index finger of the intact arm and corner of the lip at both sides were stimulated with 1000 stimulus repetitions and EEG responses averaged in order to enhance the signal-to-noise ratio. The same stimulation was repeated after local anesthesia of the stump and shoulder.

The electrical sources of these potentials were calculated and superimposed on each subject's individual magnetic resonance brain image (see below). It can be seen that a precise anatomical localization of the finger and lip region is possible in accordance with the known homuncular organization of the somatosensory cortex. After anesthesia and pain relief, the pain-related pathological reorganization of the brain representations of the phantom limb returns to normal (Birbaumer, Lutzenberger, et al., 1997).

#### 1.05.5.3.2 Magnetoencephalography (MEG)

The development of low-temperature superconducting interference devices (SQUIDs) allows recording of the magnetic fields of neural structures in the brain, which are in the femtoTesla range (equivalent to  $10^{-15}$  Tesla), the earth's magnetic field being in the range of  $10^{-4}$  T. Since magnetic fields cross all biological tissues not affected by electric resistance, they can be recorded undistorted above the head. MEG has an unprecedented local resolution of 2 mm up to a brain depth of about 5 cm. Its time resolution is like that of EEG—excellent and in the millisecond range. The limitations of MEG technology are the still high costs, the necessity of a mu-metal isolation chamber, and the biophysical fact that only horizontally located dipoles can be recorded from the brain. However, together with EEG imaging, MEG provides the best time and space resolution of all

available noninvasive recordings of brain activity.

#### 1.05.5.3.3 Functional magnetic resonance imaging (fMRI)

One disadvantage of EEG/MEG imaging is its low spatial resolution 5 cm or more below the cortical surface. In order to visualize local subcortical activity changes and for an anatomically correct representation of the individual brain, MRI has to be applied. While *structural* MRI was introduced 15 years ago as a noninvasive technique to visualize gray and white matter of the human brain, *functional* MRI (fMRI) is relatively recent and in the few years of its existence has become an extremely successful measure of regional changes in brain blood flow.

The basic physical principles of fMRI are not different from structural MRI, except that with the introduction of efficient power supplies, superconducting magnets, and rapid analog to digital converters, MR images can be taken in the range of tenths of milliseconds. In order to acquire MR images, a 1–3 Tesla magnetic field is applied through the head. The magnetic field forces hydrogen proton nuclei into an aligned direction of the magnetic field. The protons exhibit the property of “*spin*”: they rotate around their axis. In the magnetic field, the spins of the protons will all be oriented and rotating in the same direction (quantization).

Of these rotating protons in the magnetic field, more of the protons are in a lower energy state which gives the proton ensemble a net magnetization. The time to magnetization after application of the magnetic field is  $T_1$  (for water, the main constituent of brain tissue, it is 2 s). Fat molecules magnetize much more slowly. Therefore, gray and white substances magnetize differently. The uniform field gives the exceedingly small magnetic dipoles because of their alignment a uniform magnetization and a uniform rotating frequency (Lamor frequency). The size of this combined magnetization of the spins is the magnetic resonance signal. With time the spins lose phase coherence and the signal decays, depending upon tissue properties (as  $T_1$ ). This time is called  $T_2$ . At the borders of tissues, a large difference of magnetization may exist over small distances and the proton spins at those borders will be out of phase rapidly. The macroscopic field varies as a consequence of this; the macroscopic decay is  $T_2$ . In order to cause the angular orientations of the protons to align, an electromagnetic field of about 63 MHz (radio band with the Lamor frequency) is added to the magnetic field, the protons' rotation is forced away from their longitudinal axis by this

**Figure 1** Cortical representation of the digits and the lower lip before and after brachial plexus blockade in a unilateral upper extremity amputee with phantom limb pain who experienced complete pain relief due to the regional anesthesia. The preanesthesia location of the representation of the fifth digit (D%) of the intact hand is indicated by the filled square, and the locations of the representations of the lip of the intact side and the amputated side by the filled squares below. The mirror images (15,18) of the intact digit and the intact lip projected onto the amputation-side hemisphere are marked by open squares. These locations refer to the preanesthesia period. The location of the representation of the amputation-side lip postanesthesia is indicated by a filled square. Note that before brachial plexus blockade, the lip in the amputated side had shifted into the region occupied by the representation of the fingers on the intact side (mirror D5). Twenty minutes after amputation stump anesthesia, the phantom pain was almost eliminated; at the same time there was a dramatic shift of the amputation-side back toward the position occupied by the lip representation on the intact side (mirror lip, open square). The white dots are the vitamin E capsules marking the electrode positions ("Effects of regional anesthesia on phantom limb pain are mirrored in change in cortical reorganization," by N. Birbaumer, W. Lutzenberger, et al., 1997, *Journal of Neuroscience*, 17, pp. 5503–5508. Copyright 1997 by Oxford University Press. Reprinted with permission).



field; however, they all now rotate in “resonance.” This field is large enough to be detected, because it induces an electric voltage at a nearby antenna. At tissue boundaries, the detected frequencies will be different and can be transformed into color contrasts.

Figure 2 shows the result of an fMRI experiment where phobic patients and healthy subjects were exposed to neutral faces and aversive odors. An increase in blood flow (because of increased net magnetization) in the amygdala (but not in the thalamus) was observed for phobics but not the healthy controls during presentation of faces. This confirms animal experiments showing that the amygdala is the critical structure for the emotional valence of fear objects and that the thalamus does not participate in the emotional “coloring” of stimuli, but only in their sensory processing.

#### 1.05.5.3.4 Positron emission tomography (PET)

When a radioactive ligand is injected or inhaled, it is taken up by the cerebral bloodstream and binds to specific cells and regions that are metabolically active. Collision of the protons of the radioactive ligand with the electrons of the particular atom (e.g.,  $^{15}\text{O}$ ) causes annihilation of the two particles and the emission of a photon. Annihilation photons carry high energy which can be detected by a ring of many radiation detectors around the head. These detectors of  $\gamma$ -radiation count the density of radiation and reconstruct their spatial sources in a color-coded brain map.

PET has a less precise spatial and temporal resolution than fMRI or EEG/MEG imaging. However, its capacity to localize specific sources of increased or decreased metabolic activity of specific transmitters and substances such as water, glucose, fluoride, dopamine, and GABA is indispensable for understanding the link between molecular neurochemical processes and the global systemic changes recorded with all brain imaging systems. In general, PET allows the reconstruction of brain areas with increased or decreased *blood flow* relative to a specified baseline condition. However, the target mental activity has to be present continuously for several seconds to minutes and is averaged over several trials before a clear enough signal-to-noise ratio appears. The high cost and its invasive nature, however, limits its value for psychophysiology.

#### 1.05.5.3.5 Electromyography (EMG)

The measurement of action potentials from the motor units of the skeletal muscles is one of

the oldest and most useful psychophysiological assessments. DuBois-Reymond recorded for the first time the electrical activity of muscles in 1849. Surface EMGs with electrodes at the skin above a muscle group record Ach-caused depolarization of the motor endplate shortly before the actual contraction, but the correlation of muscular effort and EMG amplitude is very good. The spike frequency from a motor unit ranges from 20 to 1000 Hz; the amplitudes from 50 to 1000  $\mu\text{V}$ . The main frequencies used in psychophysiological experiments range from 70 to 1000 Hz. Today, integration techniques give a measure of total EMG output over time.

EMG recording has proven to be extremely useful for studying mental load, muscular-mental tension, and emotions, particularly facial expressions: with the use of only three facial muscles, the main basic emotions (disgust, anger, pleasure, joy, interest, sadness) can be differentiated even in the absence of visible changes in facial expression. Biofeedback of EMG from the back muscles is the most effective treatment for chronic low back pain; the same is true for trapezius or neck muscles and tension headache (cf., Birbaumer & Öhman, 1993; Flor & Birbaumer, 1993).

#### 1.05.5.3.6 Electrodermal activity (EDA)

DuBois-Reymond also discovered in 1849 that an electric current applied between both hands changed its voltage with psychological events. Obviously, the electrical resistance between superficial tissue is modified by neuronal outflow to the skin. The eccrine sweat glands are innervated by the sympathetic branch of the autonomic nervous system (ANS) and use Ach instead of noradrenaline as a transmitter. The palms of the hands and the soles of the feet contain most of the body's eccrine sweat glands. During situations of emergency, ANS activity increases and the sweat glands are filled with salt-containing sweat which reduces the electrical resistance of the skin. It is unknown which brain regions contribute to this reaction. The debate whether skin conductance reflects more aversive emotional or “pure” arousal responses is still undecided. In experiments using emotional slides, EDA varied only on the arousal dimension and had no relationship with emotional valence (Birbaumer & Öhman, 1993).

EDA has been used in all areas of psychology, neurology, and psychiatry: its application for the detection of deception (see below) is highly controversial. As an indicator of the orienting response (OR), it shows distinct changes with psychopathology: lack of OR habituation in high-risk subjects predicts later schizophrenia, a

**Figure 2** Local blood flow changes in the amygdala of healthy (left) and phobic (right) subjects during presentation of a fear conditioned stimulus (below) and an unconditioned stimulus (an odor, above). Increasing dark dots indicates flow (from Birbaumer et al., in press).

high frequency of spontaneous fluctuations is found in generalized anxiety, and nonresponder depressives and schizophrenics exhibit a more chronic course of their disease (Andreassi, 1995; Davison & Neale, 1996). The absence of EDA responses to fearful stimuli is a reliable diagnostic tool for the detection of antisocial personalities and sociopathy.

#### 1.05.5.3.7 Heart activity and blood pressure

Heart rate (HR) and blood pressure are the most frequently used psychophysiological indicators. HR is used because it is easily recorded from any two electrodes affixed at the more right or left side of the body allowing the heart to be positioned between them (i.e., both hands). Blood pressure has become more popular during recent years when noninvasive continuous measurement was realized with pulse-wave velocity devices. The obvious physiological role of the cardiovascular system explains the high correlations of HR with all kinds of behaviors: motor, mental, perception, attention, and orienting; stress, emotion, and motivation; personality, social stimuli, brain interactions, and conditioning (cf., Andreassi, 1995, for a review). The dual sympathetic and parasympathetic nervous control allows separation of the two branches of the ANS: slowing of the heartbeat indicates the chronotropic parasympathetic, speeding the increased excitability and contraction force of the sympathetic branch.

For decades psychophysiology was obsessed with discussions of the psychological significance of HR changes: while Obrist (1981) and his co-workers interpreted HR changes as variations of mobilization of blood supply for the somatic musculature (*cardiac-somatic coupling*), Lacey and Lacey (1970) proposed a close relationship with information processing in the brain: HR deceleration and negative going slow cortical potentials should accompany *information intake*, acceleration *information rejection*, and positive going slow cortical potentials. The deceleration in a signaled foreperiod reaction paradigm is caused by phasic blood pressure increase which fires the baroreceptors in the carotis sinus. This leads to parasympathetic slowing of the heart and increased cortical activity because the nucleus of the vagus located in the reticular formation fires the ARAS. Tonic stimulation of the baroreceptors through a specially designed neck cuff, however, produces heart rate slowing and positive going cortical slow waves which causes inhibition of cortical activity and information rejection (Rau, Pauli, Brody, Elbert, & Birbaumer, 1993). The accompanying stress reduction negatively reinforces the preceding blood pressure increase

and on a long-term basis causes essential hypertension in genetically prone individuals. Neither Obrist's cardiac-somatic coupling nor Lacey and Lacey's information processing hypothesis was unanimously confirmed. HR slowing clearly appears in *orienting*, while HR increase after intensive or dangerous stimuli constitutes part of the *defensive reaction*.

The interaction of stress and blood pressure increase was also confirmed in different personality patterns such as type A and hostility: type A subjects (competitive, driven, impatient) with hostile attitudes toward others, demonstrate large increases in blood pressure during stressful situations and exhibit a high risk for essential hypertension and stroke. Early behavioral intervention effectively prevents this vicious circle of blood pressure increase, stress reduction through cortical inhibition, and aggressive muscular mobilization.

### 1.05.5.4 Applications of Psychophysiology

#### 1.05.5.4.1 Biofeedback

The most successful and clinically relevant application of psychophysiology became the instrumental conditioning of physiological variables mostly realized through feedback of the relevant physiological variable on a computer screen; subjects are instructed to change the variable in a particular direction and are positively reinforced for successful modifications or receive immediate feedback for it, which also functions as a reward. The scientific and clinical history of biofeedback is severely hampered by exaggerated claims of the clinical efficacy and by a very early failure to replicate the pioneering animal studies of Miller (1969) and collaborators. Meanwhile, several successful clinical indications have emerged, while the majority of controlled clinical studies revealed that most of the early expressed hopes could not be confirmed. The possibility to condition autonomic and brain functions instrumentally without mediation of the skeletal muscles as postulated by Miller was clearly confirmed in studies with totally paralyzed, artificially respirated subjects who had no voluntary control of their muscles because all motor neurons had been destroyed (Birbaumer, Rockstroh, et al., 1994): after extensive training to self-regulate slow cortical potentials (SCP), a patient with advanced amyotrophic lateral sclerosis (ALS) achieved 100% control of his brain waves and could use his learned ability to communicate by affecting a computer system with his brain waves (Birbaumer, 1998).

Well-established indications for biofeedback treatment are chronic pain states, neuromuscular rehabilitation after CNS damage,

epilepsy, encopresis and enuresis, Raynaud's disease, and scoliosis and kyphosis. Doubtful but frequent applications relate to hyperactivity, asthma, essential hypertension, cardiac arrhythmias, anxiety and depression, tics and stuttering, and athletic performance. No lasting changes of biofeedback treatment were reported in well-controlled studies for these conditions.

#### **1.05.5.4.2 Biofeedback of slow cortical potentials**

The most extensively researched area in biofeedback is self-regulation of slow cortical potentials. These EEG potentials are recorded with very long time constants (5 s to d.c.) and reflect depolarization (negative potentials) and disfacilitation (positivity) of the upper dendritic cortical layers (Birbaumer et al., 1990). Subjects watch their own SCPs on a computer screen for 2–10 s. The SCPs appear as a stylized rocket on a video monitor moving horizontally or/and vertically. Subjects have to move the rocket to one side of the screen. If positivity of the brain is required (or right and left hemispheric negativity), an "A" appears on the screen; with required cortical negativity, a "B" appears. If the correct movement is performed, immediate reinforcement (e.g., points that later change into money or a "goal" with a smiling face) appears on the screen. After 20–60 trials with feedback, subjects have to produce the required SCP amplitude without feedback, "out of their mind." No specific instructions are given because highly individual cognitive strategies are used for brain self-regulation.

Healthy subjects are able to control their SCPs after 2 h training from vertex electrodes. More localized SCP changes such as the production of cortical negativity in the left precentral region and simultaneous right-hemispheric positivity need several sessions of training (cf., Elbert, Rockstroh, Lutzenberger, & Birbaumer, 1984). After training, substantial effects on behavior were noted: vertex negativity improves performance on many cognitive and motor tasks, hemispherically localized negativity improves performance on the contralateral hand only, positivity leads to deteriorating performance. Several clinical applications were tested in controlled studies but only two showed lasting improvements superior to other established treatments: epilepsy and brain/computer communication in total paralysis (Birbaumer, 1998).

#### **1.05.5.4.3 EMG biofeedback**

Changes of the electromyographic activity of different body muscles are amplified and

patients watch them on a screen or listen to different tones reflecting the actual amplitude of the particular muscle. Patients are instructed to reduce (or increase) muscle tension in specific situations (e.g., stress confrontation) and receive immediate feedback of their attempts. In chronic low back pain, facial pain, and tension headache, EMG biofeedback of the painful muscle region has proven to be the most effective treatment approach compared with traditional medical treatments and cognitive-behavioral treatments (Flor & Birbaumer, 1993).

EMG biofeedback is also extremely useful in neuromuscular rehabilitation after stroke and other nervous system lesions: foot drop or spastic tension is effectively removed through observation of EMG activity of extensor and flexor muscles at the paralyzed or spastic region. Insertion of EMG electrodes in the anal canal and feedback of internal and external sphincter contraction became the treatment of choice for fecal incontinence and urinary incontinence after neurological disorders such as spina bifida, partial paralysis in paraplegics, old age, and incontinence after surgery. Observation of external sphincter contraction increases sensitivity and perception of anal or bladder filling and leads to sphincter control after only a few sessions.

#### **1.05.5.4.4 Detection of deception**

Detection of deception is the most controversial application of psychophysiological methods. The issue has mainly touched the USA because in Europe the "polygraph" is banned from criminal investigation and personnel selection. The understanding of the lawmakers in Europe is that as long as the guilty knowledge test (GKT) or the control question test (CQT) have not proven 100% accuracy in detecting the innocent or the guilty, it should not be used in court. The GKT informs the suspect that several items will be named by the interrogator and the suspect has to repeat them. Only one of the items is true, and the other control items are irrelevant. For example, a robber has used a gun in robbing a bank in order to (i) to buy a house, (ii) to buy a new car, (iii) to pay the doctor's bill, etc. In the CQT several questions are asked, where only one is correct: "Did you use caliber X?," "Did you use caliber Y?," etc. Skin conductance (EDA), blood volume, and respiration are usually recorded. EDA is the most valid measure, but even in laboratory settings only 90% accuracy can be achieved. If the subjects use countermeasures such as distraction strategies, the accuracy falls to 70% at best.

The situation may change with the introduction of event-related brain potentials (ERPs, see Section 1.05.6.5) as measures to detect deception. However, so far only laboratory procedures with faked crimes and subjects playing guilty have been published. (Here, some of them reach 100% accuracy.) In a promising procedure of Farwell and Donchin (1991), three types of items are presented: probes, targets, and irrelevant. Probes are related to the crime scenario, targets could only be known by the suspect. Only larger P300 components of the ERP to targets are counted as evidence. Since ERPs are difficult to control or distort by countermeasures and a lack of P300 components in a subject is already visible in the reactions to probes, this procedure might also reach 100% accuracy in field tests.

#### 1.05.5.4.5 Clinical psychophysiology

A volume by Carlson, Seifert, and Birbaumer called *Clinical applied psychophysiology* (1994) summarized many of the possibilities of psychophysiological measures in the clinic, workforce, and personnel applications. Any psychological, psychiatric, or neurological disorder that is characterized by deficits in information processing causes changes in EEG/MEG recordings and, in the case of emotional disorders, also modifications of autonomic variables and subcortical alterations in PET and fMRI. Psychophysiological measures alone rarely allow diagnosis of a particular disorder but have to be used in conjunction with other parameters (behavioral, cognitive, biochemical, and radiological) in order to characterize a disease state fully. In the case of psychiatric diagnosis where the definition of the disease itself is often unreliable, psychophysiology cannot increase the validity of the diagnosis further because validity depends on reliability.

### 1.05.6 CONSCIOUSNESS AND ATTENTION

#### 1.05.6.1 Introduction

The problem of consciousness has been at the center of scientific and philosophical interest for millennia. Biological psychology and the neurosciences have made major discoveries about the processes that underlie consciousness and attention. This research has shown that there is no unified concept of consciousness, but rather there are several heterogeneous neuronal processes that underlie these and related functions such as selective attention and short-term memory. The common physiological characteristic of the many heterogeneous forms of

consciousness is an increase in widespread arousal that is mirrored in the transition from automatic to controlled processing on a psychological level. Consciousness is closely tied to the selective activation of certain brain regions beyond a defined level of activity as well as the balance between activating and inhibiting neuronal mechanisms. Cognitive psychology has given very detailed descriptions of the various forms of attentional processes (Matlin, 1983; Näätänen, 1990); the focus of this chapter is the biological basis of attention and consciousness.

#### 1.05.6.2 Psychophysiology of Consciousness

Patients with commissurectomies (where the two hemispheres of the brain have been separated) and split brain experiments in animals have shown that split brains lead to split consciousness (Chalmers, Crawley, & Rose, 1971; Sperry, 1964). Contents that have been learned by one hemisphere must be actively transported to the other hemisphere. Without the commissures, the left hand no longer participates in the experiences of the right hand, the visual worlds of both hemispheres are completely separate, the actions of the left side of the body can no longer be described, and verbal-syntactic tasks can only be solved if they are presented to the left hemisphere.

Controlled attention is related to changes in SCPs that develop on the cortical surface during expectancy of a relevant stimulus. In a typical experiment involving SCPs, a warning stimulus (S1) precedes a required motor response that is signaled by a second stimulus (imperative stimulus, S2) several seconds after S1. The ensuing cortical negativity shows two negative peaks: the first peak, which is especially visible in frontal brain regions, represents the expectancy and resource mobilization of the sensory input channels and stimulus processing and is thus primarily related to working memory; the second negative peak reflects primarily motor mobilization and voluntary effort directed towards the response (Birbaumer et al., 1990). The provision of resources is thus proportional to the negativity, and the consumption of resources is proportional to the positivity of the slow cortical potential. The amplitude of the SCP is usually determined by both processes.

The allocation of resources and thus the allocation of cortical activation is, however, site-specific: preactivation and lowering of cortical thresholds for responses is provided in the area where a present or future activity originates. Sudden loss of control and helplessness lead to mobilization (negativity) of

resources for analysis of the new situation and related decisions. Not only the analysis of sensory input, but also the preparation for motor acts is characterized by slow cortical negativities which are low in automatic and high in voluntary, conscious movements. Voluntary movements are also preceded by (nonconscious) cortical negativities that occur about 350 ms before the consciousness of the action. Experiments on the self-regulation of these cortical negativities have shown that performance increases with higher negativity and decreases with increasing positivity, thus confirming the role of these potentials as a basis of attentional processes.

In PET studies, which allow for the analysis of both subcortical and cortical activations, the electric and magnetic data on slow potentials have been confirmed. In addition, activation in the basal ganglia and the anterior cingulate has been described during controlled processing. The cingulate gyrus and the frontal cortex seem to be especially active when executive functions of attention (such as the formation of decisions) are required, and passive attention to a stimulus does not lead to activation of the anterior cingulate (Corbetta, Miezin, Dobmeyer, Shulman, & Petersen, 1991).

Studies using evoked potentials have shown that attention rather than lack of attention in a task can be differentiated in the averaged EEG trace. Obviously, the regulation of attention needs subcortical regulation mechanisms; the regulation itself occurs, however, via thalamocortical and cortical thresholds of activation. This cortical regulation of attention ensures that every stimulus is analyzed by the neocortex (even if it never reaches consciousness) which then inhibits well-known irrelevant stimuli on a cortical level.

### **1.05.6.3 The Ascending Reticular Activating System**

The regulation of attention and consciousness is based on the activity of many widespread brain systems. Attentive behavior and preparatory planning depend on the integrated activity of multisensory and motor systems (Lindsley, Bowden, & Magoun, 1949). Whereas the widespread activation of the neocortex and the maintenance of an optimal tonic level of activation are a function of the reticular formation (RF) of the brain stem, phasic local mobilization is related to the integrated activity of the RF, the basal ganglia, the thalamus, the cingulate gyrus, the parietal region, and the frontal cortex.

The anatomical and physiological basis of waking consciousness is the mesencephalic

reticular formation (MRF). This system (also called the ascending reticular activating system, ARAS) fulfills three important functions: (i) generation of wakefulness, (ii) maintenance of muscle tone, and (iii) facilitation and inhibition of the intake and conduction of sensory and motor impulses. EEG desynchronization and behavioral activation are initiated by ascending afferent fibers from the reticular formation to higher centers. The reticular formation is activated by collateral fibers from the specific ascending tracts. During phasic waking responses the impulses of the RF interfere with the thalamic pacemakers which leads to EEG desynchronization in the cortex. In addition, the reticular formation maintains the tonic waking level by constant maintenance of a diffuse arousal of the higher brain centers above a certain critical level. Sleep occurs when the RF is actively inhibited and the afferent input is reduced (see Section 1.05.7).

#### ***1.05.6.3.1 Neurophysiology of tonic and phasic activation***

Tonic activation is primarily a function of the MRF whereas phasic activation related to selective attention is primarily a function of the nucleus reticularis of the thalamus in conjoint action with the MRF, the frontal and parietal cortex. The intralaminar nuclei of the thalamus can also activate the cortex without involvement of the MRF; this activation is, however, only phasic. Without the MRF the general waking level is disturbed (Steriade & McCarley, 1990). On a cellular level, the activating function of the MRF (there are also some inhibitory actions) relies on a tonic level of depolarized readiness in thalamic and cortical networks which is the precondition for phasic activation, waking, and paradoxical sleep. Thalamocortical activation is expressed by elevated firing rates in neurons with long axons, elevated excitability of the thalamocortical cells, suppression of secondary excitation and inhibition related to spindles and  $\alpha$ -waves in the EEG, narrowing and focusing of inhibitory processes, and depolarization of apical dendrites in the neocortex.

#### ***1.05.6.3.2 Transmitter systems involved in attention***

Despite many pharmacologically activating substances such as the amphetamines or caffeine, the transmitters and neuromodulators of the ARAS are not well established. Although most activating substances influence the synthesis of noradrenaline and dopamine, the catecholaminergic systems probably do not have a

direct influence on the MRF-thalamocortical activating structures. The origin of the monoaminergic systems in the midbrain and their extensive branching into the superior regions turns them, however, into candidate structures for activating systems. Most attempts at an exact categorization have, however, so far led to controversial results. Electrical and chemical activation of the origins of the noradrenergic system in the nucleus coeruleus and the serotonergic system in the nucleus raphé lead to hyperpolarization and inhibition of activation in the respective neocortical projection areas. Destruction of the *noradrenergic* system has no effect on EEG activation and tonic wakefulness. Only *cholinergic* stimulation in the midbrain activates cortical regions. It seems, however, to be clear that the monoaminergic systems improve the signal-to-noise ratio in cortical regions by the inhibition of specific areas and by local increases in activation.

Dopamine is, like noradrenaline, involved in many psychological functions. Whereas dopamine is not necessary for the maintenance of tonic wakefulness, dopaminergic systems regulate the construction of goal hierarchies and selective motor attention. Amphetamine in small doses improves attentional performance primarily by an increased presynaptic dopamine release and the inhibition of dopamine re-uptake from the synaptic cleft. In high doses, this leads to motor stereotypies and psychotic behavior. The regulation of attention is especially controlled by the mesolimbic *dopamine* system. Destruction of this system by 6-hydroxydopamine causes irreversible disorganization of attentional processes: animals do not habituate, they are extremely irritable, and they do not learn well; similar disturbances are seen in attention deficit disorders. Cholinergic systems seem to have an activating influence on the cortex, whereas the direction of activation seems to be determined by monoaminergic systems and other neurotransmitters and modulators.

#### 1.05.6.3.3 Gating of attention

The ability of neuroanatomical networks to select part of the incoming information and to exclude the rest from continued processing is called *gating*. The thalamus seems to have an important role in this gating process. Within the thalamus, the *nucleus reticularis thalami* seems to be the gate to the cortex. The nucleus reticularis thalami surrounds the thalamus like a shell and shows an internal structure that is ideal for the selection of incoming sensory activation: the cells in the nucleus reticularis thalami are characterized by widespread dendrites within the nucleus and multipolar axons

with many collaterals to the specific thalamic nuclei. These long multipolar axons communicate with the rest of the thalamus and the midbrain but not with the neocortical structures. The nucleus reticularis thalami is organized in a somatotopic and a visuotopic fashion: afferents from the various regions can be differentiated on the basis of their functional significance. Depending on the origin of the afferents, only the specific gate that corresponds to the respective afferent (sensory modality) is opened by the nucleus reticularis thalami.

In addition to the nucleus reticularis, the *pulvinar*, a large nucleus in the posterior thalamus, plays an important role in visual attention. It is involved in the increase of activation that occurs in the posterior parietal cortex when attention is focused on an object. The pulvinar is also closely connected with the lateral prefrontal cortex. If visual stimuli are presented with other potentially distracting stimuli, cells in the pulvinar active and activate the striatal and extrastriatal areas of the visual cortex. In the visual cortex, a signal that has been attended to leads to an elevated evoked potential after only 60 ms. This elevated evoked potential suggests that there is a very early preconscious selection by the thalamus (Posner & Raichle, 1995). The existence of multisensory comparisons on a neocortical level before active gating occurs is based on a series of studies. For example, lesions of the right parietal lobe distort the analysis and comparison of visual and tactile and also partially acoustic stimuli. The consequence is that the contralateral side of the body is ignored as well as stimuli that are presented in that direction in spite of intact perception and motor function (neglect). Attention remains fixated on the contralateral body and face. Every shift of attention leads to an increase of blood flow in the prefrontal and inferior-parietal cortex which is further indicative of the important role of this region for multisensory comparisons. Cells in the lower parietal cortex where visual, acoustic, and tactile information enters fire only when the animal is attentive to the source of the stimulus; axons of these attention cells lead to the frontal cortex as well as the thalamus and the basal ganglia. This further underlines the role of the parietal lobe for multisensory comparison.

#### 1.05.6.4 The Psychophysiology of Selective Attention

Two important parts of the limited capacity control system are the mesencephalic reticular formation as a provider of energy and the nucleus reticularis thalami as a gate for the

distribution of activation. There is an additional “decision maker” that opens the cortical areas of the thalamic (visual or acoustic) gates before the actual conscious reduction of the threshold (increase of activation) and which gates the flow of activation to the region where it is needed for further stimulus analysis or preparation of movement. This latter function is provided by the *prefrontal cortex* and the *cingulate gyrus* which receive information about the incoming activity and the result of the nonconscious comparison process from all areas of the neighboring cortex, especially (right) inferior parietal association cortex. They also receive information from the limbic system about the motivational significance of a stimulus. The second system involves the *basal ganglia* and their connection to the nucleus reticularis thalami. This system informs the nucleus reticularis thalami about the current distribution of activation in the neocortex and prevents the continued activation of already activated areas (to a level of epileptic overactivation). This system closes the thalamic gates by increasing inhibition when activation is increased in the respective cortical module and reaches a critical level.

Efferents from the frontal cortex and the mesencephalic reticular formation converge on the reticular nucleus that distributes the thalamo-cortical activation. Stimulation of the mesencephalic formation opens the gate in a nonspecific manner, that is, the amplitudes of the event-related potentials increase, and the EEG is desynchronized. This results in a general readiness for the uptake of information and orienting. Activation of the frontal cortex closes the thalamic gates. In contrast to mesencephalic reticular formation, the afferents and efferents of the prefrontal cortex are anatomically selective in that only a portion of the connections from frontal cortex to the reticular nucleus are activated, another part is silent, and thus a gate (e.g., the medial geniculate for acoustic information) is opened; all other gates remain closed. The cingulate gyrus may activate frontal areas of working memory if difficult decisions have to be made. Stimulation of the caudate nucleus leads to inhibition (positivity, lower activation) in a prescribed cortical area. All neocortical regions project to the striatum, and stimulation of this region leads to behavioral and neuronal inhibition (Birbaumer & Schmidt, 1996). This basal system prevents an increase of activation in the cortico-thalamic feedback circuits: they increase the threshold for activation when activation of the cortical modules exceeds a critical level. The higher the neocortical activation, the stronger the neuronal influx into the basal ganglia and the stronger the

closing of the gates. The frontal cortex modulates these neocortical-striatal inhibitory circuits. Disruptions of this system are always related to disturbances in consciousness and attention. Destruction of the basal ganglia leads, for example, to unconsciousness. Incomplete lesions lead to reduction of the readiness potential and disturbances of attention. If the frontal cortex is lesioned, selectivity is disturbed, the person's behavior is guided from all the stimuli that are momentarily available, and too many thalamic gates are opened.

#### 1.05.6.5 Event-related Potentials as Indicators of Attentional Processes

Evoked cortical potentials indicate the time course (latency) and the strength (amplitude) of a certain level of information processing. There seems to be a close correspondence between these electrophysiological substrates and the psychological levels of attention (Hillyard et al., 1995). Figure 3 shows the typical form of an endogenous evoked cortical potential to attended acoustic stimuli. The more automated the attention (the more frequently the stimuli are presented without consequence), the lower the amplitude, especially of the components around 20–50 ms (positive components), of N100 and all ensuing negative components. An increase of amplitudes after 50 ms which occurs during selective attention is very site-specific. The early components are only visible in the primary projection areas. The later components (after N100), however, extend to wide areas of the cortex.

##### 1.05.6.5.1 N1/P1

N1/P1 is first component of the endogenous evoked cortical potential that responds to manipulations of attention and is measured about 100 ms after stimulus onset. N1 is recorded over the primary sensory projection areas. The temporal N1 component, for example, is very sensitive to shifts of attention: if attention is shifted to the right ear, the left temporal amplitude increases and vice versa (see Figure 3). Lesions of the respective projection system abolish N1. N1 increases with stimulus intensity but responds primarily to changes in the temporal sequence of a given sequence of stimuli.

##### 1.05.6.5.2 N2

N2 can be induced by the omission of a stimulus in a sequence of stimuli. It has therefore been termed *mismatch negativity*. In particular, the first part—N2a—which is



**Figure 3** Schematic of a typical event-related potential for the time up to 400 ms after the presentation of a simple, but task-relevant stimulus. A dichotic listening task is used where tones (120–320 ms) are presented in rapid succession to the right (5000 Hz tone) or left (3400 Hz tone) ear (see upper portion of the figure). The crosses mark slightly deviant tones (targets), to which the subject has to respond with a key press. Below the averaged evoked potentials to the attended (continuous line) and the unattended (dotted line) stimuli are displayed. After 20–30 ms an elevated potential to the attended stimuli is present (based on Hillyard et al., 1995).

modality-specific seems to be involved in neuronal comparison processes of the incoming stimulus in comparison to the previously stored stimuli of the same modality. Changes of stimulus intensity also lead to N2. N2 is not sensitive to changes in voluntary focused attention and reflects, like N1/P1, automatic attention.

#### 1.05.6.5.3 P3

P3 is not related to any sensory system but occurs in a nonspecific manner, whenever an expectancy has not been fulfilled. It occurs after all target stimuli that are incongruent with a previously formed attention and it can last for seconds, depending on the amount of deviation of expectancy. P3 is a correlate of a sensory process that makes comparisons between stimuli. The stimulus must have been identified as deviant from expectancy before P300 occurs. Thus P300 may reflect an extinction process of the content in short-term memory when an expectancy has to be corrected. P300 may thus be a correlate of reflectory inhibitory processes

in short-term memory. Its location is primarily central at the vertex and parietal.

### 1.05.7 SLEEP, DREAM, CIRCADIAN RHYTHMS, AND SLEEP DISORDERS

#### 1.05.7.1 Introduction

The rotation of the earth around its axis leads to an approximate 24-hour light and temperature rhythm in plants as well as animal organisms influences almost all physiological and psychological expressions. These circadian rhythms are based on the activity of oscillators that originate in the organism and provide well-defined oscillatory periods that are in general synchronized by *Zeitgeber* in the environment. In addition to this circadian periodicity, there are a number of endogenous oscillators with short periodicities, such as EEG, respiration, and some vegetative rhythms. Rhythms which are longer-lasting than the circadian rhythms are called infradian rhythms, the shorter ones are termed ultradian

rhythms. When isolated from the *Zeitgeber* (i.e., light) of the environment, most mammals and humans continue to show a circadian rhythm. The periodicity of those freely running rhythms is, however, somewhat longer or shorter than 24 hours, in humans about 25 hours. The endogenous oscillators seem to be innate providers of rhythm. Some adaptation to external *Zeitgebers* is possible: in humans, the maximum time to synchronize is between 23 hours and 27 hours for body temperature, and for motor activity between 20 and 32 hours. Within these limits, the circadian rhythm adapts to a *Zeitgeber*. Outside this region, the rhythms are disturbed and desynchronizations between various rhythms occur (Wever, 1979).

In humans, bright light functions as the most important *Zeitgeber*. In addition, social cues are important. Circadian rhythms also determine motivational behavior. For example, the effectiveness of feedback signals and the ensuing learning and memory performance also depend on the momentary phase of circadian rhythms. The provision of water as a feedback stimulus in a thirsty organism is much more effective during the night than during daytime hours. The closeness of the coupling of endogenous oscillators and the influence of *Zeitgeber* depends on the joined physiological function of the oscillators. Every oscillator is correlated with a specific environmental cycle, that is, the oscillators must have different sensors that convey the specific *Zeitgeber* signal on the internal clock. The sensors for light–dark cycles are probably located in the retina and for eat–fast cycles there are probably sensors in the hypothalamus. True oscillators are pacemakers which measure time independent of external cues. The circadian pacemakers are located in the CNS. Peripheral pacemakers and secondary oscillators synchronize the respective organ system.

The nucleus suprachiasmaticus has been identified as the central circadian pacemaker in mammals. Lesions of the suprachiasmatic nuclei of the ventral hypothalamus lead to complete and long-lasting loss of rhythmicity. Numerous control lesions in and outside the CNS do not have any comparable effect on rhythmicity. The nucleus suprachiasmaticus forces its endogenous rhythm on other structures by the pulsating release of hormones and the rhythmic depolarization of its neurons. In primates, bilateral lesions of the SCN lead to a complete loss in activity rhythms, including the drinking rhythm without effect on the absolute amount of fluid that is ingested (no homeostatic function of circadian rhythms). Tumors in the anterior part of the third ventricle of the

chiasma lead in patients to irregular sleeping and difficulty in waking. Similar changes have been observed in the sleep–waking cycle of animals after lesions without changes in the absolute time of sleep and wakefulness.

#### 1.05.7.2 Circadian Clocks

In humans and other animals, sleep–wake cycles are the most important expressions of central pacemakers: the daily change of awareness to non-awareness or partially conscious experience dominates our life like no other bodily rhythm. Another important circadian rhythm is that of ingestion of food and fluid. The intake of food and activity cycles are highly synchronized in mammals. The rhythm is determined by anticipation of the availability of food and thus depends primarily on previous learning. Synchronization of the rhythmicity of other body functions, for example, the production of saliva before the intake of food, depends on an intact nucleus suprachiasmaticus. The anticipatory rhythm related to the search of food is, however, not dependent on the SCN but on the ventromedial nucleus of the hypothalamus. Rhythms for drinking are usually correlated with the rhythms for food intake; they can, however, also be separated from them. The drinking rhythm is strongly related to exogenous factors as SCN lesions destroy the rhythmicity of drinking. The rhythm of body temperature is similar in most mammals: after 6 p.m. the temperature reaches a maximum (Aschoff, Daan, & Groos, 1982). In a phase of inactivation it is continuously lowered and before waking up it shows an anticipatory increase. There are also specific endocrine rhythms: for example, plasma corticosteroids are synchronized with light–dark or sleep–wake rhythms. Like temperature rhythms they are difficult to influence by external factors. The rhythms of psychological variables are coupled to circadian oscillators. For example, pain perception, reaction time, vigilance, and arithmetic speed as well as immediate memory covary with circadian rhythms (Folkard & Monk, 1986).

If circadian rhythms are disturbed, such as in shift-work or during “jet-lag,” lasting consequences for physiological and psychological functions ensue. Additional disturbances of circadian periodicity are related to sleep disorders, depression, and epilepsy. The main characteristic of long disturbances in rhythm is the desynchronization of usually highly correlated physiological and psychological variables or extreme synchronization of usually uncorrelated variables. Night-work and shift-work lead

to long-lasting disturbances of periodicity and the connected physiological systems. Even experienced shift-workers maintain a low in performance after midnight; additional consequences are gastrointestinal disorders, sleep disorders, respiratory problems, pain, and immunological effects. Jet-lag leads to similar problems such as sleep disorders, gastrointestinal disorders, reduction of vigilance, feelings of nausea, and reduced immune function. The more time zones a flight covers, the more intense are the disorders. The change of only one hour from summer-time to winter-time and vice versa leads to measurable alterations, especially in older persons.

### 1.05.7.3 Sleep and Dream

Although humans spend about 30% of their time asleep, sleep became a topic of research only in the 1950s. The discovery of rapid eye movement sleep (REMS) by Aserinsky and Kleitman (1953) revealed that the brain is not passive during sleep (as was previously assumed) but that one phase of the sleep cycle (REMS) is very similar to alert wakefulness (Hobson & Stickgold, 1995). The various stages of the sleep cycle are as follows: (i) transition from wakefulness to sleep with grouped occurrences of  $\alpha$ -waves; (ii) stage 1 of the sleep cycle characterized by the loss of  $\alpha$ , low-amplitude, high-frequency  $\beta$  activity and low amplitude  $\theta$ ; (iii) stage 2 with low-amplitude fast activity with sleep spindles and K-complexes; (iv) stage 3 sleep with about 10–50% of  $\delta$ ; and (v) stage 4 sleep with  $\delta$  ( $>100\mu\text{V}$ ,  $<3\text{ Hz}$ ) occurring for more than 50% of the time. In the REM stage, low-amplitude EEG with low  $\theta$ -waves, so-called see-saw waves, occurs, otherwise the EEG is similar to an attentive waking state. K-complexes might be the correlates of a strong internal discharge of sensory systems, and spindles signal the inhibition of sensorimotor areas. From stage 1 to 4, EMG activity decreases to a complete muscular atonia during REMS.

There is considerable variability across nights and persons in the course of the sleep stages of an individual person. The first NREM stage usually lasts about an hour. The duration of the REM phases is about 104 min overall. In mammals, *slow wave sleep* (SWS: stages 3 and 4) always precedes REMS. A REM–NREM cycle lasts about 90 min, with the first cycles being shorter, and the late cycles being longer. Stage 2 covers about 50% of the entire sleep and extends in time over the course of the night, SWS is very rare in the last cycles. The duration of an individual REM phase extends from

5–10 min at the beginning to 22 min in the last phase. The approximately 90 min long REM–NREM phase has also been termed the basic rest–activity cycle (BRAC) since it may continue during wakefulness. A number of rhythms (such as eating, drinking, heart rate) seem to be synchronized to it (Jovanovic, 1971).

REMS has been called paradoxical sleep because an active brain is coupled with a more or less “paralyzed” body. Rapid eye movements occur in groups over the period of several records (up to 23 s) with variable intervals (200 ms to 23 s) between them. About 20–120 ms before EEG desynchronization during REMS, *ponto-geniculo-occipital waves* (PGOs) become active and remain visible throughout the entire REM phase. They have especially been studied in the cat and nonhuman primates, but their role in human sleep is not yet clear. They can be recorded from widespread areas in the brain including the pons, the lateral geniculate body, and the occipital cortex. The PGO waves of REMS can also be observed during the waking state and seem to be indicators of the activation of neural mechanisms indicative of orienting. They seem to originate in the dorsal pons near the midbrain and are generated by cholinergic neurons between the pons and the midbrain (McCarley, 1990).

REMS is also accompanied by brief phasic muscle activity (myocloni), whereas muscle tone ceases to exist (atonia). This atonia is related to hyperpolarization of  $\alpha$ -motoneurons, and the myocloni are related to descending excitatory bursts from the brainstem. In REMS presynaptic inhibition of group I primary afferents and thus suppression of monosynaptic reflexes is found in addition to descending inhibition of polysynaptic reflexes. Concurrent with sensory inhibition in the periphery, there is increased sensory activity in the brain (increased neuronal activity in the specific thalamic nuclei) which seems to be relevant for perceptual stability during REMS and the development of sensory structures in the growing organism (McCormick & Bal, 1997). During phasic REMS the threshold for waking up is highest, whereas in phases of paradoxical low-voltage, high-frequency sleep without REM there is slightly improved response. The ability to respond to internal stimuli decreases continuously from stage 1 to stage 4. During REMS, activity of the sympathetic nervous system is increased. Whereas the regulatory function of the hypothalamus continues during NREMS, it is diminished during REMS. Penile erection and vaginal lubrication during REMS can be used to differentiate organic from psychological factors in erectile dysfunction and lack of vaginal lubrication.

A look at the evolutionary aspects of sleep suggests that small and short-lived animals with a relatively high metabolic rate and a short BRAC sleep longer than mammals who spend a large part of the day searching for food. The duration of sleep seems to be inversely related to the length of the sleep cycle. Large animals with high predator vulnerability seem, however, to have less REMS. In younger people, SWS may constitute up to 40% of total sleep time. Older persons sleep less, wake up more often, show shorter REM phases and generally reduced SWS. Infants show maximum REM activity in the pre- and postnatal phase. REMS thus seems to be correlated with the maturation of the CNS: the high rate of REM in babies might compensate for the lack of structured sensory input from the not yet fully developed sensory system and activates the formation of synaptic connections.

#### **1.05.7.4 The Neurobiology of Sleep**

Multiple brain systems are involved in sleep regulation. Sleep-regulating structures seem to be located in the caudal region of the midbrain down to the medulla. SWS seems to be generated by the basal forebrain, the lateral preoptic region of the hypothalamus and the nucleus tractus solitarius which inhibits the activating reticular formation. Whereas the nucleus raphé seems to be involved more in the regulation of NREMS, the nucleus coeruleus seems to be important for REMS coordination. The raphé nucleus synthesizes serotonin, the nucleus coeruleus noradrenaline, and portions of the reticular formation acetylcholine. But SWS is not exclusively dependent on the nucleus raphé, nor is REMS dependent on the nucleus coeruleus. The dorsal serotonergic raphé nuclei lower their firing rate from the waking state to SWS, and during REMS they are completely silent. Nonadrenergic synapses from the nucleus coeruleus probably mediate the phasic activation of the dorsal serotonergic raphé nuclei to external and internal stimuli which might be related to waking up during SWS and REMS. During immobilization in various sleep stages, the dorsal raphé nuclei are inhibited. Immobilization is caused by the activity of nuclei in the pontine tegmentum that activate the nucleus magnocellularis of the reticular formation which in turn inhibits the spinal motoneurons.

Cholinergic reticular structures (reticular formation, pontine and midbrain tegmentum, posterior thalamus, and hypothalamus) are hyperactive during REMS. In contrast, it is assumed that during the very similar waking state, noradrenergic and serotonergic systems

are active while the cholinergic influence may be dampened. During REMS, the aminergic systems may be inhibited and the cholinergic systems are dominant. The state of consciousness depends on the reciprocal interaction of cholinergic and aminergic systems. During dreams, the visual input no longer originates in the retina but in the brain stem, which is coupled with reduced serotonergic and noradrenergic activity. The ensuing disinhibition of cholinergic neurons leads to PGO waves which create the subjective images of dreams (Hobson & Stickgold, 1995).

Endogenous sleep factors also seem to play a role in sleep generation. They include several substances such as factor S, sleep-promoting substance, 8-sleep-inducing peptide, and vasoactive peptide. Whereas some of these substances seem to induce sleep, others are produced and released during sleep.

#### **1.05.7.5 Psychophysiology of Sleep Stages**

The sleep stages are important regulatory physiological processes. Sleep during the first three hours of the night seems to have an important function for endocrine and immunological responses. Long-term deprivation leads to considerable consequences for the organism: REM and SWS episodes occur during the day, optic and acoustic illusions and hallucinations occur, and after four days paranoid ideation may be reported. The metabolic and behavioral changes are less severe. REM rebound usually occurs when selective REM deprivation is instituted; in general sleep deprivation REM is increased only after about four waking days. Barbiturates and some benzodiazepines suppress SWS and/or REM sleep (both are experienced as aversive), lead to REM rebound in dreaming or wakefulness nights, and therefore have an addictive potential. Dreams occur during REM and NREM sleep but seem to be experienced differently. Dream reports following REMS seem to be more vivid, more visual, and emotional than those experienced in NREMS where the dreams seem to be more abstract. Whereas dreams in the first half of the night seem to be more reality-oriented, those of the second half are more unusual and unreal.

Sleep seems to have an important function in the consolidation of learned material into long-term memory. REM deprivation leads to deficiencies in retrieval of learned material. This reduced consolidation seems to be related to reduced RNS synthesis when REM sleep is deprived. Hippocampal  $\theta$ , which occurs during the transition from short- to long-term memory,

is also present during REMS. In addition, elevated DNS concentrations in the brain have been found subsequent to learning and are also seen subsequent to REM episodes.

Borbely's (1984) theory of sleep and dream regulation suggests that two processes, *S* (SWS pressure) and *C* (REM pressure), are active at different points in time. Process *S* seems to depend on the duration of the waking state and increases in an exponential fashion. Process *C* is related to the reciprocal value of the temperature oscillator. The entire "sleep pressure" is related to the sum of *S* and *C*. This model makes exact predictions about the effects of sleep deprivation on depression, implies the existence of active sleep substances as the basis of *S*, and views the circadian oscillator of the nucleus suprachiasmaticus as the neuroanatomical basis of *C*.

### 1.05.7.6 Sleep Disorders

Although psychological variables are most important in most sleep disorders, biological factors contribute significantly to delayed sleep phase insomnia (DSPI), drug insomnia, sleep apnea, narcolepsy, somnambulism, and sleep disorders in depression. In DSPI persons go to bed late, but often need an hour or more to fall asleep and wake up very early. The cause of DSPI is probably a lack of flexibility of the SWS oscillator to alter the circadian period forward, that is, to induce sleep before the beginning of the circadian period. These patients can in general adapt their endogenous rhythm of 25 hours to a 24-hour cycle, but are unable to correct phase delays due to external factors. Chronotherapy has been used in these patients: based on the assumption of a *Zeitgeber* period of about 27 hours, the patients were instructed to delay sleep by three additional hours per day.

Drug insomnia is related to alterations in the sleep profile by barbiturates and benzodiazepines: REM sleep and SWS are reduced, stages 1 and 2 and  $\beta$ -activity are increased. Cessation of the hypnotic agents leads to REM rebound with nightmares, which are suppressed by renewed drug intake. Rebound insomnia occurs after three days of hypnotics intake with no effect on the original insomnia (Borbely, 1984).

Narcolepsy is characterized by frequent sleep attacks during the day that are related to loss of muscle tone and may cause sudden loss of posture of the person affected by them. They are characterized by the intrusion of REM episodes into the waking state. Narcoleptics enter immediately upon sleep onset into an REM phase which suggests that inhibition of REM-

activating structures is reduced. Nightmares with sleep paralysis (the inability to move the limbs) are usually REM episodes with extreme motor inhibition and concurrent awakening. Sleep is distorted in several psychiatric conditions such as depression and post-traumatic stress disorder. In *major depression*, REMS often appears very early and rapid eye movements may be more frequent. This has been related to enhanced cholinergic activity and/or a deficit in noradrenergic and serotonergic activity.

## 1.05.8 LEARNING, PLASTICITY, AND DISORDERS OF LEARNING AND MEMORY

### 1.05.8.1 Introduction

The simplest form of learning is *nonassociative learning* such as *habituation* and *sensitization*. These refer to a decrease or an increase in a behavioral response dependent on the number and intensity of stimulus presentations. Associative learning such as classical and instrumental conditioning involves the pairing of two stimuli or a response and a subsequent stimulus in a time-contingent manner. *Classical conditioning* is characterized by the (usually) frequent pairing of a neutral stimulus and a biologically relevant unconditioned stimulus (US) which elicits an unconditioned response (UR). After several trials, the formerly neutral, conditioned stimulus (CS) comes to elicit the conditioned response (CR) which may be agonistic or antagonistic to the UR. Extinction occurs when the CS is presented without the US: the CR slowly ceases to be. CRs may occur towards stimuli that are similar although not identical to the CS: this phenomenon is called *generalization*. Modern theories of learning (Rescorla, 1988) emphasize that stimulus associations rather than stimulus-response associations are learned: the signal value of a stimulus determines the speed of acquisition. In a latent inhibition design, for example, the CS is presented several times without the US before the onset of a conditioning phase. This retards the later acquisition of a CR to the CS. Imprinting is a special form of conditioning. It denotes a sensitivity for the acquisition of certain S-S or S-R connections in a certain developmental stage.

*Operant conditioning* is characterized by the learning of a response which is acquired by providing positive or negative reinforcers subsequent to its execution. Although operant and classical conditioning show similarities, it is questionable whether they have a common physiological basis. For example, instrumental learning of autonomic responses is difficult to

achieve, whereas classical conditioning of visceral responses is easily obtained and vice versa: classical conditioning of muscular responses needs more learning trials than operant conditioning.

#### 1.05.8.2 Mechanisms of Plasticity

Although memory and learning are closely related, memory research has for a long time focused on the encoding of abstract materials such as numbers and words rather than on the acquisition of skills. The specificity of a memory trace seems to be determined by (i) the location of the neuronal circuit underlying the memory and (ii) the anatomical and physiological characteristics of the cell assembly and the subsequent synaptic modifications. Plastic changes in the development of the organism are based on the branching and growth of dendrites and their dendritic spines. This growth is not random but occurs along "guiding" structures with common chemical affinity: specific chemical growth gradients of the glial cells probably guide axons and dendrite growth (Gazzaniga, 1995). The strong increase in weight of the brain in the first years of life is related to an increase in the number of synapses, myelination/enlargement of the cells, the dendrites, and dendritic spines, and an increase in glial cells and of the capillary net of the brain. Adequate sensory and motor or emotional stimulation is required for this development.

Sensory or motor deprivation lead to reduced growth and atrophy which is more severe and long lasting the more pervasive and earlier in development it occurred (Rosenzweig & Leimann, 1982). Enriched environmental conditions compared with impoverished environmental conditions have profound effects on dendritic growth and other markers of brain growth which are related to the long-term effects of behavioral learning (only active interaction with the environment causes these changes). Learning also occurs after completion of developmental processes and is probably mediated by sprouting as well as disinhibition of silent connection and changes at spines and their synaptic connections (Cotman & Nadler, 1978).

*Sprouting* or reactive synaptogenesis has been described as a consequence of nervous system lesions. It refers to the development of new synaptic connections and has been related to recovery of function. The activation of previously inhibited or silent synaptic connections may happen within minutes after a lesion. Recent animal work (Kaas, 1995) has shown that neuronal plasticity of the primary sensory

and motor areas is present in the adult organism. Both nervous system lesions as well as behavioral training alter the architecture of sensorimotor maps in the cortex. The functional significance of these cortical changes has recently also been demonstrated in humans. In human amputees, reorganization of the primary somatosensory cortex is highly positively correlated with the magnitude of phantom limb pain. In chronic back pain patients, chronicity of the pain and cortical reorganization show a high positive association. To what extent these reorganizational changes cause these pain phenomena needs to be determined (Birbaumer et al., 1995; Flor, Elbert, et al., 1995).

#### 1.05.8.3 The Formation of Memory Traces

*Engrams* refer to the electrochemical processes that underlie specific memory contents. Hebb (1949) proposed that reverberating *cell assemblies* are the basis of these memories. These reverberatory circuits are characterized by interconnected excitatory synapses that maintain the neuronal activation. A cell assembly is usually formed by several of these reverberatory circuits that must exceed a certain level of activation for a cell assembly to "fire." The activity in reverberatory circuits may be viewed as the neurophysiological basis of the consolidation of memory traces. Continuing reverberation leads to structural synaptic and cellular changes that represent long-term memory. A certain time of undisturbed reverberatory activity is needed for consolidation, otherwise interference with memory formation occurs. Weak synaptic connections between neurons increase in strength during the consolidation phase if neurons are activated in close temporal proximity or simultaneously in an "associative" manner. These temporally closely coordinated synaptic activations are the basis of conditioning and learning. Once the synaptic connection has been formed, activation of a part of the cell assembly will activate the entire cell assembly. High-frequency synchronous oscillations in the  $\gamma$ -band range seem to be an expression of the mechanism of formation of cell assemblies (Singer & Gray, 1995).

A fundamental principle of neuronal plasticity is based on the Hebb rule (1949): if the axon of neuron A excites neuron B and causes repeatedly or constantly the firing of neuron B (i.e., the suprathreshold excitation of neuron B), then the efficacy of neuron A in exciting neuron B increases either through a growth process or a metabolic change in one or both neurons. Realization of the Hebb rule usually depends on

two presynaptic (synapses 1 and 2) and one postsynaptic cell. Assume that synapse 1 is excited by a neutral tone which by itself is not sufficient to excite the postsynaptic cell on which converge synapses 1 and 2. Synapse 2 (which may, for example, be excited by a tactile receptor in the eye) is excited together or shortly after synapse 1 by an air puff to the eye which elicits activation of the blink reflex in the postsynaptic cell. Firing of the postsynaptic cell (caused by synapse 2) now reinforces the activity of all synapses that were simultaneously active at the postsynaptic cell, thus it also increases the excitability of the weak synapse 1. After several pairings of the two stimuli, the tone alone will excite the postsynaptic cell sufficiently to cause it to fire and will thus elicit the blink reflex. Thus, classical conditioning of the blink reflex has been established.

According to Hebb's theory, the excitatory configuration must reverberate in the same cell assembly for some time before structural long-term memory changes occur. The information the CNS needs for memory storage is not the activity of one or a few cells, but the *concurrent* coherent firing pattern of an entire cell assembly. Coherence is formed by the simultaneous firing of an excitatory pattern in the cell assembly where structure and frequency are specific for a memory content. A measure of the coherence is the amplitude of the evoked potential of the EEG or the evoked field of the MEG above a certain cell assembly or the cross-correlation function of the various firing patterns between several cell assemblies. The higher the amplitude of the evoked potential, the more cells must fire synchronously, the more complex the learned materials, the larger the cell assemblies involved, and the longer the time the reverberatory activity must be active in order to attain a critical shift of metabolic activity in the cells.

Every cortical cell is connected to about 4000–10000 other cortical cells. If there were only fixed connections for the representation of memories, then we would quickly run out of connections to store all memory traces. Since one cell can participate in the representation of many different contents, that is, participate in many different cell assemblies, we have a nearly unlimited number of combinations of functional connections that build the basis of our implicit learning capabilities. This summation of synchronous input to an assembly is necessary because the convergence of only a few synapses would not reach the critical firing threshold. If information is connected its individual elements appear in temporal synchrony and thus allow for the formation of Hebb synapses. The larger the assembly and the more complex the context, the slower the reverberatory activity in the cell

assembly. Slow EEG oscillations in the frequency band of 0–15 Hz depend primarily on thalamocortical excitatory circuits and their nonspecific input from reticular subcortical structures. They determine the excitatory thresholds of cortical tissue in the waking and sleep state. Since most of our memories are cortical, cortico-cortical assemblies are necessary for their formation. Calculation of conduction velocities in cortical assemblies suggests that optimal distances (also for widely separated cell assemblies) of two synchronous depolarizations are in the vicinity of 20–60 Hz. It is just the oscillatory frequency in the  $\gamma$ -band range which may be detected in the EEG and MEG when *gestalts*, meanings and words or dreams are perceived. They also appear when new concepts are formed during learning.

#### 1.05.8.4 Neuropsychology of Memory

In 1880, the Russian neurologist Korsakoff first described the *amnesic syndrome* which is characterized by anterograde amnesia, retrograde amnesia, and confabulation. The neurophysiological basis of this syndrome is a destruction of diencephalic and limbic structures and often also the mamillary bodies and the dorsomedial nucleus of the thalamus (for example in alcoholism, by lack of thiamine). The main lesion in Korsakoff's syndrome is, however, located in the hippocampus. The lesions in Korsakoff's syndrome are too diffuse to allow for exact determination of the relevance of temporal structures for memory. Patients with surgical removal of the temporal lobe (e.g., due to epilepsy, as in the famous case H.M.) make more exact examinations possible. The data from these patients suggest that the medial temporal lobe system is essential for declarative memory.

The *medial temporal lobe system* plays an important role in *declarative learning*. The hippocampus receives information from all association areas of the neocortex via the entorhinal cortex as well as from limbic areas, especially the cingulate gyrus, the orbitofrontal cortex, and various regions of the temporal cortex. All these connections are reciprocal in that the hippocampus also has efferent connections to the association cortices where the actual long-term changes of memory storage must take place. The medial-temporal lobe system must be active during the presentation and repetition of memory-related materials so that associative connections can be formed between the various stimuli that are present during encoding. The hippocampus and the adjacent entorhinal cortex connect the various representations of

the entire environment that is present during learning with respect to temporal and spatial aspects. Creation of such a context is especially necessary when new situations and new learning materials have to be encoded since new perceptions and new thoughts that have so far not been associatively connected have to be associated. As soon as these contents are associatively learned, a small aspect or a part of the situation is sufficient to reproduce the entire situation. The hippocampal system thus connects the cortical representations of certain situations with each other so that they build one general memory content ("binding").

Whereas declarative learning is heavily dependent on an intact medial-temporal lobe system, nondeclarative learning does not need an intact medial-temporal lobe. General cortical processes are less important for the formation of nondeclarative memories although the acquisition of habits and motor skills involves the motor and prefrontal cortical areas of the cortex. Specific subcortical regions have been identified for specific nondeclarative or implicit learning processes. For example, the already described classical conditioning of the nictating membrane of the eye in the rabbit is dependent on an intact cerebellum and the conditioned emotional responses to aversive stimuli in the rat are dependent on an intact amygdala. In general, the learning process takes place in the location where the two sensory informations that are to be associated meet. For example, if the conditioned stimulus is a tone and the unconditioned stimulus an aversive tactile stimulus, the associative connection in the rat is formed in the medial section of the medial geniculate nucleus where both channels of information converge.

#### 1.05.8.5 Cellular Mechanisms of Learning

The cellular basis of learning has been primarily studied in simple organisms such as the sea snail *Aplysia* whose nervous system consists only of 20 000 neurons, and the common fruit fly *Drosophila melanogaster*. Sensitization, habituation, and operant and respondent (Pavlovian) conditioning have been described.

Associative learning in complex organisms may follow similar principles as learning in simple organisms such as *Aplysia*. Since the 1970s it has been shown that habituation, sensitization, and classical conditioning are all related to specific pre- and postsynaptic modifications in neuronal systems with few cellular connections. In *Aplysia*, stimulation of the siphon or the mantle shelf leads to contraction of the siphon, the mantle shelf, and the gill

(unconditioned defensive reflex). About 10 stimulations of the siphon lead to habituation of the defensive reflex. This response reduction is related to the reduction of transmitter release in the sensory neuron. Every new action potential reduces the  $\text{Ca}^{2+}$  influx into the sensory synapse. Long-term habituation (weeks, months) is related to a reduction in the number of vesicles that release transmitter in the synapse. When sensitization occurs, increased transmitter release of sensory interneurons at the motor neuron occurs (Hawkins & Kandel, 1984).

Classical conditioning in *Aplysia* entails the following processes:

(i) Stimulation of the tail activates a group of facilitator neurons that lead to transmitter release at the synapses of the sensory neuron (presynaptic facilitation).

(ii) The transmitter (serotonin) activates the enzyme adenylyl cyclase which increases cyclic AMP in the synapses of the sensory neurons.

(iii) Increase of cAMP activates a second enzyme, a cAMP-dependent protein kinase.

(iv) The protein kinase closes a specific type of  $\text{K}^{+}$  channel in the membrane and reduces the number of open  $\text{K}^{+}$  channels during the action potential. The protein kinase also activates the synaptic vesicles.

(v) The decrease of the  $\text{K}^{+}$  outflow leads to a broadening, that is, a temporal extension of the next incoming action potential, which causes more  $\text{Ca}^{2+}$  to enter the synaptic terminal and more transmitter release since the binding of the transmitter to  $\text{Ca}^{2+}$  is a prerequisite of its release.

(vi) The increased  $\text{Ca}^{2+}$  binds partially to calmodulin which attaches to the adenylyl cyclase and increases the potential for cAMP production.

In the cortex and hippocampus where glutamate is the excitatory transmitter, NMDA receptors most likely initiate these processes (Linden & Connor, 1995). In hippocampal and cortical dendrites, *long-term potentiation* (LTP) has been described as a potential physiological mechanism of the encoding and retrieval of information in short-term memory. *Homosynaptic LTP* occurs when a cell is stimulated for about 1 s at a 100 Hz frequency. The cell increases its firing frequency and also its synaptic strength when a test stimulus is later presented. The incoming stimuli lead to depolarization and  $\text{Ca}^{2+}$  influx into the cell because the ensuing  $\text{Mg}^{2+}$  blockade opens the NMDA (or AMPA) receptor.

*Associative LTP* occurs when slow stimulation (5 Hz) of an axon is coupled with high frequency activation of an adjacent axon. The connection of the slow-frequency stimulated axon synapse with the postsynaptic membrane



leads to a long-lasting increase in excitation and a strengthening of the synaptic connection upon renewed stimulation. *Long-term depression* (LTD) protects the synapse from extreme (epileptiform) LTPs and facilitates the reduction of LTP, thus increasing the possibility for renewed excitation. In its *heterosynaptic form*, one synapse is stimulated tetanically while the adjacent synapse is not preactivated. *Associative LTD* occurs during asynchronous (anti-Hebb) stimulation of two synapses. Low-frequency stimulation (1 Hz for 10 min) leads to *homosynaptic LTD*.

LTD is probably the mechanism responsible for forgetting and affects lateral inhibition in LTP. Whereas LTP leads to increased intracellular  $\text{Ca}^{2+}$ , the activation of protein kinase (G-protein), and phosphorylation of proteins in the cell and subsequent gene expression with the synthesis of new proteins, LTD leads to dephosphorylation of the receptors and reduced conductivity.

#### 1.05.8.6 Neuronal Bases of Learning and Memory

Simple classical conditioning does not require an intact cortex. Thompson and Krupa (1994) showed the development of an engram in the hippocampus and the nucleus interpositus of the cerebellum during classical eyeblink conditioning in the rat, rabbit, and cat. In the rabbit, closure of the nictating membrane of the eye was used as UR, a tone as CS, and an air puff to the eye as US. Cells in the CA1 layer of the hippocampus become active in the CS-US interval several trials prior to the expression of the CR nictating membrane closure to the tone. The mean firing frequency of these cells exactly predicts the form of the later CR. A similar firing frequency was found in cells of the nucleus interpositus of the cerebellum from which efferents of the cerebellum to other brain regions originate. If these cells are destroyed, the CR is abolished and a new CR (but not the UR) can no longer be acquired. The learning mechanism is the previously described LTD of Purkinje cells in the cerebellar cortex. The tone CS is transported via the auditory tract to the cerebellar cortex and the nucleus interpositus, and the air puff US reaches these structures about 100–300 ms later via the nucleus trigeminus and the olive. The motor UR and CR are relayed from the nucleus interpositus (which is inhibited from the cerebellar cortex and has an excitatory connection to it) to the motor nuclei of the brainstem. Learning occurs in the plastic Purkinje cells: LTD is initiated by the shortly temporally spaced firing of the parallel mossy fibers (CS) and the climbing fibers (US) at the

synapses of the parallel fibers of the Purkinje cells which have AMPA receptors. Thus, cerebellar cortical LTD disinhibits the nucleus interpositus (which is usually inhibited by the cerebellar cortex) and the CS can elicit the CR. This engram is thus stored in the cerebellar cortex, not the afferent or efferent structures.

In humans, destruction of the cerebellar cortex likewise leads to disruption or elimination of classical eye lid conditioning, with intact responses to the CS and US (Daum et al., 1993). The cerebellum is, however, only involved in motor learning. The learning of emotional responses or declarative learning are unaffected by cerebellar lesions.

#### 1.05.8.7 Neurochemistry of Memory

The biosynthesis of proteins seems to play an important role in transferring information from short-term into long-term memory. It is unlikely that qualitative changes in the amino acid sequences of the RNS are the underlying mechanism for long-term storage, since these changes only seem to reflect unspecific activation or stress (Davis & Roitblat, 1984). Alterations in gene expression may be viewed as the basis of learning-induced physiological changes.

Learning and other environmental influences modify the binding of proteins that activate transcription of the genetic code via regulatory proteins. Every gene has three regions: a code region which is transcribed by mRNS and translated into a specific protein, and a regulatory region which consists of the promoter region, and an enhancer region which contains binding locations for regulatory proteins which ensure tissue-specific gene expression for every specific type of cell. The promoter region consists of a brief sequence of nucleotides which must first be activated by a regulatory protein before RNS-polymerase can transcribe the structural gene. Phosphorylation of the regulatory proteins is essential for their function as a key in the lock of the regulatory region.

Various antibiotics inhibit cerebral protein biosynthesis during the translation of tRNS into the respective amino acids at the ribosome. About 80–90% of cerebral protein synthesis can be blocked temporarily without severe deficits in behaviors other than memory. Encoding and the input of information is not disturbed if the training time does not overlap too much with the time it takes for antibiotics to exert their effect. Even weeks after the completion of training, the retrieval of memory contents is interfered with. The strongest amnesia occurs when protein synthesis is inhibited shortly before the beginning of training, that is, if protein synthesis is

disturbed during training. The retrieval of memory contents is not influenced by protein biosynthesis inhibition because injections of the respective antibiotics at the time of retrieval does not show an effect on learned materials. This means that the protein biosynthesis is only needed for a critical phase of consolidation during and after training. Interestingly, short-term memory is not affected by the inhibition of protein biosynthesis.

Both long-term potentiation and long-term depression lead to a modification of early and late gene expression. Excitation as well as damage to nerve axons (e.g., after deafferentation during amputation) leads to long-lasting LTP or LTD (based on the cell type). Subsequent to injury, hyperexcitability, synaptic facilitation, and growth occur in the deafferented neurons. These may occur within hours and explain the quick and lasting chronic pain that may occur subsequent to amputation. Although this is not an associative learning mechanism, the molecular changes are probably similar to those of associative learning. There is a critical time period during which the content of a memory can be moved from short-term to long-term memory. This critical time period is determined by the duration of the change in gene expression. After LTP, the so-called immediate early genes are altered and subsequently late genes are activated that lead to a permanent synaptic modification by transport of the newly synthesized gene products from the nucleus to the cell membrane. This could be the structural basis of long-term memory.

Except for protein biosynthesis, no single substance or class of substances has been shown to have a causal influence on memory. This suggests that memory can be coded in a number of synaptic and cellular processes that all lead to the same final result: long-term changes in the firing rate of a cell assembly. Whereas central catecholamines seem to have a minor role in learning and memory, peripheral catecholamines seem to play an important role in the consolidation of memories. For example, lesions of the medulla of the adrenal gland lead to a severe reduction of peripheral noradrenaline and concurrently to severe amnesias. The activation of  $\beta$ -adrenergic receptors on the central or peripheral level by emotional stimuli seems to have a strong influence on retrieval from long-term memory. If the  $\beta$ -adrenergic receptors are blocked (e.g., by propranolol), retrieval of emotional but not neutral events from memory is severely disturbed at a later point in time (Cahill, Prins, Weber, & McGaugh, 1994).

Acetylcholine seems to play an important role in learning. There are several cholinergic

systems in the mammalian brain and precursors of acetylcholine and metabolites participate in almost all behaviors directly (as transmitters) or indirectly (as neuromodulators). It is therefore especially difficult to show the role of cholinergic systems in associative learning. Scopolamine, which blocks most cholinergic receptors, leads to memory loss for birth incidents if it is given to mothers during delivery. It has sometimes been reported that postsynaptic cholinergic stimulators improve retrieval from memory in patients with Alzheimer's disease. In patients with morbus Alzheimer's disease where neuronal aging processes seem to occur at a very rapid rate, reduced acetylcholine is found, along with a lower density of muscarine receptors and a lower number of cholinergic neurons. The low efficacy of cholinergic stimulation alone suggests that cholinergic and noradrenergic systems have to converge in layers 1 and 2 of the cortex in order to make normal memory and consolidation possible, although they may not be involved in the encoding and storage process. In Alzheimer patients, degeneration of nucleus coeruleus cells has been observed. In animal experiments long-lasting stimulation of the nucleus coeruleus in aging mice led to an inhibition of the forgetting of a shock avoidance response, which would otherwise occur because of the aging process. These results suggest that memory processes depend on the interplay of several transmitters in specific brain regions, primarily in the cortex, hippocampus, and limbic regions.

Just like the neurotransmitters, centrally as well as peripherally injected peptides modulate memory functions. They all seem to have an indirect influence; none of the peptides that have been examined so far are necessary preconditions for learning and memory processes: vasopressin, ACTH, and somatostatin enhance learning and encoding, endorphines and oxytocine have negative influences. Angiotensin, cholecystokinin, and substance P interfere with encoding if they are directly injected into the central nervous system.

Opioid peptides and low doses of morphine-containing substances lead to forgetfulness; naloxone and opioid antagonists improve encoding in various tasks. This memory-enhancing effect may be related to the inhibition of catecholaminergic receptors by the opioids and the blockade of the facilitatory effect of noradrenaline, dopamine, and acetylcholine. Naloxone also prevents retrograde amnesia related to electric shock which is accompanied by a massive output of beta-endorphine and methionin-enkephaline.

The high concentration of glutamate in the hippocampus and neocortex suggests that this

amino acid has an important role in consolidation processes. It is not clear if the increase of glutamate receptors and the correlating growth of dendritic spines following long-term potentiation of the hippocampus plays a causal and specific role for memory processes. The use of glutamate to increase learning and memory has so far not shown consistent results. Although Alzheimer's disease is related to a marked reduction of CNS glutamate (60%), treatment efforts that influence glutamate metabolism have so far not shown consistent results.

## 1.05.9 MOTIVATION AND MOTIVATION-RELATED DISORDERS

### 1.05.9.1 Introduction

The term motivation is used to describe an *internal state of the organism* that modulates the frequency and intensity of behavioral responses. Motivational states may be based on *innate* drives or may be induced by *acquired* drives. Some drives follow *homeostatic* principles: they are less dependent on environmental influences and the learning history of the individual but are rather elicited by deviations from the internal homeostasis of the body (e.g., hunger, thirst). They show stable levels of attainment that motivate a stereotypical sequence of behaviors if they are exceeded or not attained. Non-homeostatic drives (such as sexuality, exploration) show variable levels of attainment and deprivation times that are highly determined by learning processes and other environmental variables (such as availability, incentive). Behavior is determined by *drives* as well as *reinforcement*. Whereas drives provide the "energy" for a certain behavior, stimuli that increase the probability of the occurrence of a response are termed reinforcers, and stimuli that reduce the probability of the occurrence of a response are termed punishing stimuli. Reinforcement is the increase in response probability in the presence of a discriminative situation by the delivery of reinforcing stimuli subsequent to a response. Drives and reinforcements are based on different neuronal substrates.

In addition to drive reduction, incentive motivation plays an important role in the instigation of goal-directed behavior: after some reinforcing trials not only the drive but also the reinforcement will move the behavior in a specific direction. *Incentive motivation* is determined by (i) the neuronal substrate for reinforcement that is activated by behavioral consequences, (ii) the emotion related to reinforcement which is associated with reinforcement-related stimuli via classical con-

ditioning, and (iii) incentive salience, objects and behaviors that were associated with reinforcement become more prominent ("salient") than other stimuli, capture more attention, and motivate approach or avoidance behavior.

### 1.05.9.2 Hunger and the Eating Disorders

Hunger is one of the homeostatic drives. Under physiological conditions, a reduction in blood glucose concentration serves as signal for hunger. The glucosensors are located in hypothalamus, brainstem, and liver. Upon the ingestion of food, hunger ceases fairly rapidly (*short-term presorptive satiety*), whereas the ingestion of food is terminated only when the blood glucose levels have again reached their predetermined level (*long-term resorptive satiety*). Classical conditioning usually plays an important role in motivating food intake. Social and environmental stimuli such as dinner time, taste and appearance of food, or persons present at dinner determine the time and the amount of food ingestion much more than physiological factors. Taste and odor cues serve an important function in the selection and ingestion of food (e.g., taste cues increase appetite despite complete reduction of hunger (Carlson, 1991; Legg & Booth, 1984). In addition to *glucostatic* factors, *thermostatic* (food intake proportional to changes in temperature) and *lipostatic* mechanisms (food intake proportional to the metabolism of lipoproteins) have been discussed (Carlson, 1991).

Presorptive satiety is determined by the activation of odor, taste, and mechanoreceptors in the otolaryngeal cavity and trachea as well as by chewing. Additional factors are pressure-sensitive receptors in the stomach and the colon as well as chemoreceptive vagal afferents from the stomach and upper colon that respond to the glucose and amino acid content of food. Resorptive satiety is determined by chemoreceptors of the digestive system as well as all enteroreceptive sensory processes related to hunger feelings. The enhanced glucose availability, enhanced heat production as well as changes in lipid metabolism, activate central receptors that signal satiety. Gastrointestinal hormones are also important in the generation of long-term satiety. For example, cholecystokinin, a neuropeptide, leads to satiety probably mediated via cholecystokinin receptors in the ventromedial hypothalamus. Based on the glucostatic model of hunger and satiety, the activity of the lateral hypothalamus (LH) is viewed as a signal for hunger and the ventromedial hypothalamus (VMH) is viewed as important for satiety. Both centers are

assumed to be reciprocally inhibitory. The lateral hypothalamus seems to serve the function of a glucostat and inducer of hunger. An increase in glucose levels inhibits LH activity, activates VMH, and initiates satiety. Destruction of the VMH leads to overeating and obesity. The VMH and its connections to the paraventricular nuclei of the hypothalamus and the efferent vagal nuclei as well as to the nucleus tractus solitarius seem to code taste as nutritionally adaptive and maladaptive. The limbic system and associated cortical structures as well as the motor system are also involved in the planning of food intake, probably via catecholaminergic fiber systems that connect the brain stem, cerebellum, basal ganglia, and cortex. The search for food is probably instigated by neurons in the lateral hypothalamus that are connected to motor regions by dopaminergic fiber bundles.

Eating disorders such as anorexia or bulimia are primarily caused by cultural and psychological variables (see Volume 6). Biological variables are, however, important in the course and maintenance of these disorders since dieting is followed by serious biological sequelae. Ongoing dieting leads to major disruptions of the endocrine system, especially the pituitary–adreno–cortical axis. The regulation of sexual and reproductive functions is disturbed. Reversible reductions of brain mass have also occasionally been reported which are associated with negative consequences such as psychological disorders and ongoing weight problems by 30% of patients. Biological and hereditary factors of metabolic rate seem to be much more important in obesity than in other eating disorders. However, dieting also has negative consequences in the case of obesity where the lost weight is regained and maintained at a higher than prediet level (Carlson, 1991), referred to as “cycling.”

### 1.05.9.3 Sexual Function and the Sexual Disorders

#### 1.05.9.3.1 *The sexual response*

Four phases of sexual activity can be differentiated: sexual attraction, appetitive behavior, copulatory behavior, and postcopulatory behavior. They have to be synchronized during sexual activity between the involved partners and they are based on clearly differentiable neuronal and hormonal mechanisms.

Sexual attraction and all other phases of sexual behavior are influenced by the *androgen* levels of the male and the *estrogen* levels of the female in most species. Odors, posture, and color changes contribute to sexual attraction.

Appetitive behavior involves “invitation” to approach and to mount, changes in posture, erection, and the emission of sounds. Appetitive behavior is disorganized when the cortex is removed, whereas copulatory behavior (intromission, orgasm) does not depend on the neocortex. Orgasm involves ejaculation (male) and contractions of the pelvic muscles and vagina (female) or the penis and the pelvic musculature (male).

In humans, the sexual responses of both sexes are fairly similar. Women usually need a longer appetitive (plateau) phase to attain orgasm and most men have an absolute postcopulatory (refractory) period: subsequent erection and ejaculation is only possible after a certain recreational phase.

The sexual response of the male is initiated by erection which is caused by dilatation of arteries to and in the corpora cavernosa, the corpus spongiosum urethrae, and the sinusoids of the corpora cavernosa via the activation of postganglionic parasympathetic neurons. These neurons are activated by afferents from the penis and surrounding tissue and, in addition, by supraspinal (cortical) structures via psychological mechanisms. In addition to vasodilation, the venous reflex from the corpora cavernosa is reduced by venous constriction; both mechanisms lead to vasocongestion. The sacral spinal cord is the locus of the erectile reflex. Strong activation of afferents from the sexual organs leads to the activation of sympathetic afferents in the lower thoracic and lumbar spine. This sympathetic activation then provokes the release of semen and fluid into the internal urethra. Following this emission, ejaculation is initiated by afferent activation from the prostate and the internal urethra in the pelvic nerves. Both sympathetic and parasympathetic neurons to the sexual organs show maximum activation during ejaculation. The sympathetic afferents cause tonic–clonic contractions of the pelvic musculature and erectile tissue, causing fluids and semen to be expelled via the external urethra.

The female sexual response is characterized by a venous blood occlusion and vascular dilation of the labia majora and minora. Glans and corpus clitoris swell and increase in size and length due to parasympathetically induced vasocongestion. Just as in the male, both afferents from the genital region (especially the clitoris) and supraspinal inputs lead to these changes. Within 6–30 s after stimulation (afferent or supraspinal), lubrication of the vagina ensues in sexually mature females and formation of an “orgiastic cuff” in the outer third of the vagina. The “orgiastic cuff” contracts during orgasm and this activity can be compared to

emission and ejaculation in the male. The uterus changes to an erect and enlarged position and contracts during orgasm.

Some persons with complete spinal cord lesions show erection, lubrication, and orgasm when the sexual organs are stimulated but all sexual sensation from the genital region is absent. These patients report, however, “phantom sensations” that are independent of peripheral stimulation.

#### 1.05.9.3.2 *Sexual differentiation and sex hormones*

The development of a male or female organism is determined by the male reproductive cells in sperms: if they contain a Y chromosome the organism will be male, if they contain an X chromosome, the organism will be female (all ova have X chromosomes). Up to the eighth week of pregnancy the organism is dimorphic; only after this period the sex hormones determine if the organism will have male or female sex organs. In humans, weeks 12–22 of pregnancy and the first six weeks after birth are periods that are sensitive for androgens. During prenatal development, the androgens also act on the CNS and create the gender-specific differences in the hypothalamus and the limbic system, among others. This androgen action on the brain also seems to determine later sexual preferences (hetero-homo-bisexualism).

If an XY chromosome is present, precursors of the testes form in the seventh and eighth week which produce androgens (e.g., testosterone) that are essential for the formation of a male organism. If androgens are lacking, a female organism will develop. Secondary gender characteristics (such as a beard in the male, or breasts in the female) develop in puberty when the hypothalamus begins to produce precursors of the gonadotropic hormones that excite the release of estrogens and testosterone via luteinizing and follicle stimulating hormones of the pituitary gland. These secondary changes can be partially altered in later life if sex hormones are withdrawn or added. *Hermaphroditism* denotes a disorder where the internal or external sexual organs are malformed and consequently no clear gender assignment can be made.

#### 1.05.9.3.3 *Hormonal basis of sexual behavior*

Regulation of the sex hormones depends on the activity of hypothalamic cells that excrete peptides into the local circulation and through neural pathways or via the pituitary gland into the systemic circulation. These peptides either act as hormones and bind to receptors often

very distant from the hypothalamus or they act like transmitters at the synaptic cleft where they exert a tonic modulating influence on neuronal excitability. All sex hormones are regulated by luteinizing hormone releasing hormone (LHRL), also called gonadotropin releasing hormone (GnRH), which is excreted by several hypothalamic and extrahypothalamic cell systems. LHRH stimulates the excretion of luteinizing hormone (LH) and follicle stimulating hormone (FSH) from the anterior pituitary. Whereas dopamine and serotonin have an inhibitory influence, noradrenaline has an excitatory influence on the LHRL cells in hypothalamus. Estradiol and progesterone reaching the CNS via the blood system increase and testosterone decreases its release.

LHRH is bound to the cells that release LH, FSH, and prolactin. Only *rhythmic pulsative* LHRH activity will lead to hormone release (every 3–4 h except for the first phase of the menstrual cycle, where women release hormones every 90 m). LH and FSH stimulate the growth and transformation of the follicle in the ovaries and the production of estrogen; in the male they maintain spermatogenesis and the production of testosterone. In the male organism, they are released at a constant level: testosterone inhibits LHRH (negative feedback). Environmental stimuli (e.g., anticipation of sexual activity) influence FSH and LH release.

In the female organism, LH and FSH are released in a cyclic pattern: increase of estrogen leads to increase of LH and FSH release in the first phase of the menstrual cycle. When the follicle grows in the first days of the cycle, estrogen and progesterone slowly increase, on day 12 on average LH production steeply increases (because of a positive feedback circle of estradiol on LH and FSH). This burst causes ovulation (about 24 h later). The follicle then releases the ovum and the remaining follicle cells increase progesterone synthesis. The follicle turns into the corpus luteum and releases more progesterone and estradiol. This positive feedback circle is followed by negative feedback: the increase of progesterone leads to LH and FSH inhibition, estrogen and progesterone release is terminated around day 24 when LH and FSH release decrease further. Menstruation follows when the mucosa of the uterus loses vitality owing to progesterone reduction.

The influence of the sex hormones on behavior is less powerful in humans than in other mammals. Appetitive and copulatory behavior is maintained after ovariectomy and castration if prior learning of sexual behaviors has taken place. Castration after puberty has only delayed effects on sexual activity (slow decline) and is reduced if prior sexual experience

is present. Contraceptives that suppress the rhythm of hypothalamic, pituitary, and ovarian hormones do not influence appetitive and copulatory behavior, whereas testosterone levels have a stronger influence on sexual appetite in the female than estrogens.

*Amenorrhea* (cessation of menstruation) is often related to psychological factors that suppress LH increase but may also be related to physical factors (e.g., lack of nourishment in anorexia) or diseases such as tumors of the pituitary. Intake of LHRH suppresses LH and FSH release and induces reversible castration in the male (e.g., for treatment of tumors of the prostate or in sexual delinquency). In the male, the normal level of available testosterone is usually unrelated to sexual behavior. However, if a certain level of testosterone ( $\geq 350$  ng/l) is lacking, impotence may occur and may be corrected by substituting testosterone. Helplessness and depression reduce, and anticipation of sexual activity increases, testosterone production.

*Pheromones*—substances that are secreted by an organism and influence the other organism's behavior via the sense of smell—have important consequences on partner selection, sexual behavior, and pregnancy in rodents. Their role in human sexual behavior is less clear.

#### 1.05.9.3.4 *Neuronal mechanisms of sexual behavior*

Most research on neuronal mechanisms of sexual behavior is based on research in rodents. The applicability of these findings to human sexual behavior is questionable. Within the same organism two different neuronal networks for male and female sexual behavior exist: this *sexual dimorphism* seems to be a universal characteristic of all organisms. The apparent behavior of the organism results from the inhibition and/or excitation of the respective networks. Thus both sexes are “represented” in the brain (Swaab & Hofman, 1995).

In both sexes, the hypothalamus is the central regulating structure controlling sexual behavior in concert with sensory and motor reflexes in the genitals, the autonomic extraspinal ganglia and fibers, and spinal reflexes. Cortex and limbic system exert a modulating influence, primarily on the hypothalamus. The hypothalamus functions as both a neuronal control center and secretory organ. The limbic system, the hypothalamus, and their most important afferents and efferents are all structures with a high content of sex hormones. This system has also been referred to as the *paracrine heart of the neuraxis* since it has a central function in the regulation of homeostatic and nonhomeostatic

drives, excitability of moto neurons, the fight-flight response, reproduction, aggression, and territoriality. The anterior hypothalamus has been identified as the region that integrates the various sex-related reflexes into a goal-oriented behavior pattern.

In the male, cutaneous mechanoreceptors of the penis have an important role in the induction of copulatory behavior. If both destruction of olfaction and social isolation are present, male sexual behavior is suppressed in the rat. In the monkey, the medial preoptic region is essential for copulatory behavior. Additional important modulators of male sexual behavior are the frontal and somatosensory cortex and the basolateral nucleus of the amygdala. The so-called *Klüver–Bucy syndrome* in the monkey (tamelessness and hypersexuality) which occurs after removal of the temporal lobe and the amygdala is probably related to the loss of inhibitory input of the amygdala on the medial preoptic region of the hypothalamus.

Female sexual behavior is likewise coordinated by hypothalamic structures in concert with lower reflexes: the medial preoptic region of the hypothalamus inhibits and the ventromedial nucleus facilitates the lordosis reflex in the female rat. The hypothalamic efferents and the somatosensory afferents converge in the central gray of the midbrain and the dorsolateral reticular formation. The basic hormonal level (primarily estrogens) determines to a large extent the strength of the hypothalamic influence. It thus has a *priming function* on the entire chain of behavior, whereas the individual motor and vegetative elements depend on spinal reflexes.

#### 1.05.9.3.5 *Influence of sex hormones on the brain*

Androgen- and estrogen-sensitive cell systems have been identified in the hypothalamus and the limbic system. Their growth depends on the amount of peripherally available hormone levels. Axons and dendrites of these cells are modified by androgens and this leads to anatomical differences of these structures in men and women. This might be related to the often reported advantages of verbal vs. spatial abilities in the female vs. male sex and the increased plasticity with respect to verbal defects in the female. Creative musical talent has been associated with profiles of sex hormones that resemble those of the opposite sex. Despite these prenatal sex hormone influences on gender differences, cultural and social factors are paramount.

The interaction of hormonal influences and sexual behavior in humans is best studied by the

analysis of disorders in the endocrine glands that occur pre- or postnatally. The *androgenital syndrome* is based on excessive release of male hormones in the fetal stage of development. As a consequence, the external female organs become male (penis but not testes develops) even if the organism is chromosomally female, based on the sex chromosomes, and has internal female organs. These genetically female children have been raised as males and have developed attraction to females in the postpubertal phase because of brain masculinization. In some cases (especially if the external genitals were not well formed) these women were treated postnatally to reduce virilization and their external genitals were surgically corrected; their later sexual orientation remained, however, "male": 48% were reported to be bisexual and 17% homosexual (compared with 2–5% among females in general). Prenatal masculinization of the brain is obviously sufficient to induce lifelong changes in sexual orientation.

In the *androgen-insensitivity syndrome*, a genetically determined insensitivity of the androgen receptors at the androgen effector cells, leads to external female sexual organs in a genetic male. These children are usually raised as females and reportedly show average heterosexual behaviors (i.e., they prefer males as sex partners). In *male hermaphroditism* a lack of androgen production in a genetic male leads to undefined external sexual organs. These children are, therefore, often raised as males or females, independent of their genetic gender. If raised as girls, these genetic males accept men as sexual partners.

In *5- $\alpha$ -reductase-deficit disorder*, a genetic defect leads to lack of the enzyme 5- $\alpha$ -reductase which transforms testosterone to dihydrotestosterone, which is responsible for development of the external male sexual characteristics. This defect does not, however, influence brain development. Thus, a "male brain" is found in a "female body" in a chromosomally male organism. In the first generation, the children were raised as female, when it became clear that masculinization set in during puberty, the next generation of children was raised as males with less behavior problems than the first generation "macho" and often lesbian females (Money, 1987). Thus, brain development seems to have a more substantial influence on sexual orientation than environmental influences and reinforcements of sexual behavior.

#### 1.05.9.3.6 Brain processes and homosexuality

Homosexual behavior seems to be primarily dependent on hormonal changes in the brain. This statement refers to primary (exclusive)

homosexuality which seems to have a prevalence of about 5% in men and 2–3% in females across cultures.

In animals (e.g., rats), sexual preference has been modified in the pre- and postnatal phase by altering the action of male or female hormones. For example, extreme stress of the mother often leads to androgen suppression in the male fetus (Bailey, Willerman, & Parks, 1991). Although these rats develop external male sexual organs, they show female copulatory behavior. If female rats are prenatally exposed to androgens, the reverse behaviors occur. A genetic component also seems to be relevant for homosexuality: the X chromosome of some homosexual males seems to have specific markers for homosexuality in the 928 region (Le Vay et al., 1995). This assumption is substantiated by the observation that children who are raised by homosexual couples do not show an increased prevalence of homosexuality, thus rendering the role of environmental factors unlikely. Defense reactions of the maternal immune system against androgens of the male fetus have also been implicated in the development of "female brains" in male bodies.

The common final path of these influences seems to be the hypothalamus. Male and female brains show the most pronounced differences in this structure. It is, however, not clear if these alterations are really at the basis of differences in sexual orientation. Dimorphic regions of the hypothalamus are not fully developed by the postnatal period; rather, the sexually dimorphic nucleus of the preoptic region can only be differentiated between the sexes at age four and there are substantial anatomic and neurochemical alterations in this structure in puberty and in later age. It is therefore likely that some gender differences in the brain may not relate to different brain development but to differential cell death of brain regions in the sexes. The vasopressin-containing subnucleus of the nucleus suprachiasmaticus was found to have twice the size in homosexual as compared with heterosexual males, whereas no reduction (demasculinization) of the sexually dimorphic nucleus of the preoptic region was observed. In transsexuals, similar deviations have been observed (LeVay, 1996).

#### 1.05.9.4 Acquired Motivation and Substance-induced Disorders

The substance-induced disorders with their accompanying addictive behaviors are a "model" for acquired motivation which yielded important information about the neuronal mechanisms of drives and incentives. Although a genetic risk has been proposed for the

addictions, they reflect primarily learned behavior patterns that are determined by psychological and biological factors. Although psychosocial factors may play the most important role in the development of the addictions (see Volume 6), their maintenance is strongly determined by learning and central nervous system variables.

The positive and negative reinforcing properties of substances and the time course of their intake are important determinants of addiction. Whereas previous definitions of substance-induced disorders focused mainly on the phenomenon of tolerance (reduction of the efficacy of a drug over time related to pharmacokinetic (metabolic), pharmacodynamic (receptor-related) mechanisms, and withdrawal symptoms), current formulations (American Psychiatric Association [*DSM-IV*], 1994) focus more on compulsive drug seeking and craving as determinants of drug abuse (Robinson & Berridge, 1993).

#### **1.05.9.4.1 The neurobiology of addiction**

The psychobiological basis of drug abuse and drug dependence has been well established. Important—although not necessary—characteristics of dependence are the development of tolerance and withdrawal symptoms with repeated drug usage. Tolerance refers to the decrease in efficacy of a drug with repeated administration and has been related to both pharmacokinetic and pharmacodynamic processes. Pharmacokinetic tolerance is based on the increased availability and efficacy of drug-metabolizing enzymes that lead to enhanced absorption of the drug into the blood stream. Pharmacokinetic tolerance is related to changes at the receptor level, for example, dopamine receptors of limbic cells. Both tolerance and the development of withdrawal symptoms are related to neuroadaptive processes whose molecular bases have been described to some extent (Di Chiara, 1995; Self & Nestler, 1995). A structure of special relevance for the development of addictive behaviors is the mesolimbic dopamine system. This system seems to be the final common path of the positive reinforcement system of the brain that was discovered by Olds and Milner (1954) in their experiments on intracranial self-stimulation.

#### **1.05.9.4.2 Learned motivation and addiction**

In 1954, Olds and Milner first showed the phenomenon of intracranial self-stimulation (ICSS) (Olds & Milner, 1954). The implantation of electrodes in the septum led rats to self-stimulate at a very high rate up to complete

exhaustion. Stimulation of the lower structures in the midbrain (the periventricular system) had an opposite effect: the animals attempted to prevent any kind of electrical stimulation. Olds therefore called those centers pleasure or punishment centers. Persistent ICSS can be elicited from many subcortical and cortical regions: the optimal regions in the rat are the descending medial forebrain bundle, the lateral hypothalamus, and the frontal cortex. Preferred sites of self-stimulation are those fibers of the medial forebrain bundle that lead from the lateral hypothalamus to the posterior and ventral tegmentum. Those sites coincide to some extent with the dopamine system. The dopaminergic fibers from which intracranial self-stimulation can be elicited are mainly ascending in the direction of the forebrain. This includes the part of the medial forebrain bundle that leads to the nucleus accumbens as well as the ventral tegmental bundle, the posterior medial forebrain bundle, and the capsula interna, which have connections to the caudate nucleus, the putamen, and the amygdala. Thus the dopamine system seems to be the common final path of the descending medial forebrain bundle–lateral hypothalamus system and of endogenous opiate cells. Neuroleptics block this final common path of the positive incentive system and lead to anhedonic behavior by reducing incentive salience of reinforcing aspects of the environment.

In humans, rather than complete anhedonia, reduction and blunting of positive affect and reduced incentive motivation seem to be the consequence of the intake of neuroleptics. Whereas dopaminergic neurons seem to be related to the energizing incentive motivational aspects of positively motivated behavior, the endogenous opiates seem to be important for the positive affective tone of reinforcing stimuli. This effect of the endogenous opiate system is probably primarily related to the inhibition of noxious inputs. Opiate receptors are primarily located close to the pain-processing centers in the central nervous system. They modulate pain perception in the dorsal horn of the spinal cord over the periaqueductal gray (where descending pain inhibition can be elicited) to the thalamic relay stations for painful stimuli in the amygdala, the temporal cortex, as well as the striatum, where they end in ascending dopaminergic neurons. The high addictive potential of the opiates is probably based on this aversion-reducing effect.

The development of tolerance and the increase of incentive motivation are based on intracellular sensitization and neuroadaptation. It is assumed that the increase of incentive motivation and incentive salience after the



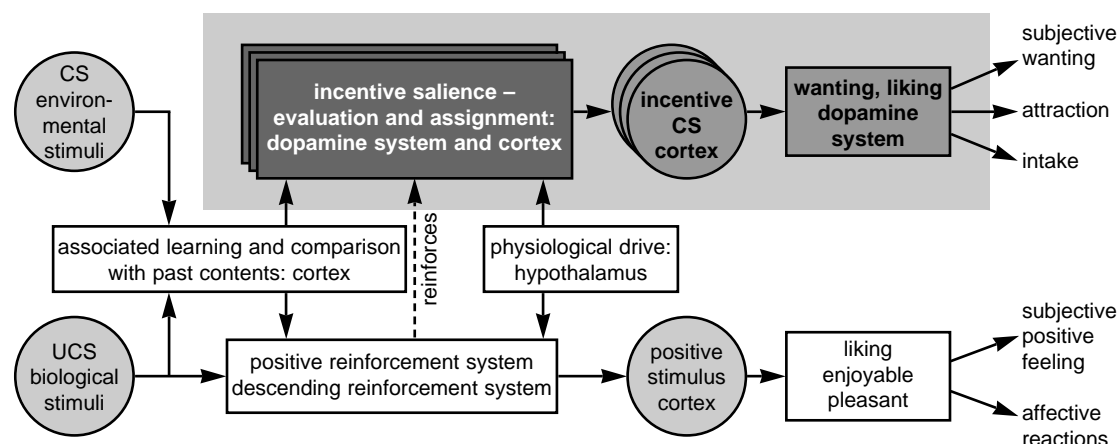
intake of drugs is related to a sensitization process of the mesolimbic dopamine system and that tolerance and development of dependence are related to an intracellular process of neuroadaptation. The positive reinforcing value seems to be elicited by the binding of dopamine to  $D_2$ -dopamine receptors or opiate receptors (especially  $\mu$ - and  $\delta$ -receptors). They activate G-proteins that lead to reduced adenylyl cyclase activity, reduced cAMP, and reduced cAMP protein kinase. G-proteins directly regulate  $K^+$  and  $Ca^{2+}$  channels. The reduced cAMP-dependent protein kinase activity leads to reduced phosphorylation of proteins that regulate neuroexcitability such as ion channels, enzymes, and receptors. The infusion of substances that activate protein kinase block positive reinforcing effects (Sp-cAMPS); inhibition of protein kinase increases reinforcement by blocking phosphorylation of the proteins that are necessary for excitation of the nerve.

In the course of chronic drug intake, the cAMP system is adjusted at a higher level in a compensatory fashion in order to compensate for cAMP reduction by the drugs and to guarantee normal excitability of the cells. This leads to systematic changes in gene expression of the proteins that are important for excitability and could explain the long-term effects of incentive motivation, while cAMP reduction is an immediate effect of the direct (with ICSS or amphetamine) or indirect (e.g. with food) positive reinforcement. It is assumed that persons (or animals) who have a genetically higher risk for the abuse of substances show a molecular structure of the dopaminergic cells which is similar to that of persons who are already dependent. In those persons, externally ingested substances have a weaker effect and have to be ingested at a higher level. Tyrosine hydroxylase (TH) synthesizes dopamine (DA) and binds to dopamine receptors that are coupled to G-proteins and neurofilaments (NF). The neurofilaments transport gene products from the axon to the synapse. The cAMP-dependent protein kinases (PKA) modify the ion channels and stimulate the transcription factors (e.g., CREB, c-fos). The nucleus accumbens (NAc) that projects back to the VTA also has connections to the basal ganglia, especially the ventral pallidum (VP), and the hippocampus (HP) and receives projections from the amygdala (AMYG), the olfactory and gustatory system (Olf), and the cortex. After repeated intake of cocaine or morphine in an animal, a dopaminergic cell is extremely easily conditionable to drugs: now the VTA cells and its axon have shrunk, less dopamine is synthesized and released. Adenylyl cyclase and cAMP show a compensatory elevation and PKA leads

to excessive transcription and channel activation. The dopaminergic system becomes over-reactive and this leads to abstinence symptoms (Robinson & Berridge, 1993). In general, the increase of positive reinforcement (euphoria) and incentive salience after repeated drug intake and the activity of the dopaminergic system are parallel processes (American Psychiatric Association, 1994). The courses of wanting and liking in the development of an addiction are not necessarily parallel. Whereas drug wanting may increase with repeated drug intake, the euphoria and positive effects of the drug may slowly decrease. Figure 4 shows an integration of the most important systems and processes for the development of liking and wanting, based on the theory of Robinson and Berridge).

The contiguous pairing of CS and US leads to their associative connection (primarily in cortex); the strength of the associative connection also depends on comparison with similar stored contents. The direction of the attention towards a certain stimulus is guided by this memory process. Activation of the dopaminergic incentive system and the positive reinforcement system is also determined by current drive states. Drugs can stimulate each of the two systems, the positive reinforcement system and the incentive system directly and independently, which means that wanting and liking can be separated. Drugs do have joint effects (cross-tolerance), specific psychophysiological effects, that also determine the course of the dependency. The term cross-tolerance denotes the fact that the adaptation to certain substances (e.g., morphine) also leads to tolerance towards another substances (e.g., alcohol), although one does not have ever to have used this drug.

Some of the endogenous opiate systems are in a close functional relationship with the dopaminergic positive reinforcement structures. The euphorizing (reinforcing) effect of exogenous and endogenous opiates seems to be related to the sensitivity and increased excitability of positive reinforcing structures rather than to tolerance and withdrawal symptoms. The identification of neurochemical mechanisms for reinforcement, tolerance, and withdrawal is complicated by the fact that we have at least three endogenous opiate systems with at least four types of receptor classes. Each of these three systems and receptors could be related to different behaviors that occur during addiction. It is, however, clear that cells show compensatory intracellular processes when they bind their receptors with externally added opiates. Withdrawal leads to a sudden loss of these compensatory effects and counter-regulation leads to an overshoot of the compensation. In alcoholism, the unconditioned dependence



**Figure 4** Comprehensive model of the development of addiction. Bottom: development of pleasure; top: development of craving. *Biologische Psychologie*, by N. Birbaumer and R. F. Schmidt, 1996, New York: Springer. Copyright 1996 by Springer. Reprinted with permission.

(neuroadaptation) seems to be at least partially mediated by opiate receptor binding: during oxidation of alcohol in the liver by the enzymes alcohol dehydrogenase and aldehyde hydrogenase, aldehyde metabolites reach the central nervous system where they create condensation products together with central monoamines which form alkaloids that have a morphine-like structure. These products (tetrahydro-isoquinoline, TIQ) act as “false” transmitters and bind to the opiate receptors. This distorts the opiate neuroadaptation that has been described above.

Nicotine seems to stimulate nicotinic cholinergic receptors at low levels; at higher levels the cholinergic receptors are blocked and relaxation occurs. Nicotine also activates the production of catecholamines and serotonin in the brain which probably leads to increased attentional levels. The blockade of nicotinic cholinergic receptors by mecamylamine reduces smoking, but leads, however, to very significant side effects. These neurobiological principles should be taken into account when addictions are treated.

### 1.05.10 EMOTION AND THE EMOTIONAL DISORDERS

#### 1.05.10.1 Introduction

Emotions are behavioral responses to either aversive or appetitive stimuli that are characterized by responses on the verbal-subjective, the motor-behavioral, and the physiological level. *Emotions* vary along the two basic dimensions of valence (positive-negative) and arousal (arousing vs. calm). Several primary emotions (fear, anger, surprise, disgust, happiness-joy, sadness) have been identified

that seem to be cross-culturally invariant (Ekman & Davidson, 1984). *Moods* are longer-lasting response tendencies towards certain emotional states. Emotional states or feelings are usually short-lived, rarely exceed more than a few seconds, and are characterized by specific muscular responses of the facial muscles with a high communicative value as well as specific approach-avoidance patterns. Moods, by contrast, tend to influence cognitive processes rather than elicit motor responses.

Based on the work of Lange, William James proposed that the conscious experience of an emotion is dependent on the physiological changes we perceive from the periphery of our body. Thus the James-Lange theory of emotion proposes that we “feel sorry because we cry, angry because we strike, afraid because we tremble” (James, 1890). In contrast, the Cannon-Bard theory (Cannon, 1929) suggested that emotions were not characterized by a specific peripheral physiological response pattern and could therefore not be detected based solely on the peripheral response. This theory based the source of the emotion in the brain and considered it as a result of the perception and interpretation of emotion-inducing events in the higher nervous system. This latter view is correct: the stimulation of specific brain regions leads to the experience of specific primary emotions. It is, however, likely that peripheral input also contributes to the experience of an emotion. The work of Ekman and Lang and their colleagues suggests that very specific peripheral physiological patterns are related to the primary emotions (Ekman, 1984). Related to this theoretical controversy is the question, to what extent does cognition precede emotion or to what extent emotions are primary and are only followed by cognitions? The great

emphasis on cognitive processes in clinical psychological models of emotional disorders in the 1970s and 1980s made this question very pressing (Lazarus, Kanner, & Folkman, 1980; Zajonc, 1980). Schachter and Singer (1962) showed in a number of experiments that emotional states that were artificially induced (e.g., by adrenaline) and could not be explained by the subjects were readily attributed to the situational cues that were presented to the subjects. These results were taken as evidence that cognitive variables (attributions) are primary and the specific peripheral activation pattern is secondary in the interpretation of an emotion. There is, however, evidence that emotions may develop irrespective of conscious evaluations of the situation. For example, subliminal perception experiments or experiments with masked stimuli suggest that emotions may be evoked by these stimuli although they are not consciously perceived (LeDoux, 1995; Öhman & Birbaumer, 1993). Aversion to odors or tastes can also be conditioned in an anesthetized state.

### 1.05.10.2 Fear and Anxiety and the Anxiety Disorders

#### 1.05.10.2.1 Avoidance, fear, and anxiety

The learning of anxiety and fear responses has been well described in psychology textbooks (Birbaumer & Öhman, 1993) and will not be reviewed here. In accordance with modern theories of learning (Rescorla, 1977), it is important to note that fear conditioning is stimulus, not response, conditioning: the CS acquires the ability to drive brain systems that activate a defense response that is well established in cerebral networks. Animal experiments have shown that the cortex is not needed in the learning of a conditioned emotional response: obviously the thalamo-amygdaloid connection is sufficient to establish a conditioned fear response. Destruction of the amygdala will eliminate the conditioned emotional response (Mackintosh, 1974).

The cortex is, however, needed for differential conditioning where higher order interpretation of the stimulus is important and for the extinction of a conditioned response. Both the primary sensory and the medial prefrontal cortex, which is closely connected to the amygdala, are needed for extinction. The amygdala thus plays an important role in the acquisition of fear. The instrumental maintenance of fear seems, however, to be mediated by the septo-hippocampal system which regulates the expectancy of safety signals and selective motor avoidance responses. The hippocampus

is necessary for the interpretation of contextual cues, whereas the lateral nucleus of the amygdala governs the conditioned response. If the central nucleus is destroyed, motor, autonomic, and endocrine CRs are abolished.

Lesions of the septum and hippocampus lead to reduced exploratory behavior in novel situations, reduce orienting to expected (but not unexpected) aversive stimuli, facilitate avoidance and escape behavior, and disturb passive avoidance. Both peripheral sympathetic and central noradrenergic systems act in concert when intense stressors have to be dealt with. Noradrenaline seems to have a nonspecific activating function on the neocortex and increases the signal-to-noise ratio, thus leading to increased attention to intensive stimuli (Cameron, 1994). The noradrenergic fibers are organized into a dorsal bundle (periventricular, originating in the locus coeruleus), which runs to the cortex and hippocampus, and a ventral bundle (tegmental, not originating in locus coeruleus) which runs to the amygdala and the septum. Destruction of the dorsal fibers does not affect learning but selectively prolongs extinction in animals. In this case the filtering of irrelevant materials is disturbed and passive avoidance behavior is maintained. In humans, the findings on regulatory dysfunctions of the noradrenergic system are much more controversial. Instead of a general hyper-reactivity of adrenergic systems, a specific dysregulation seems to be present that is specific for the various anxiety disorders. For example, in *post-traumatic stress disorder*, marked increases in peripheral autonomic responses (e.g., heart rate, startle response) have been found in general, and especially during confrontation with the traumatic event (Lang, Bradley, & Cuthbert, 1993). In *panic disorder*, clonidine, a noradrenergic agonist, leads to a paradoxical inhibition of the release of growth hormone (GH) from the pituitary gland, whereas in healthy controls noradrenaline leads to an increase in GH release. It is assumed that long-lasting NA release has led to excessive levels of NA in the hypothalamus, which have reduced the sensitivity or number (downregulation) of postsynaptic  $\alpha_2$ -adrenoreceptors. The subjective improvement of the anxiety attack that follows clonidine is in accordance with the hypothesis of reduced sensitivity of central receptors.

#### 1.05.10.2.2 Startle reflex modulation and fear

The startle reflex is a fast, protective, reflex response of the muscular system to loud noise or other intensive surprising stimuli. In humans, activation of the muscularis orbicularis oculi

can be measured by electromyographic recordings about 30–50 ms after a 95–110 dB loud noise was presented. The amplitude of the startle response is influenced by the emotional valence of stimuli that are presented concurrently with the startling noise: fear increases, positive emotions decrease the response (Lang et al., 1993). This affective modulation of the startle response occurs independently of attention and activation but is related to the response disposition of the organism: if the organism is geared toward approach, the reflex is inhibited, avoidance and escape facilitate it since motor responses need to be interrupted when new stimuli are presented which require immediate action. Reflexes that have an approach character such as the closure reflex of the hand or the foot show reverse modulation (Davis, Hitchcock, & Rosen, 1991; Öhman & Birbaumer, 1993).

The brain structures and neurotransmitters involved in modulation of the startle response have been well studied in the rat. Whereas the amygdala is essential for the potentiation of fear-induced startle response in rats, the nucleus accumbens, an incentive and reinforcement structure, shows an inhibitory influence on fear-potentiated startle (Koch & Bubser, 1994). The medial prefrontal and the orbitofrontal cortex primarily inhibit fear during extinction and mediate delay of reinforcement based on expectancies. The medial septum and the hippocampus are activated during stimulus discrimination and passive avoidance of fear stimuli.

Measurement of the startle response has proven to be an important instrument in differentiation of the emotional valence of emotional stimuli. The *valence* of the conditioned stimuli (positive, negative, neutral) determines the potentiation of the startle response, whereas the *arousal* of a stimulus determines changes in skin conductance level (Lang et al., 1993).

#### 1.05.10.2.3 Psychopharmacology of fear and anxiety

Benzodiazepines such as diazepam (Valium) are effective inhibitors of anxiety and bind to GABA (gamma amino butyric acid) receptors prevalent in the limbic system. Benzodiazepines inhibit anxiety by binding to the benzodiazepine receptor which regulates (together with the GABA<sub>A</sub> receptor) Cl<sup>−</sup> influx into the cell. The Cl<sup>−</sup> influx into the cell is enhanced when the benzodiazepine receptor is activated and this increases the hyperpolarization of the cell. This explains why benzodiazepines are not only generally relaxing and soothing but why they

have anti-epileptic effects. Since the benzodiazepines do not have a specific effect on anxiety-related neuronal networks, they cannot effectively reduce anxiety and sleep disorders on a long-term basis, although they do have beneficial short-term effects. In addition, they show a substantial addictive potential. Barbiturates, benzodiazepines, and alcohol seem to inhibit the behavioral inhibition system selectively and may thus selectively influence conditioned but not unconditioned fear responses of the passive but not of the active avoidance type. They do not influence approach behaviors. This is in accordance with the finding that phobias respond well to benzodiazepines, whereas obsessive-compulsive disorders respond badly to them. The necessity of cortical involvement in the extinction of fear suggests that pharmacological reduction of fear may not be indicated when long-term reduction of fear and anxiety, for example by behavioral treatments such as confrontation, is desired.

#### 1.05.10.3 Sadness, Depression, and the Affective Disorders

##### 1.05.10.3.1 Sadness and depression

*Sadness* is a basic negative feeling that accompanies separation and the loss of attachment that is interculturally relatively invariant. *Depression* is a complex mixture of feelings that contains sadness, but also disgust, anger, rage, hostility, fear, guilt, and shame. Thus, depression is always related to a multitude of psychological, social, and biological factors. The focus of this section will be on the biological preconditions of depression.

Sadness and depression are clearly distinct from other feelings not only in subjective experience but also in electromyographic and electrodermal responses. In addition, the expression of different feelings is different in healthy and depressed persons. In electroencephalographic recordings, the induction of negative or depressive moods has been found to correlate with a right frontal activation, but PET studies suggest that the left amygdala and the left prefrontal cortex show increased metabolism. This increase in blood flow in fronto-limbic areas could result from the lack of activity in the positive reinforcing dopaminergic mesocortical connections that are usually inhibitory and could have caused overactivity in the frontal cortex and the amygdala. If one assumes that the left hemisphere and the amygdala show more dopaminergic activity during depressed emotions and more activation, this could lead to an increased inhibition on the left side and an elevated activity in the

electrocortical measures in the right frontal lobe (Davidson, 1993).

The psychological factors that are important in depression and bipolar disorders are reviewed in Volume 6. As in other chronic disorders, genetic predisposition may play an important role especially for bipolar affective disorders. Twin studies as well as adoption studies suggest increased rates of concordance in biological relatives of depressed patients. Studies of circadian rhythms in depressed patients suggest that the endogenous clock may be accelerated. Days and weeks before a depressive episode, desynchronizations in temperature periodicity are found. The circadian temperature curve is flattened or completely irregular. The release of growth hormone is reduced. The amount of REM sleep increases, the amount of slow wave sleep decreases, the entire sleep period becomes shorter, frequent awakening, especially in the morning, occurs. REM latency is shortened, probably due to a low slow wave sleep pressure (see Section 1.05.7). Sleep deprivation tends to improve depression the following day.

#### **1.05.10.3.2 Neurochemistry of depression**

The effectiveness of antidepressant medications has been based on their ability to increase the availability of noradrenaline and/or serotonin in the synaptic cleft. The monoamine theory of depression has, however, not been substantiated: substances that do not directly affect the noradrenergic and serotonergic system are also effective in treating depression (e.g., lithium). Post mortem analyses of depressive persons show no changes in noradrenaline or serotonin content, the precursor of noradrenaline synthesis, the enzyme dopamine- $\beta$ -hydroxylase is unchanged, and the metabolites of central noradrenaline (MHPG) and serotonin (5-HIAA) are also not reduced in depressed patients. Thus, either mechanisms other than the increase of the aminergic transmitters in the synaptic cleft must be assumed or neurotransmitters other than noradrenaline and serotonin cause changes in these substances as a secondary effect.

Antidepressives lead to a loss of noradrenergic  $\beta_2$  and  $\alpha_2$  receptors. The clinical effect of improvement in depression seems to be associated with the speed of reduction of receptor binding and not with the increased availability of noradrenaline and serotonin. Depressions could, therefore, also be caused by too much availability of central noradrenaline and only the destruction of postsynaptic noradrenergic receptors by the drugs (this lasts usually days or weeks) improves the depression. On the other

hand, reduction of the number of receptors is associated with a compensatory increase of activity of the remaining  $\beta$ -receptors.

Tricyclic antidepressants lead to a reduction of the synthesis and the metabolism of noradrenaline and serotonin by increasing their availability in the synaptic cleft. The noradrenergic cells fire at a lower level and MHPG in blood and urine is lower. During mania, however, a clear increase of noradrenergic activity has been found that is reduced by lithium. In depressed patients, but also in obsessive-compulsive patients, a lack of sensitivity of the presynaptic  $\alpha_2$  autoreceptors seems to be present. These suppress noradrenaline release when noradrenergic cells are active. When the autoreceptors are blocked, more transmitter is being released. If the receptors are underactive, less noradrenaline is released. The reduction and subsensitivity of the  $\alpha_2$  autoreceptors could, however, also be the consequence of increased noradrenaline and serotonin activity in depression.

Persistent stress seems to increase central as well as peripheral noradrenaline. Long-lasting stress and helplessness lead to a central loss of noradrenaline (Peterson, Maier, & Seligman, 1993). This would coincide with the monoamine theory of depression, but not, however, with an overabundance of noradrenaline. Some of these controversies could be resolved if one considers the dynamic course of action of antidepressants and the dynamic course of coping with stress. In depression and after uncontrollable stress, the activity of cells with  $\beta$ -adrenergic receptors in the brains of animals seems to be low. If demands are addressed to the individual that need energy-mobilizing coping behavior, networks that are connected to noradrenergic systems cannot sufficiently respond (learned helplessness). In contrast, successful coping with stress (immunization) or treatment with antidepressants leads to increased activity of postsynaptic cells with noradrenergic receptors on their postsynaptic membranes. Since  $\beta$ -adrenergic receptors are widely distributed in the brain and can coexist with other receptors on the same neuron, the chronic intake of antidepressants or successful stress management can increase the reactivity of serotonergic and dopaminergic cell systems. The reduced number of  $\beta$ -adrenergic receptors after antidepressant treatment or after stress management with a concurrent increase in the activity of  $\beta$ -adrenergic cells is probably related to a desensitization process of the  $\beta$ -adrenergic membrane. An initially increased noradrenergic activity due to stress or antidepressant intake leads to reduction in the number of  $\beta$ -receptors. The receptors that remain, however, increase

their efficacy manifold, which improves the information processing and distribution of these cells.

#### 1.05.10.4 Aggression and Aggression-related Disorders

##### 1.05.10.4.1 Development of aggression

In animals, several subtypes of aggression such as between-male, between-female, fear-induced, maternal, and sexual aggression have been identified. All subclasses of aggression can be reduced to two environmental events: (i) the presentation of aversive, painful stimuli and (ii) the withdrawal of positive stimuli (frustration). If aggression or fear/flight are elicited by certain stimulus configurations, this depends on evaluation of the stimulus configuration; neuronal structures that are involved in these emotions are overlapping.

Aggressive behavior is not a homeostatic drive but primarily a learned behavior whose probability of occurrence is also influenced by constitutional and hormonal factors. The heredity of aggressive behaviors of male mice, for example, is about 0.3–0.5 (with a maximum of 1) based on selective breeding experiments. The variance related to environmental factors varies therefore between 50 and 70%. The genetic transmission for aggressive behavior in humans is not known: the concordance rates of mono- and dicygotic twins seems not to be different, which suggests that the hereditary component is not important. The idea of localization of an aggression gene on the male Y chromosome has not been supported, nor has the relationship between crime and the existence of an XYY gene configuration in men been substantiated (Rose, 1995).

##### 1.05.10.4.2 Neuronal basis of aggression

The localization of aggressive behavior in one or a few brain structures is not possible owing to the heterogeneity of aggressive behavior within and between the species. Some important brain areas for aggression have, however, been identified. Stimulation of the amygdala usually leads to fearful behavior in both humans and animals, but frequently also to aggression. The lateral and medial hypothalamus seem to be integrating structures for aggressive behavior. Stimulation of the lateral hypothalamus leads to prey aggression and stimulation of the medial hypothalamus to affective aggression. Stimulation of the dorsal hypothalamus leads to flight or fear aggression. Experimental lesions in the cortico-medial part of the amygdala can lead to extremely aggressive attacks on

living and inanimate objects. It is assumed that the amygdalae regulate the hypothalamic aggression structure via the stria terminalis. The so-called *Klüver–Bucy syndrome* where the destruction of the anterior temporal lobe leads to tameness and hypersexuality in the rhesus monkey seems to be related to destruction of the amygdala and loss of the temporal lobe.

The integrity of normal social interaction in primates and humans seems to be dependent on intact structures and connections of the amygdala, temporal lobe, and prefrontal cortex. These three structures are anatomically connected by extra- and intrathalamic connections. The anterior cingulate gyrus seems to play a central role. The amygdala, the posterior orbitofrontal cortex, and the anterior cingulate gyrus receive input from all higher sensory cortex areas and project themselves again to the higher motor and premotor areas. Their connections with the reinforcing structures therefore predestine them for the analysis and adequate response to the social meaning of a situation.

The anterior cingulate gyrus seems to be necessary for the generation and maintenance of energy for attention. Its destruction leads in humans to *akinetic mutism*, where patients no longer speak and if asked indicate that they have no energy since nothing is of any meaning to them. Lesions of the orbitofrontal cortex frequently cause *pseudopsychopathy* and a loss of social responsibility. The effects of lesions of the amygdala, the cingulate gyrus, and frontal cortex show that the associative connection of peripheral physiological input and the evaluation of a situation is of central significance for emotional experience since the association is formed in this high-level social vegetative muscular regulation system. Persons with epilepsy of the temporal lobe frequently show emotional disturbances and psychiatric complications especially if the right anteromedial temporal regions that are closely connected to limbic structures are impaired.

##### 1.05.10.4.3 Hormonal basis of aggression

Hormones can have an activating or organizing influence on aggressive behavior. Adult animals who have already been involved in fights no longer need testosterone to maintain their rank. Without the presence of androgens in the fetus and in the postnatal phase the neuronal connections that are needed to form aggressive behavior do not develop. In the hypothalamus and septum of certain strains of mice, androgen- and estrogen-sensitive neurons have been found that are correlated with various types of aggressive behavior.

Castration during puberty seems to inhibit postpubertal increases of aggression. In general, testosterone seems to influence the development of intermale aggression; other types of aggression seem to be less sensitive to variations in testosterone levels. Aggressive behavior seems to be also related to cholinergic activity: anticholinergic drugs suppress aggressive attacks in mice almost completely. Long-lasting learned helplessness leads to endorphine-mediated analgesia and immune suppression, as well as inhibition of the production of androgens. Animals that lose in fights also show opiate-induced analgesia.

#### 1.05.10.4.4 Psychopathy

Psychopaths are persons who repeatedly commit antisocial aggressive acts without being impressed by punishment or negative consequences. They seem not to show any remorse or guilt following antisocial activities, however, they are usually intellectually capable of understanding their behavior as well as its consequences. In general, psychopaths seem to be characterized by reduced autonomic responding in situations that usually elicit fear. In passive avoidance situations, psychopaths are often impaired since they are unable to suppress behaviors that have previously been punished. Rather they continue to execute punished sequences, indicating an inhibitory behavior deficit (lack of fear). Consequently, adrenaline and amphetamine seem to improve the behavior of sociopathic persons, and barbiturates and alcohol can increase psychopathic behavior. In a series of studies, Patrick (1994) has shown that fear potentiation of the startle reflex is absent in psychopaths. Lesions in the orbitofrontal cortex lead to a pseudopsychopathic state that is related to a lack of delay of reinforcement and an inability to learn from punishment or the lack of reinforcement. Therefore, psychopathy might be related to deficits in both the amygdala and the orbitofrontal cortex.

#### 1.05.10.5 The Neocortical Hemispheres and Emotion

Differences between the right and the left hemispheres have mostly been discovered in split-brain patients or in patients with specific lesions of the right or the left hemisphere during presentation of tasks that force the unilateral processing of information (e.g., dichotic listening, fixation). Usually, there is continuous exchange between the hemispheres with the left hemisphere playing an important role in the interpretation and attribution of causes. Le-

sions of the right hemisphere often lead to emotional indifference or euphoric disinhibition, lesions of the left hemisphere may lead to catastrophic reactions with severe depression. In the interpretation of these results it is important to consider that lesions of one hemisphere may result in a disinhibition and thus overactivation of the other hemisphere. Emotional expression is impaired after lesions in right frontal cortex, emotional recognition and discrimination after right posterior lesions. In right parietal lesions the existence and consequences of disease and/or emotional contents are often denied (sensory and emotional neglect), and emotional expression fades or is inadequate. Electroconvulsive treatment for depression is significantly more effective when applied to the right rather than the left hemisphere. By contrast, a left-sided Wada test leads to a depressed state. For the Wada test a sedative drug is injected in the right or left main brain artery, putting the respective hemisphere to sleep. The sensitivity of the right hemisphere for negative feelings is also supported by the fact that aversive stimuli such as pain or unpleasant odors primarily activate the right and positive stimuli primarily activate the left hemisphere.

In depressive disorders, increased right-frontal activation has been reported, and for mania, increased left-frontal activation. Since motor activity and control of right-handers are regulated predominantly by the left hemisphere, right-hemisphere overactivation leads to difficulty in controlling verbal and motor behaviors. This is supported by the fact that left-handers and ambidextrous persons often show emotional, verbal, and psychosomatic disorders. Dyslexias and allergies as well as hyperactivity and irritable bowel syndrome are more frequent in left-handed persons who have a larger corpus callosum with more fibers and a larger right hemisphere. This has been associated with a larger influence of testosterone during development which facilitates growth of the right hemisphere and inhibits growth of the thymus gland (immune competence) in animal experiments.

#### 1.05.10.6 Behavioral Medicine: Application of the Psychophysiology of Emotion to Disease

Behavioral medicine is an interdisciplinary field concerned with the integration of behavioral and medical knowledge in the prevention and treatment of disease with a strong focus on behavioral intervention methods. One of the most important intervention methods in behavioral medicine is *biofeedback* treatment where a

biological signal is recorded, amplified, and subsequently converted to a sound or visual signal which can be perceived by the patient. Thus the control over physiological processes that function normally outside our conscious awareness becomes possible. Biofeedback treatment has been used to successfully influence chronic pain, epilepsy, scoliosis and kyphosis, and Raynaud's disease, among others (Schwartz, 1995). Biofeedback follows the principles of operant learning. It is so far not known to what extent control over autonomic function is possible without involvement of the muscular system which is under voluntary control. The curarization experiments by Miller (1978) and his colleagues designed to address this issue yielded controversial results, but supported in general the notion that operant conditioning of autonomic function is possible. The self-regulation of brain potentials without the alteration of peripheral physiological variables also favors this interpretation.

### **1.05.11 COGNITIVE PROCESSES AND COGNITIVE DISORDERS**

#### **1.05.11.1 Introduction**

Cognitive functions are all conscious and nonconscious processes that occur during the processing of information from within or outside the organism, for example, encoding, comparison with stored information, distribution of information, decoding, and language. The focus of this section will be on the neurobiological foundations of these processes.

#### **1.05.11.2 Functions of the Cerebral Hemispheres**

The term cerebral asymmetry denotes the finding that the functioning of both neocortical hemispheres is an important prerequisite for the regulation of various behaviors and mental functions. Although the right and the left hemispheres usually have a synergistic action in most higher cognitive functions, there is hardly any response where one of the two hemispheres of the brain would not have a certain advantage compared with the other. Although the idea that the two hemispheres subserve different cognitive functions originated in antiquity, it was Paul Broca who provided empirical data to substantiate the different functions of the two cortical hemispheres. Broca found that persons with aphasia all had lesions in the left frontal hemisphere. Shortly after, Karl Wernicke published his observations on patients with left posterior lesions—especially the superior temporal

gyrus—which caused a so-called sensory or receptive aphasia; patients could speak but had difficulty in understanding what was told them. Wernicke developed the concept of the regulation of language that is still valid today: the connections of the primary auditory area to the upper posterior temporal lobe (Wernicke's area) and from there to the lower posterior frontal lobe (Broca's area). He also introduced the term "disconnection syndrome" by postulating specific deficits if the connections between these three areas were interrupted. His student Hugo Liepmann described a right-handed patient who showed an apraxia of the left hand (inability for voluntary movement) subsequent to a lesion of the corpus callosum. The patient also showed agraphia on the left side (inability to write). In post-mortem studies it was shown that the corpus callosum had been destroyed. Liepmann concluded from this case that (i) commands for left-sided movements had to be transferred by the corpus callosum to the right hemisphere and (ii) the left hemisphere was not only relevant for language but also for complex learned movement. This second postulate is supported by the fact that apraxias occur predominantly after left-hemispheric lesions. Subsequent to these lesions, complex voluntary movements cannot be correctly performed even after much training. These apraxias are also called ideomotor apraxias. It is, however, known today that planning, initiating, and execution of voluntary movements can also be initiated in the right hemisphere. The so-called constructive apraxias occur after lesions of the right hemisphere: the deficits that occur here relate, for example, to the construction of puzzles, drawings of clock-faces that can no longer be performed, and drawings and model buildings that can no longer be constructed. Apraxias can, however, also occur after lesions of the basal ganglia and the thalamus.

In the twentieth century, a counter localization position was strongly formulated by Karl Lashley who introduced the principle of equipotentiality. Lashley postulated that specific brain centers were not important for learning and memory but the mass action of several areas of the brain was important. The larger the lesion, the stronger the defect. The position that is taken today is a combination of the two views: certain cognitive processes such as language or motor commands are relatively dominant in one of the hemispheres, other cognitive processes are not specialized to certain brain regions.

Sperry's work on split-brain preparations in cats and monkeys and later a split-brain analysis in patients with complete resection of the corpus callosum have made important contributions to



this question (Sperry, 1952, 1964). A number of influences on sensory processing have been revealed in split-brain patients. If the anterior commissure and the corpus callosum are separated, odors from the right nasal mucosa can no longer be named since the olfactory tract is uncrossed. The right hemisphere can, however, use the left hand to choose between objects of different odors. The right hemisphere is more strongly activated by odors from the right nostril than from the left.

Although the auditory tract is only partially crossed, dichotic presentation of words leads to a preference of the right ear. Sounds presented to the right ear are no longer moved to the left hemisphere in split-brain patients, but simple instructions can also be understood from the right hemisphere. Objects that are not viewed in the right visual field cannot be described, although the right hemisphere can correctly identify them (disconnection agnosia).

Tactile discrimination of the hands cannot be transformed from the secondary somatosensory cortex of the contralateral to the ipsilateral side. This means that learned tactile discriminations remain localized in the hand of the contralateral hemisphere. Separate presentations of visual contents to the right and left visual fields have shown that the right hemisphere is dominant in the analysis of visual spatial patterns and manipulative spatial tasks. Although the right hemisphere is in almost all patients expressively aphasic, drawings are only copied correctly by the left hand. Geometric figures are recognized better when they are projected to the left visual field and geometric and tactile tasks are also solved better by the left hand. The right hemisphere is dominant in face recognition, but not, however, in tasks where the faces are semantically categorized. The presentation of chimeric stimuli leads to separate completions of the entire picture in each hemisphere, although all patients report seeing one unified picture.

The sequential lesion of various fiber bundles including the posterior corpus callosum and the anterior commissure in monkeys showed all the brain structures that are involved in visual discrimination. Visual information leaves area 17 via areas 18 and 19 (visual memory), then crosses to the contralateral side and runs on both sides of the lower temporal lobe and from there to the amygdala and the orbitofrontal cortex (emotional and motivational significance). Here again a transfer between both sides takes place. This tract is therefore called the *what-pathway* compared with the upper parietal connection which has been termed the *where-pathway* (Mishkin & Ungerleider, 1982). Unilateral lesions of these systems have few consequences in the monkey; however, discon-

nection of efferents from areas 17, 18, or 19 in the corpus callosum with additional disruption of connections to the temporal areas leads to blindness, despite preserved optic systems. In addition to apraxia of the left side of the body, agraphia of the left hand to verbal commands and acopia (an inability to copy writing and drawing) of the right hand, tasks that require a cooperation of fine motor movements of both hands are impaired. The few-split brain patients who have expressive language in the right hemisphere suggest that the left hemisphere is rather sequential, analytic, and causal, and that the right is more holistically oriented with rather parallel and intuitive processing.

In new-born babies a preference of left hemispheric processing of language (e.g., right ear) can be detected in dichotic listening tests and in elevated amplitudes for evoked potentials for language on the left side. However, left hemispheric lesions lead to compensatory uptake of language functions by the right hemisphere up to the 10th year of life. Complete compensation seems only to be possible in the first years of an individual's life. Obviously there is an innate predisposition for the localization of language on the left side. New-born babies already have a larger left-sided planum temporale which is the region that lies within the Sylvian fissure behind the auditory cortex. In adults, the left planum temporale is about 2 cm longer than that of the right side. The better a language has been learned, the smaller the cortical area that is needed to produce a certain language performance. This holds at least for cortical blood flow in PET and fMRI studies: the second less well-learned language has significantly larger increases of blood flow on the right and the left hemisphere. Women have less activation of blood flow during language processing than men.

In addition to the genetically determined anatomical preconditions for laterality, auditory experience during development seems to be a central influencing factor for its development: language-deprived children, that is, children who had very little external stimulation to speak, and are therefore also impaired in speaking, show less left lateralization in dichotic listening tests similar to persons who are born deaf. The perception and motor regulation of sign language seem, however, to be primarily left-dominant which depends on the superiority of the left hemisphere for sequential information processing as well as its dominance for complex and learned movements.

The Wada test has been used as an assessment instrument for the analysis of dominance. This test is used before neurosurgical operations to diagnose the dominance of one hemisphere.

Usually a narcotic agent (sodium amytal) is injected into the carotid artery. This leads some minutes later to ipsilateral anesthesia of the entire hemisphere. For the left hemisphere there is usually complete right-sided paresis and global aphasia. Some 96% of right-handed and 70% of left-handed persons have language localized on the left side and 4% of right-handed persons have language localized on the right side; 15% of left-handed persons have a bilateral language localization based on the Wada test.

The relationship between laterality and handedness is positive but far from perfect. The localization of language is in humans usually a much better predictor of an anatomic organization than hand preference. Left-handedness is usually not related to an improved ability of the right hemisphere for expressive and receptive language; however, left-handed people often show less language problems after left hemispherical lesions. Although the causes for left-handedness are not clear, there seem to be two groups of left-handers: one group with a strong genetic component and a second group where left-handedness may be the consequence of pre- or postnatal brain damage of the left hemisphere with a subsequent compensation of the right hemisphere. The first group is not intellectually different from right-handers, except for a certain increase of musical and artistic talents among left-handers.

Tactile tasks that require the recognition of forms and figures seem to be solved better by the left hand in right-handers if the material is presented in a static manner. In the case of sequential presentation the right hand makes less mistakes. The asymmetry of information processing is complemented by an asymmetry of the planning and execution of movement: although the left hemisphere is dominant in right-handers in learned skill tests (thus apraxias are often correlated with aphasias), we move the head and the eyes more to the right side when we solve verbal problems, when we solve spatial problems we move more to the left. Simultaneous speaking interferes more with activities of the right hand, the humming of melodies interferes more with the left hand. There seems to be an interaction of left dominance for motor activity and preferred cognitive processing style.

There is some convergent opinion that the preference of the right hand in about 75% of humans has to do with the upright gait of humans. The preference for the right side of the body is already present at birth. Hand preference develops later than the superior ability of the right hemisphere to process visuospatial tasks. The lateralization of visuospatial functions in the right hemisphere might be caused by the

preferred activation of the left fetal vestibular organs and thus the right hemisphere during pregnancy. The hypothesis of preferred stimulation of the left vestibular organ and the right ear during pregnancy attempts to explain a number of differences in lateralization: for example, that women have a superior ability in verbal fluency (left hemispheric function) but have on the other side a less well-developed ability for spatio-geometric tasks which are solved better by men. Lesions of the left hemisphere lead equally often to aphasias in men and women, although within the hemisphere women tend to be aphasic and apractic rather after anterior, men rather after posterior lesions. In most studies on healthy persons, women were less lateralized in all tasks than men, which suggests that a strong lateralization does not necessarily mean better performance. All the differences between the performance of the left and right hemisphere we have named could be based on a joint anatomical difference, the more variable and therefore flexible intracortical communication of the left hemisphere.

Lateralization of cognitive functions may depend on anatomical differences between the two hemispheres. Left-right differences have not only been found in different areas of the cortex, for example, Broca's and Wernicke's regions, but also in subcortical areas, for example, in the thalamus. These differences are not only on a macroscopic level, but are also visible on a microscopic level in the neuroanatomy of individual neurons, for example, the size of the soma of a pyramidal cell or the structure of their dendritic branches, implying that neuroanatomical differences lead to functional differences (Kolb & Wishaw, 1995; Springer & Deutsch, 1993).

### **1.05.11.3 Evolution and Neurophysiology of Language**

For thousands of years language has been considered as a typically human achievement that separates humans from animals, especially the primates. However, it has been shown that differences between human and animal language are quantitative rather than qualitative. The increase in speed and the heightened variability of the exchange of neuronal communication in the human cortex seems to be a precondition for the achievements of human language as a means of communication.

Vocal language seems to have begun only about 60 000 years ago, and written symbols have been found from up to 30 000 years ago. Language could have developed out of non-verbal communication: gestures as well as vocal sounds are generated primarily in the left

hemisphere, and both functions are lost when lesions of the left parietal cortex occur. Emotional sounds and expressions that can, for example, be observed in primates, are less likely to be the beginning of language: emotional sounds are difficult to be conditioned in order to be used as signs; gestures, however, can also be conditioned in primates. Emotional expression seems to be primarily a frontal and basal ganglia phenomenon in human as well as in primates. Nevertheless, the development of language has begun from gestures and the increasingly perfect control of the facial musculature. Gross movements of the body could have been replaced by subtle movements of the lips and the tongue (Crosson, 1992).

The acquisition and use of language can be explained by associative connections of phoneme, word, and sentence assemblies of cortical and subcortical neuronal assemblies. The neocortex may be—especially in its associative areas—viewed as a large associative storage area. The storage of words, sentences, and syntax is part of the functions of both hemispheres. Like the visual cortex, the auditory cortex also has hierarchically built interconnections of neurons that range from simple to hypercomplex and that respond selectively to various aspects of sound: individual cells answer preferentially to certain frequencies, the beginning and the end of sounds and phonetic characteristics of syllables (ba, pa) and consonants (b, g). Many neurons that represent such simple aspects are interconnected to higher-order assemblies if they are frequently excited together. With respect to the language lexicon, therefore, assemblies of phonemes are built that are characterized by different properties. These coupled phonetic networks are connected during development by further associative connections to syllables and word forms. For example, certain cells in the upper temporal cortex show a very characteristic frequency when a word is acoustically presented, since the same cell is part of a different cell assembly based on the type of word that was presented.

Neonates are already more sensitive to syllables of the mother tongue than to control syllables in the pre- and postnatal phase. Between the sixth and the 12th month of life, syllables that are heard and last about 200 ms (auditory cortex) are frequently repeated (inferior frontal cortex) and are thus associatively interconnected to transcortical syllables and later to phonetic assemblies. These articulations lead, of course, also to proprioceptive stimuli from the articulatory musculature that activate an inferior parietal assembly and are thus connected to an entire assembly in the perisylvian region.

When a child learns that certain word forms always occur in certain contexts (at about the age of 2–4 years), this leads to the simultaneous activation of cell assemblies in many cortex areas (e.g., with respect to a drinking glass: visual for glass, tactile for touching the glass, gustatory for taste, etc.) Cell groups are activated that are then the assembly for the content word that represents an object or an action (nouns, verbs, and adjectives). Depending on the associative context of the word, certain brain areas are activated in an assembly that can be assessed by  $\delta$ -band EEG activity. For example, verbs elicit more motor association and substantives elicit more visual association. This is visible in  $\gamma$ -band oscillations that are more pronounced in the occipital cortex for the nouns and more pronounced in the frontal cortex for the verbs.

The neuronal basis of function words (how, it, is, etc.) that have syntactic functions and are not associated with specific environmental stimuli seems to be the left perisylvian region. When semantic errors occur in a sentence, an event-related brain potential is detected—usually a strong negativity (around 400 ms)—whose location in the brain seems to vary with the location of storage of the respective word. During syntactic errors, late positivities in the left perisylvian regions (around 600–800 ms) seem to occur or late frontal negativity.

It is, however, not clear to what extent those event-related potentials are language specific or to what extent they reflect the excitatory thresholds in semantic or syntactic cell assemblies as has been described before. N400 and other negativities after surprising events seem to indicate a new readiness state of the brain region with new search processes for strategies to solve problems. It is, however, possible to see from the context of a language task based on the event-related potential whether a word or part of a sentence has been detected as right or wrong, when this happens in the brain, and at what location. This can be used for lie detection: the cortical lie detector is a higher positive wave (P300) to words or objects compared with similar control stimuli only the perpetrator can know. If the sequence of the crime is known and test stimuli are well presented, the event-related potentials can lead to very high lie detection quota of 90–100% which are much higher than lie detection based on skin conductance readings.

During imagery of an object, all those brain areas are activated that are also active during its perception. The sequence of the activation seems, however, to be reversed: where the real visual stimulus first activates area 17 (primary visual area) and then the extrastriatal area,

especially area 18, imagery first activates area 18 and only afterwards area 17. The time course is, however, comparable to real perception: evoked potential changes in area 18 are already visible about 200 ms after the beginning of an image. Blood flow changes as measured in PET and MRI are in general in the same region as the perception occurred; however, the retrieval of visual contents from memory leads to PET activation, especially in the left temporal parietal, right parietal, and bilateral frontal cortex which occurs in addition to the activation that occurs during perception (Farah, 1995). The frontal activation which is also visible in EEG is probably related to the activation of working memory. The left perisylvic brain region is always necessary during imagery if it is language related.

#### 1.05.11.3.1 Language disorders

We will not describe language disorders in detail but rather discuss their neurobiological basis. *Aphasias* are language disorders that occur in humans who have already learned a language. The cause is usually an ischemic or hemorrhage-related cerebrovascular insult, a tumor, encephalitis, or trauma. All language modalities are usually disturbed. Although lesions of the left hemisphere usually lead to aphasias, right-hemispheric processes are also involved in language processing. Aphasias also occur in subcortical lesions of the white matter, the basal ganglia, or the thalamus. These subcortical aphasias with initial mutism are usually quickly reversible. The cortical aphasia-causing lesions are usually situated in areas close to the Sylvian fissure: Broca's region (Brodmann's areas 44 and 45) and Wernicke's region (area 22) can be differentiated.

All aphasias include disturbances in the naming of objects, the production and understanding of sentences, reading, and writing. The currently most widely used aphasia test, the so-called Token Test, examines to what extent a patient is able to perform manual manipulations with a number of colored sheets. Almost all aphasics show deficits in this test.

The following aphasia syndromes are clinically significant:

- (i) Broca's aphasia, where language production problems are predominant;
- (ii) Wernicke's aphasia, where language perception problems are predominant;
- (iii) global aphasia, where the entire perisylvic region is lesioned and where both language production and language understanding are severely incapacitated;
- (iv) amnesic aphasia, which is usually based on lesions of the gyrus singularis or other areas

that are close to the left perisylvic regions, sometimes also lesions in the right hemisphere—this aphasia is characterized by minor language deficits, deficits that are characterized by semantic paraphrases and disorders in naming objects;

(v) transcortical aphasias that are characterized by deficits in language production and language understanding or both capabilities and are based on lesions in most of the left area perisylvic area;

(vi) conduction aphasias which show a strong disorder in the ability to repeat words with lesions in the fasciculus arcuatus;

(vii) subcortical aphasias which show an initial mutism and subsequent paraphasias.

An acquired inability to understand written language is called *alexia*. Alexias occur with and without *agraphia*, based on localization of the lesion in the brain tissue. Alexia without agraphia and aphasia seems to be a disconnection syndrome where the connections from the right visual association cortex through the corresponding language area in the left gyrus angularis are interrupted. Alexias with agraphias usually show an isolated lesion in the left gyrus angularis. Agraphias occur very frequently together with aphasias. They can, however, also be assessed independently which points towards partially separated brain structures with regulation of both functions. Lesions in several, often widely different brain regions can cause disorders of writing, which is understandable with respect to the complexity of writing which involves semantic, visual, spatial, and motor functions.

#### 1.05.11.4 The Association Areas of the Neocortex

In a simplifying manner, the three large associative areas of the cortex could be connected to the three main psychological functions: whereas the temporal lobe with its limbic connections seems to subserve primarily memory functions, the frontal cortex seems to regulate motor and motivational behaviors, and the parietal association cortex seems to be the basis of sensory and cognitive function. The posterior striatum is, however, also an important part of parietal function because it provides an indirect subcortical connection specifically to prefrontal regions.

##### 1.05.11.4.1 Parietal cortex

The parietal cortex with its multisensory integration function therefore has an important role as a command center for structuring movements that are directed towards a goal

of motivational meaning. The large number of cognitive disorders related to lesions of the parietal region is based on its central anatomical role between the three modalities of vision, hearing, and somatic sense which provide the parietal lobe with information. The posterior parietal lobe with the gyrus angularis, the gyrus supramarginalis, and the upper parietal lobe is disproportionately large on the right hemisphere in humans. This seems to be related to spatial information processing and the regulation of goal-directed movement in space. The efferents of the posterior parietal cortex project to the frontal and temporal association areas, thalamus, striatum, midbrain, and spinal cord. In addition to afferents from the three primary and secondary projection areas, the parietal lobe also receives input from the lateral and posterior thalamus and hypothalamus.

Engrams for reafferent motor and visuokinetic spatial functions are localized in the parietal cortex. The copying of movements and gestures is disturbed after left parietal lesions, and spatial activity related to drawing is disturbed after lesions of the right hemisphere. A number of lesions lead to disturbances, which inhibit the initiation, planning, and execution of complex and goal-directed movement sequences in left-hemispheric ideomotor *apraxia*. The sequential spatial functions of the left parietal region need to be separated from the perceptive functions of the right parietal region. Visual-perceptual skills are, for example, the recognition of visual objects, the synthesis and comparison of visual objects, line orientation, drawing of lines, and recognition of weights. Visuospatial skills are the location of objects in space, judgment of the direction and distance-topographic orientation in space, and the localization of one's own body or body parts in space. Lesions of the parietal lobe therefore frequently lead to topographic *agnosia* and amnesia. Objects, landmarks, and one's own position with respect to them and orientation are disturbed or are not remembered. For example, the orientation of hand movements to a visible goal is not possible. In parietal-occipital lesions, the cell assemblies for individual characteristics of a stimulus, for example, color, form, and location, can no longer be associatively connected. The consequences of this are completely separate experiences of individual elements of the environment and the loss of orientation, for example, the color of letters can no longer be recognized and the location and size of geometric objects can no longer be compared.

Especially remarkable are disorders of face recognition (*prosopagnosia*): the lack of discrimination of unknown faces occurs during

lesions of the right parietal lobe, the disorder of the recognition of known faces including one's own in the mirror, however, depends on bilateral occipital-parietal lesions. Contralateral neglect occurs especially after lesions of the lower right parietal lobe. The person no longer responds to visual tactile and acoustic stimuli contralateral to the lesion (usually the left side of the body). The person also reports no contents of this side and never orients towards this side if new stimuli are represented. *Neglect* can be viewed as a spatial perceptual disorder or an attention disorder. That the perception is disorganized is suggested by the neglect of the left side independent of attention. If the patients, for example, are to view the square in front of the dome of Milano, they only report about the right side of the dome during perception as well as during imagery. If a patient, however, changes his or her location so that the left visual field now receives the previously neglected side of the dome, the patient now reports in reality as well as in imagery the previously ignored part of the dome. The representation of the external object is obviously lost depending on the local frame of reference of the person.

The *parietal cortex* seems to be part of a widely distributed cortical-subcortical attentional system. The multimodal, parietal, and superior-temporal association areas have the task of comparing incoming excitatory patterns with stored ones and to extract from them the significance of the pattern. Whereas temporally the "what" of objects seems to be analyzed, the parietal region seems to analyze the special location "where." Motivational and activating structures in the limbic system, the basal ganglia, the reticular formation, thalamus, and the frontal cortex play an important role in this attentional system. Unilateral lesion of each of the subsystems can cause unilateral neglect. The frontal and temporal regions modulate the thalamic filter system of the nucleus reticularis by inhibitory connections. Reticular formation activates all cortical regions, but inhibits the nucleus reticularis, which leads to selective gating of the thalamus for incoming regions by its inhibitory influence on all specific afferents. The right hemisphere is dominant in the regulation of these processes during spatial attention. Neglect can therefore occur after every lesion in any of these regions, especially, however, on the right side. The patient can no longer learn to concentrate on the contralateral side. Lesions of the parietal lobe also lead to disturbances of short-term memory and therefore also long-term encoding, especially of visuospatial material. This does not mean that short-term memory is an exclusive

function of the parietal cortex, but only that important steps in the processing of information in declarative short-term memory depend on multisensory integration of the parietal region.

#### 1.05.11.4.2 *The frontal lobe*

Even extensive lesions of the *frontal cortex* which forms about 30% of the neocortex usually create no sensory or motor dysfunction. They have, however, severe psychological consequences. The frontal cortex, especially the dorsolateral frontal and prefrontal cortex, has an important role in the creation of stable contingencies between responses and their consequences especially if those follow with delay; associations between a cue, the following motor response, and the biological–social consequences lead to the formation of stable expectancies. The frontal cortex plays an important role in those expectancies: if the frontal cortex is lesioned behavior loses its future orientation, behavior is difficult to predict, is irregular and extremely stereotypical and perseverative.

In general, three subsystems of the frontal cortex can be differentiated: (i) the motor and premotor regions including Broca's area and the frontal eye fields (areas 4, 6, 44), (ii) the dorsolateral frontal cortex with areas 8, 9, 10, 44, 45, and 46, which is called the prefrontal cortex together with the orbitofrontal cortex, and (iii) the orbitofrontal cortex which includes areas 11, 12, and 47. The prefrontal cortex has often been called the granular frontal cortex because it has an especially well-developed layer IV of stellate cells and spherically organized short dendrites. Areas 4, 6, 8, and 44 have primarily motor functions, the dorsolateral frontal cortex is related to attentional functions, working memory, and the formation of expectancies; the orbital system is related to motivational functions. The most important afferents to the prefrontal cortex come from the medio-dorsal nucleus of the thalamus. In addition, the anterior nucleus of the thalamus, the hypothalamus, the amygdala, the limbic cortex, and the cingular gyrus as well as the mesencephalic reticular formation and the nonprimary sensory and motor association areas project to the prefrontal cortex. All connections are reciprocal, the prefrontal cortex projects especially to area 7 (somatic), area 22 (auditory), and area 21 (visual). The orbitofrontal cortex also has olfactory connections. The efferents of the frontal cortex go to the basal ganglia, the hippocampi, and the limbic region. The connections of the basal ganglia are especially important in the regulation of attentional behavior.

Lesions of the prefrontal and orbitofrontal cortex lead to disorders of the temporal sequence of behavior and therefore to secondary social disturbances. Lesions of the prefrontal cortex lead to typical changes in behavior that have been described in many textbooks using the example of Phineas Gage, whose brain was damaged by an iron rod as a consequence of premature detonation of explosives. Lesions of the frontal cortex damage the ability to plan ahead and to select the right behavioral sequences, to ignore distracting stimuli, and to continue with behavior that has begun, and to remember what has already been done previously. This temporal organization of behavior by the prefrontal cortex needs detailed information about sensory stimuli and the context of the situation from the parietal and temporal cortex as well as the motivational value of a specific given and stored situation. In animal experiments lesions of the dorsolateral frontal cortex, especially of the stria principalis, lead to disorders that include attention as well as learning functions. Tasks that require delayed responses are especially disturbed. Delayed matching to sample (DMS) tasks are also affected: in these tasks a visual stimulus is usually presented and with some delay the same stimulus is presented together with other stimuli and the animal must choose which stimulus has previously been seen; the right choice is reinforced. The target stimulus is changed on a trial-to-trial basis. DMS tasks are also affected after temporal lesions, since they require visual discrimination. Table 1 gives an overview of the various deficits after lesions of the frontal cortex including its motor and premotor areas.

In expectancy situations, animals as well as humans show a characteristic slow cortical potential. Between a warning stimulus and a second imperative stimulus which requires motor or cognitive responses, two negativities or components of the EEG are formed: one after the first stimulus and one before the second stimulus. The first negativity usually has a prefrontal localization and the second one can vary topographically over various cortical areas depending on the response that has to be executed. This first component is reduced over the frontal cortex when motor acts become automatic, when stimuli habituate, and when stimuli have no signal value, informative stimuli increase this component. The first frontal component of the SCPs seems to be related to preparatory activation of memory contents after presentation of the first warning stimulus. The expectancy of the stimulus is the subjective correlate of this preparatory activation. In patients with bifrontal lesions this first component is absent and explains the disturbance of

**Table 1** Overview of the main symptoms after lesions of the frontal lobe.

<i>Symptom</i>	<i>Site of lesion</i>
<i>Disorders of movement</i>	
Loss of fine motor skills	Area 4
Loss of force	4,5, dorsolateral
Faulty planning of movement	Premotor, dorsolateral
Voluntary fixation of eyes	Frontal eye fields
Disturbed corollary discharge	Dorsolateral, premotor
Broca's aphasia	44
<i>Loss of divergent thinking</i>	Orbital
Reduced spontaneity	
Disorders in behavioral strategies	Dorsolateral, orbital
<i>Stimulus control of behavior</i>	
Deficient inhibition of responses	Dorsolateral
Search for risk and loss of rules	Prefrontal
Disorders of associative learning	Dorsolateral
<i>Problems in memory for time</i>	
Disturbed perception of frequency	Dorsolateral
Disturbed retrieval of sequences	Dorsolateral
Delayed responses	Dorsolateral
<i>Disordered social behavior</i>	Orbital, dorsolateral
<i>Altered sexual behavior</i>	Orbital
<i>Disordered olfactory discrimination</i>	Orbital

Source: Kolb and Whishaw (1995).

expectancy processes with longer time intervals between two stimuli as found in delayed response tasks. The basic learning of the association between a response and its consequences or the association between two stimuli seems to depend considerably on prefrontal structures. Between CS and UCS or response and reinforcement, repetitive temporal pairing results in an electrophysiological connection that is mirrored in surface negativity. The prefrontal cortex has an important role in the temporal regulation of information and motor acts for other neocortical regions. This active mechanism takes its energy from limbic and subcortical structures. If this source of energy is absent or distributed temporally independent of learned cues, the temporal sequence of thought and behaviors becomes irregular and the probability for goal-directed behavior is reduced. This has been confirmed for cases of bilateral lesions of the prefrontal cortex.

The prefrontal cortex seems to be activated in all situations when a stimulus requires a temporal discrimination. This is the case with all warning stimuli and conditioned stimuli: the organism estimates the probability of occurrence of a certain stimulus and mobilizes the respective sensory and motor systems in a preparatory manner. If the temporal estimation of future events is faulty or destroyed, the time of

sensory and motor mobilization is not correctly chosen or it may be suppressed. The consequence is the symptomatology of the frontal patient, which has been described as difficult to predict, with irregular behavior, especially in waiting situations and with extreme distractibility. In lesions of the dorsolateral frontal cortex it has been noted that verbally formulated action programs lose their influence on behavior. The disorganization of behavior plans is accompanied by distractibility, the inability to give up response strategies that have once been chosen (perseveration). The inflexibility and perseveration is especially caused by lesions of left area 9, the distractibility occurs in prefrontal regions in general. In many types of disorders (e.g., anxiety, schizophrenia, tics, obsessive behavior, and criminal behavior), the frontal lobe used to be destroyed in order to reduce symptomatology. Bilateral lesions lead to a pseudopsychopathic state, especially if the right frontal cortex is lesioned and a pseudodepressive state occurs after left frontal lobe lesions. Although there is similarity in those behavioral deficits to psychopathic behaviors, there is less stability in behavior in frontal lesions: within a few minutes usually incompatible behaviors can occur in a fast sequence. Self-control requires a series of cognitive operations that require intact prefrontal regions.

#### 1.05.11.4.3 The temporal lobe

The *temporal lobe* has acoustic, visual, and memory functions that are reflected in its anatomical structure. Based on its anatomic substructures, the temporal lobe has sensory functions (primary and secondary auditory system) and portions of the tertiary visual system (superior and inferior portions); the medial and limbic portion by way of contrast has memory functions and is responsible for affective tone. Whereas the parietal cortex is related to the location of function, the temporal cortex is related to the differentiation of objects. The temporal lobe comprises neocortical regions 20, 21, 22, 37, 38, 41, and 42, which are also called archicortex, and the medial phylogenetically older, tripartite portions of paleocortex: gyrus ambiens, parahippocampal gyrus, and uncus, the entorhinal (area 28) and perihinal cortex (areas 35 and 36) belong to the medial temporal memory system—the hippocampus and amygdala are closely associated with the paleocortex.

Table 2 shows the most important functional disorders after lesions of various temporal regions, based on Kolb and Whishaw (1995). The role of the inferior gyrus temporalis for visual discrimination has been demonstrated in animal as well as human experiments. Bilateral lesions of the auditory cortex do not lead to cortical deafness as is the case in the primary visual field. However, tone discrimination is impaired, especially the minimal time that has to pass in order to discriminate two tones or two vocal sounds. The minimal stimulus duration for tone discrimination is 50 ms, but after lesions it can increase substantially which leads to an inability to perceive language if the sounds and words are not presented for a very long time. This is especially true for the left superior posterior temporal lobe. This sound discrimination seems to be impaired in *dyslexias* and in disorders of language comprehension in children. Extensive training of the discrimination ability can reconstitute the cortical capability in these children. In monkeys it has been shown that extensive training of acoustic discrimination in the superior temporal lobe and training of visual discrimination in the lower temporal lobe leads to an increase in frequency of action potentials to tone sequence and visual patterns of more than 30% compared with the time before training. As in the somatosensory cortex there is an expansion of complex receptive fields related to learning. Musical talent seems also to be related to the interplay between the two temporal cortices, for example, musicians with and without absolute pitch differ in the size of the left planum temporale (larger in persons

with absolute pitch) (Schlang, Jancke, Huang, & Steinmetz, 1995).

#### 1.05.11.5 Thought Disorders

One type of thought disorder with extensive loss of intellectual function and memory are the *dementias*. More than half of all persons with *dementias* suffer from *morbus Alzheimer*. Very little is known about the causes of Alzheimer's disease. The early occurring severe forms of Alzheimer's might be a dominantly transmitted anomaly in cell metabolism. The mutation of a gene (*s182*) on chromosome 21 may be involved. This gene codes for the precursor protein of  $\beta$ -amyloid, which is responsible for the destruction of cells and can be found in large amounts in the brain of these patients (Levy-Lahad, Wijsman, & Nemens, 1995). Other researchers have also found mutations of chromosome 1, the function of which seems to be similar to that of chromosome 21. Typical for Alzheimer's disease is the atrophy of the gray matter, especially of the association cortices, atrophy of the dendritic branches, and the presence of histologically very well visible senile plaques and neurofibrillary tangles. Senile plaques are deposits of dead nerve cells with a high content of neurotransmitters and proteins. Neurofibrillary tangles consist of excessive multiplication of normal neurofibrils that usually have supporting function for the cells.

In *Parkinson's disease* a loss of dopamine in the nigro-striatal system has been found. The causes of idiopathic Parkinson's disease are unknown; the uptake of pesticides or drugs that destroy dopamine cells might be one cause which may, however, not be relevant for all patients. Parkinson's symptoms may also occur as a consequence of chronic intake of antipsychotic medication with antidopaminergic effects. Since Parkinson's disease is characterized by a loss of dopaminergic cells in the substantia nigra and the adjacent ventral tegmentum, and since the substantia nigra is part of a complex system with motor, cognitive, and motivational functions, motor deficits alone are improbable in Parkinson's disease. For example, Parkinson patients show disorders which are comparable to those in patients with frontal lesions.

The *schizophrenias* are a heterogeneous group of disorders of attention, perception, and language. The neurobiological causes of schizophrenia seem to be related to an overactivity of the mesolimbic dopamine system and a disorder of the left mediotemporal prefrontal cortex region. The neurobiological basis of type I and type II schizophrenia seem to be different: type



**Table 2** Disorders after lesions of various temporal regions.

<i>Symptom</i>	<i>Site of lesion</i>
1. Disorders of acoustic perception	Areas 22, 41, 42
2. Disorders of the selection of visual and acoustic stimuli	Areas 20, 21, 22, 37, 38
3. Disorders of visual perception	Areas 20, 21
4. Disorders of acoustic perception	Areas 41, 42, 22
5. Disorders of organization and categorization	Areas 21, 38 left
6. Disorders of context memory	Hippocampus
7. Disorders of language perception	Area 22 left
8. Disorders in long-term memory	Area 21 (hippocampus and surrounding tissue)
9. Changes in personality and affect	Areas 21, 38 and amygdala
10. Changes in sexual behavior	Amygdala and temporal?

Source: Kolb and Whishaw (1995).

II patients show clear signs of a degeneration of the brain and have expanded ventricles correlating with intellectual decay. The genetic vulnerability of type II schizophrenia for a virus that might later lead to the schizophrenic disorder may be larger than in type I schizophrenia. Similarities between the type II group and patients with multiple sclerosis suggest an immunological involvement of the CNS in the disorder. In the cerebrospinal fluid of a subgroup of schizophrenics, antibodies against a viral infection similar to the herpes virus have been identified.

However, type I schizophrenics also show neurophysiological changes. Blood flow in the frontal hemispheres, especially the left hemisphere, compared with the occipital region, seems to be reduced, and the frontal glucose uptake in PET also seems to be lower. Event-related brain potentials show a decrease rather than an increase in negativity when attention is directed to a stimulus which might be an indicator of the attentional deficits that have been shown in schizophrenia. Structural changes have also been observed in the temporal hippocampal region. The hippocampus of a schizophrenic shows a disorganized orientation of cells. The growth of the cells in this region takes place in the second and third trimesters of pregnancy and this provides an additional argument for the embryonal cause of some types of schizophrenias. Potentially androgens have a triggering function in the brain of schizophrenics since schizophrenia usually occurs after puberty in men and after the age-related reduction of estrogens in women.

The *dopamine hypothesis of schizophrenia* was based on findings that medication which influences the dopaminergic system has a clear antipsychotic effect in schizophrenic patients. A substance seems to be effective if it quickly binds to dopamine receptors in the cell membrane of the mesolimbic system and if it

inactivates the receptor. This suggests that an overactivity of the mesolimbic dopamine system may be a major cause of the positive symptoms in schizophrenia. It is assumed that the so-called D<sub>2</sub>-receptors in schizophrenics are either increased in number or overly sensitive. Substances that block the D<sub>2</sub>-receptors have the best therapeutic effect, whereas blockade of the D<sub>1</sub>-receptors only has small effects on the symptomatology. A general overactivity of the dopaminergic neurons has not been shown. D<sub>2</sub>-receptors are activated by apomorphine and are blocked by neuroleptic substances. Dopamine agonists such as amphetamine and cocaine can induce schizophrenia-like symptoms in healthy persons and may increase schizophrenic symptoms in schizophrenics. Whereas the mesolimbic dopamine system seems to be overactive, the frontal cortex seems to have lower metabolic activity as shown in PET studies. This could be related either to a compensatory dopamine suppression in mesocortical dopamine fibers or to the inhibitory effect of dopamine in the prefrontal cortex.

The use of dopamine antagonists leads to dyskinesias and a loss of quality of life as important side effects. The most frequent effects are Parkinson-like disorders in movement such as loss of facial expression and tremor. About 10% of the patients also develop irreversible tardive dyskinesias subsequent to long-term medication: tics related to the face and the tongue, incomprehensible gestures, problems in speaking, gross movement of the arms, etc. The receptors seem to develop a denervation supersensitivity: the ongoing inhibition of the receptors by the drugs causes compensatory irreversible supersensitivity to the blocked transmitter (Wise, 1982). Blockade of the overactive mesolimbic dopamine system by neuroleptics not only leads to a suppression of thought disorders but also to anhedonia. As previously described, the dopaminergic system

plays an important role in intracranial self-stimulation: its inhibition reduces the efficacy of positive reinforcement and therefore leads to a general sedation that is medication-induced. The overactivity of the mesolimbic dopamine system as an incentive system could explain some of the symptoms of schizophrenia: since this system creates positive reinforcement and incentive motivation, overactivity leads to reinforcement of a number of stimuli and responses. Unimportant and not connected stimuli become suddenly important and relevant for behavior. The selection of attention breaks down. These are the most important symptoms of the schizophrenic disorder.

### 1.05.12 SUMMARY

This chapter has given an overview of the psychobiological foundations of clinical psychology. The basic fields of psychobiology, such as psychophysiology, psychopharmacology, psychoendocrinology, psychoimmunology, and behavioral genetics, were defined and important principles of these fields were described. The neurobiology of basic psychological functions such as emotion, motivation, memory, cognition, and language was then described and the psychobiological basis of their dysfunction in clinical-psychological disorders was delineated.

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# 1.06

## Fundamental Psychopharmacology

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## 1.06.1 INTRODUCTION AND SCOPE

This chapter is intended to give the well-educated reader an overview of how psychotropic drugs work to produce changes in behavior, mood, or perception. Psychotropic drugs fall into three classes, psychotomimetic (drugs inducing a psychosis), psychedelic (drugs producing a change in one's state of consciousness or mood), and psychotherapeutic (drugs relieving some biopsychological problem). The major psychotherapeutic drug classes are antipsychotics, anxiolytics (anxiety reducing), antidepressants, and stimulants. Accordingly, we will describe how and why health professionals use specific drugs to treat certain neuropsychiatric disorders. It is not our intention to give a comprehensive account of all the relevant topics. However, we will provide citations of current reference works where more technical information can be found. We will introduce the reader to neurophysiology, briefly describing how the targets of psychoactive drugs in the central nervous system (CNS) normally function. Next, we will examine the neuropharmacology of the major classes of neurotransmitters, whose different functions specific drugs can target, and then address the applied biology of drug action (pharmacology). Necessarily, we will discuss some principles of how psychoactive drugs affect behavior (behavioral pharmacology or psychopharmacology). Finally, we will review the psychobiology of neuropsychiatric disorders and briefly describe what medications have been found useful in the treatment of these disorders (clinical psychopharmacology).

To understand how drugs work in the CNS, it is necessary first to understand something about the structure (neuroanatomy) and function (neurophysiology/neuropharmacology) of neurons. The reader can refer to any of three excellent references for more in-depth coverage of topics in these areas (Cooper, Bloom, & Roth, 1996; Feldman, Meyer, & Quenzer, 1997; Kandel, Schwartz, & Jessell, 1991). Carlson (1994) also provides a brief, but excellent, glossary of neurobiological terminology.

The later sources will introduce the reader to key definitions, but a brief overview may be in order here. The key target of drugs is the nervous system, whose basic units are cells called neurons. As described below, neurons talk to one another chemically. The principal chemical signal is the neurotransmitter (NT), a molecule released by one neuron (say Cell "A"). In the simplest scheme, another neuron (say Cell "B") has receptors (again particular types of molecules) designed to detect and respond to the NT from Cell "A." As described below, a host of endogenous (other brain chemicals) and exo-

genous (external compounds like drugs) factors can influence the communication between neurons. Further, when the chemical messages are too strong or too weak, or when the reception is too sensitive or too poor, the normal neural, physiological, and behavioral functions may be disrupted. Here is where clinical psychopharmacology looks for drugs that will normalize the disrupted function of brain chemistry.

## 1.06.2 CNS NEUROPHARMACOLOGY

### 1.06.2.1 Neuroanatomy and Neurophysiology of Neurons

The CNS generally contains two types of cells: neurons and glial cells. Glial cells support the integrity and function of neurons, which serve the brain's sensory or input, integration, and motor or output functions. The principal targets of drugs in the CNS are the neurons, whose components are the cell body or soma, dendrites, and axons. The supporting glial cells are found around all parts of the neuron (astrocytes among axons and dendrites, oligodendrites near cell bodies, and Schwann cells surrounding peripheral axons).

Neurons have five basic features. First, neuron cell bodies contain the genetic material and apparatus necessary to synthesize or make new molecules (amino acids, proteins, peptides, etc.). Cell bodies also play a critical role in initiating a traveling wave of electrical signals (the action potential). Second, neurons generally talk with each other via special extensions from the cell body, called axons and dendrites. Axons form output or efferent paths from the cell body that convey rapid, spiked signals called the action potentials. Dendrites (sometimes called the dendritic tree) provide multi-branched afferent or input paths to the cell body, conveying slower, graded potentials. Third, at the end or terminal of axons lies the synaptic junction or cleft, that is, the point where neurons make contact. An axon may synapse on dendrites, the cell body, other axons, or itself. The presynaptic side of the junction produces specialized molecules called neurotransmitters, packages them in vesicles, and usually releases them into the synaptic junction when the action potential of the presynaptic neuron arrives at the axon terminal. Fourth, on both the presynaptic and postsynaptic sides of the synaptic junction are receptors designed to detect and bind the neurotransmitter (NT), in a lock and key fashion. NT receptors are proteins (i.e., long chains of amino acids), which may reside outside the cell, within the cell membrane, and inside the cell. Receptor activation by an NT then leads to certain events or signals that

ultimately result in some change in the pre-synaptic or postsynaptic neuron, for example, initiating its own action potential. Thus, communication between neurons is basically chemical, not electrical. The final feature is that all parts of the neuron, the cell body, axons, and dendrites, have special double-layered membranes separating the inside from the outside of the cell. In addition, the membrane is semipermeable. That is, it contains ion channels through which some ions (charged atoms, e.g., calcium, potassium, chloride) can pass passively in and out of the cell. Other ions or molecules (e.g., sodium) may require an active transport process involving a chemical energy process called an ion pump.

A neuron's structure or neuroanatomy also determines the ways it can communicate with other neurons. For example, monopolar neurons usually have only an axon (e.g., sensory dorsal root ganglion cells), while bipolar neurons have both dendrites and axons with a simple structure (e.g., granule cells of the retina, olfactory mucosa, and auditory nerve). On the other hand, multipolar neurons have large dendritic fields with one major axonal output. The latter can be short or long and straight or highly branched, contacting other neurons (including itself). The latter arrangements mean that even single neurons may participate in at least three very complex functions. For example, they may receive and integrate input or information from many sources. Next, they may produce similar effects in many different neuronal groups simultaneously (e.g., activation functions during arousal or alerting conditions). Finally, they may produce an organized array of different effects in different neuronal groups (e.g., functions related to induction of "states," like rapid eye movement sleep).

### 1.06.2.2 Features of Neuronal Activity and Synaptic Neurotransmission

Psychoactive drugs produce behavioral changes by altering the ways neurons normally function. We now turn to a brief description of bioelectric features of neuronal activity and transmission, namely, how neurons produce and detect the electrical or chemical signals just described.

#### 1.06.2.2.1 Membrane potentials and neural activation

First, to generate neuronal activity some membrane potential must exist. The neural membrane is semipermeable, meaning that it contains ion channels through which some ions

(charged atoms, e.g., calcium, potassium, chloride) can pass passively in and out of the cell. At resting baseline conditions, the neuron is polarized, that is, is more negatively charged inside the cell (low in sodium ( $\text{Na}^+$ ) and high in potassium ( $\text{K}^+$ ) ions) than it is outside the cell (high in  $\text{Na}^+$  and low in  $\text{K}^+$  ions). An active transport process (the  $\text{Na}^+$  pump) maintains this steady state by extruding  $\text{Na}^+$  from inside the cell through ion channels in the membrane. This resting state of affairs is called polarization. Certain neurotoxins work by altering the way ion channels normally function, for example, tetrodotoxin can block and tetraethylammonium can activate sodium channels.

Certain events (e.g., synaptic transmission) can produce changes in the membrane potentials of the postsynaptic neuron. In the discussions below, we arbitrarily designate Cell "A" as the presynaptic neuron relative to Cell "B," the postsynaptic neuron. If events started by postsynaptic NT-receptor interaction lead to depolarization of the membrane of Cell "A" (i.e., influx of sodium into the cell and a shift toward intracellular positivity), then excitatory postsynaptic potentials (EPSPs) are produced. If, however, the latter events lead to hyperpolarization of the membrane of Cell "A" (i.e., influx of chloride ions and/or outflow of potassium ions and a further intracellular shift toward negativity), then inhibitory postsynaptic potentials (IPSPs) are produced. The membrane potential of the postsynaptic neuron (Cell "A") results from the summation of EPSPs and IPSPs. Once the postsynaptic membrane reaches a threshold level of depolarization (usually at the junction of the cell body and axon), Cell "A" will "fire" (excitation). The latter event involves the propagation of an all-or-nothing action potential (spike) of marked depolarization through the axon to the neuron terminals. Ultimately, the arrival of this action potential at the terminal could result in synaptic transmitter release, which in turn can act on postsynaptic receptors of Cell "B." Again, however, if Cell "B" becomes hyperpolarized, then the generation of an action potential requires much more excitatory input.

#### 1.06.2.2.2 Presynaptic events and modulation

Small spherical vesicles and other organelles involved in making or storing NTs are found in the presynaptic nerve terminals. Only some synaptic vesicles are bound to the presynaptic membrane. The arrival of the action potential at the presynaptic terminal, plus the copresence of calcium ions ( $\text{Ca}^{2+}$ ), causes some vesicles to fuse with the membrane and releases packets of NT into the synaptic cleft. Generally, how much



NT gets released depends on the relative size of the presynaptic action potential and/or the presynaptic neuron's firing rate. The latter depend on the presynaptic membrane's baseline potential and activation of presynaptic receptors.

Two general kinds of presynaptic receptors exist. Autoreceptors are found on the axon terminal, the cell body, or the dendritic region of the same neuron that releases the NT. NTs from a different neuron activate heteroreceptors. Excitatory synaptic input to the presynaptic terminal produces presynaptic inhibition. Inhibition occurs because a depolarized presynaptic membrane potential will result in a smaller amplitude action potential and less NT release. Inhibitory synaptic input to the presynaptic terminal produces presynaptic facilitation. Here, the hyperpolarized presynaptic membrane potential will result in a larger amplitude action potential (and, perhaps, opening of calcium channels) and therefore, enhanced NT release. Again, modulatory factors include the presynaptic membrane's resting potential, adequate synthesis and storage of the NT, the copresence of neuromodulators (e.g., vasopressin) and  $CA^{2+}$ , the proportion of membrane-bound vesicles, and the active transport of the NT back into the neuron or into synaptic vesicles. Psychoactive drugs can act on any of these presynaptic processes, for example, calcium-channel blockers can lower NT release by reducing the availability of this essential ion.

The next issue is whether the NT will effectively activate the postsynaptic receptor. The persistence of NT in the synaptic cleft depends on the three ways of NT removal: its metabolism by extracellular enzymes, its reuptake into the presynaptic terminal (where the NT can undergo intracellular metabolism or be taken up into the vesicles), and its attachment of NT to a postsynaptic receptor. Again, drugs can produce changes in any of the latter processes.

#### **1.06.2.2.3 Postsynaptic events and modulation**

Events started by the interaction between Cell "A"'s presynaptic NT and the postsynaptic receptor of Cell "B" can lead to depolarization of the Cell "B" membrane, producing excitatory postsynaptic potentials (EPSPs). However, the latter events also may lead to hyperpolarization of the cell membrane, producing inhibitory postsynaptic potentials (IPSPs). The membrane potential of the postsynaptic neuron results from the summation of EPSPs and IPSPs and must reach a threshold of depolarization for that cell to fire.

Sources of modulation of postsynaptic receptors are more limited. Removal of one presynaptic input to the postsynaptic cell is

insignificant, since thousands of neurons may synapse on a given postsynaptic cell. On the other hand, any direct modulation of the postsynaptic cell will alter all subsequent signal communication in that pathway. The postsynaptic cell's responsiveness to an NT can be changed in two principal ways. First, the number of postsynaptic receptors may chronically change. Down-regulation refers to a decrease in receptor sites with prolonged use and/or exposure to an agonist. Up-regulation refers to an increase in receptor sites with prolonged disuse and/or exposure to an antagonist. Here, the agonist or antagonist agent can be an endogenous substance or an exogenous drug. Second, changes in how tightly an NT binds to the receptor (i.e., affinity) can influence the effectiveness of the NT–receptor interaction. NTs, neuropeptides, neuromodulators, or drugs can alter affinity (see Section 1.06.2.3).

#### **1.06.2.3 Neural Receptors**

NT receptors, obvious targets for psychoactive drugs, reside outside the cell, within the cell membrane, and inside the cell. These receptors can be found anywhere on presynaptic and postsynaptic neurons. A receptor is a protein membrane site to which a molecule, the ligand, or the NT, binds. Proteins are long peptides (two or more amino acids linked by peptide bonds) that serve in a structural capacity or as enzymes. When the ligand normally activates the receptor, we call it an agonist. An antagonist refers to a ligand that blocks or prevents the receptor's normal activation by an agonist. Ligands described as inverse agonists have receptor effects opposite to those produced by agonists, for example, closing rather than opening an ion channel. Partial agonists and partial inverse agonists have the same but weaker effects as full agonists or inverse agonists. NTs may produce depolarization (postsynaptic excitatory and presynaptic inhibitory synapses) or hyperpolarization (postsynaptic inhibitory or presynaptic excitatory synapses). Here, whether the postsynaptic neuron is more or less likely to be activated defines excitation and inhibition. Research has identified two major receptor classes. Iontotropic receptors are ion-gated and rapid acting, while metabotropic receptors depend on second-messenger processes and are slower acting.

### **1.06.3 NEUROTRANSMITTERS AND NEUROMODULATORS**

Besides the reference works cited above, the interested reader can also refer to three other

resources (Bloom & Kupfert, 1995; Hardman & Limbird, 1996; Schatzberg & Nemeroff, 1995). The classical definition of an NT typically involves the sequences of events described in the previous section. That is, the transmitter may be synthesized in the cell body and transported to the axon terminal or may be synthesized in the axon terminal; in either case, it is stored in the terminal. Presynaptic nerve firing releases the transmitter into the synaptic cleft, where the transmitter can produce some variety of synaptic membrane potential change in presynaptic or postsynaptic receptors. Finally, the transmitter is removed from the synaptic junction by some mechanism. Neuromodulation refers to how each step in the synaptic transmission process can be affected by endogenous substances or drugs. This section will review the major classes of CNS NTs. Where relevant, we will mention the peripheral nervous system (PNS), which also may use a particular NT that is also found in the CNS. For each major NT, we briefly introduce the reader to the NT's functions and then provide a short description of its synthesis and metabolism, its neuropharmacological action (i.e., depolarization or hyperpolarization), the effects of agonist or antagonist drugs on its functioning, and the localization/function of neural pathways that use the NT.

### 1.06.3.1 Acetylcholine (ACh)

ACh is an NT found in brain, spinal cord, nerve endings in muscle, ganglia of the autonomic nervous system (ANS), and postganglionic terminals of the parasympathetic division of the ANS. As described below, this transmitter plays a major CNS role in arousal, memory, and other functions. In the periphery, besides its role in parasympathetic function, it is the major transmitter innervating somatic muscles. Not surprisingly, peripheral dysfunction produces motor impairment and central dysfunction can produce both alterations in states of consciousness (e.g., sleep) and memory function (see discussion in this section and in Section 1.06.6.11).

#### 1.06.3.1.1 Cholinergic neurotransmission and receptors

Cholinergic neurons release ACh from axon terminals. ACh is synthesized from choline, which is transported into neurons. The enzyme acetylcholinesterase metabolizes ACh. Importantly, anticholinesterases, such as those found in insecticides (e.g., physostigmine-reversible) or nerve gases (soman-irreversible), result in

increased ACh levels. Two major classes of ACh receptors exist, nicotinic and muscarinic. The muscarinic receptors are metabotropic and can lead to either depolarization or hyperpolarization (for subtypes, see Cooper et al., 1996).

#### 1.06.3.1.2 Cholinergic neuropharmacology

Pharmacological intervention can occur at most of the steps in cholinergic neurotransmission. The neurotoxin NVP blocks ACh synthesis and vesamicol, its transport into synaptic vesicles. Black widow spider venom promotes ACh release, while botulinum toxin and other substances inhibit its release. Rabies and curare block nicotinic receptors, and atropine and pirenzepine block muscarinic receptors. Cholinergic neurotransmission sometimes involves negative feedback onto presynaptic muscarinic receptors which, when blocked with atropine, results in increased release of ACh.

#### 1.06.3.1.3 Cholinergic pathways

ACh also serves as the NT at the neuromuscular junction and at synapses in spinal cord autonomic ganglia and the parasympathetic autonomic nervous system. Myasthenia gravis, an autoimmune disease associated with progressive loss of muscle tone, results from damage and antagonism of the ACh receptor. Within the brain, cholinergic neurons may be short regional interneurons (e.g., nucleus accumbens, caudate-putamen) or long projection neurons connecting several brain areas. The most common degenerative neurological disorders involve some loss of normal cholinergic function, for example, Huntington's chorea (striatum) and Alzheimer's disease (nucleus basalis, neocortex, and hippocampus). Unfortunately, attempts to promote ACh activity have not worked well (choline, anticholinesterase, ACh-receptor agonists).

The two main cholinergic projection pathways are the basal forebrain system and the pons-midbrain system. Cholinergic neurons with cell bodies in the basal forebrain send axons to most of the nonstriatal telencephalon. The important higher cognitive functions include attention and memory. Axons of cholinergic neurons with cell bodies in the midbrain and pons (the reticular activating system) bifurcate. They send projections both rostral (up) and caudal (down) to affect wide areas of the brain. These pathways serve both activating and integrative functions, promoting arousal, waking, and even rapid eye movement sleep.

### 1.06.3.2 Norepinephrine (NE) and Epinephrine (Epi)

NE is a major NT in the brain and in the postganglionic terminal endings of the sympathetic division of the ANS (notably the cardiovascular system). NE, along with Epi, also serves as a hormone (release from the adrenal medulla) involved in a host of peripheral ANS functions (notable cardiovascular activity). In the CNS, as described below, NE is the NT for cells in the brainstem which serve arousal functions involved in attention, mood, and waking. Accordingly, dysfunctions in these CNS systems have been implicated in mood and anxiety disorders (see Section 1.06.6).

#### 1.06.3.2.1 Catecholaminergic neurotransmission and receptors

Three NTs make up the group called the catecholamines (CA)—NE, epinephrine (Epi), and dopamine (DA)—so-called because of the common catechol chemical structure. Adrenergic NTs include just NE and Epi and biogenic amine NTs include CA-NTs plus indole amine NTs (e.g., serotonin). The primary synthesis pathway for all three CA-NTs starts with the amino acid tyrosine that undergoes enzymatic conversion to DOPA (3,4-dihydroxyphenylalanine). From DOPA, the synthesis path leads first to DA, then to NE (via DA- $\beta$ -hydroxylase), and finally to Epi. Monoamine oxidase (MAO) primarily degrades CAs inside the presynaptic terminal, while catechol-*O*-methyltransferase (COMT) metabolizes CAs in the synaptic junction. However, most of the CA-NT is taken up into the presynaptic cell and restored in vesicles. Many factors affect the rate of CA synthesis, including the concentration of the NT itself (end-product inhibition). The rate and pattern of CA neuron firing can increase its synthesis and turnover or metabolism. A major metabolite of central NE is 3-methoxy-4-hydroxy-phenethyleneglycol (MHPG), measured in blood or urine as an indicator of central NE production.

In mammals, NE is the major NT for the peripheral sympathetic nervous system (PNS) and the CNS, with lesser concentrations of Epi found in both CNS and PNS. Noradrenergic, adrenergic, and dopaminergic neurons respectively use NE, Epi, or DA as their NT. NE and Epi appear to act at one or more of four kinds of adrenergic receptors, located presynaptically and/or postsynaptically:  $\alpha_1$  (a postsynaptic receptor in the brain and in the periphery, where activation contracts smooth muscle),  $\alpha_2$  (located in the brain and in the periphery, where it is a presynaptic receptor modulating NE

release),  $\beta_1$  (located in the brain and in the periphery, where activation produces cardiac stimulation) and  $\beta_2$  (located in the brain and in the periphery, where activation relaxes smooth muscles). Adrenergic receptors in the periphery (adrenal gland, heart, blood vessels, etc.) are called adrenoceptors. NE and Epi are equally effective at  $\beta_1$ -adrenoceptors, but Epi is more effective than NE at  $\beta_2$ -adrenoceptors. CA receptors on non-CA neurons are generally postsynaptic receptors, with presynaptic autoreceptors located in the CA neuron itself.

CA-NTs acting on autoreceptors regulate the release of all CAs. Activation of presynaptic  $\alpha_2$ -adrenergic, muscarinic, opiate and DA autoreceptors produces inhibition of NE release, while presynaptic  $\beta_2$ -adrenergic, nicotinic, and angiotensin II receptor activation results in facilitation of NE release. Further, prolonged exposure to any of the CA or agonist-like drugs (e.g., reuptake blockers) can lead to a subsensitivity or desensitization of the receptor and even a decrease in the number of receptors (down-regulation). This reduction in responsiveness to stimuli, inputs, or agents can be nonspecific (many agents) or specific (one agent). On the other hand, prolonged reduced exposure to any of the CA receptors to the NT can lead to a supersensitivity of the CA receptor to agonist agents and even an increase in the number of receptors (up-regulation). The latter situation could arise from destruction of CA neurons or from a reduced synthesis or increased metabolism of CA-NTs. Essentially, drugs acting on the CA systems can affect both cardiovascular and a host of other neural activities.

#### 1.06.3.2.2 NE neuropharmacology

In PNS and CNS NE neurons, drugs can affect adrenergic activity in at least four general ways. Drugs can act: (i) presynaptically to block NE synthesis ( $\alpha$ -methyltyrosine, FlA-63), storage (reserpine, tetrabenazine), release (amphetamine), and reuptake (amphetamine, cocaine, desipramine); (ii) postsynaptically to block (phenoxybenzamine, phentolamine) or mimic (clonidine) the action of the NT on postsynaptic receptors; (iii) on autoreceptors to indirectly influence catecholamine activity ( $\alpha_2$ -agonist—clonidine;  $\alpha_2$ -antagonists—yohimbine, piperoxan); and (iv) to inhibit the metabolism of NE in the synaptic cleft (pargyline) or presynaptic terminal (tropolone).

#### 1.06.3.2.3 Noradrenergic pathways

Since late 1960s, fluorescence histochemical and other methods have permitted a quite

detailed picture of catecholamine pathways. The *locus coeruleus* (LC) comprises the main brainstem area from which axons of NE neurons arise. These neurons act to inhibit cells on which they synapse by way of the slower metabotropic-receptor hypopolarization process. Cell bodies of the large LC neurons are found in the caudal pontine central grey. LC axons typically form long and extensively branched projections over five major pathways to widely separated areas of the brain. Their targets include all the cortices (probably  $\beta$ -adrenergic receptors) and all the specific thalamic and hypothalamic nuclei. The projection patterns of central adrenergic neurons suggest some general action on most of the forebrain and on autonomic activity. Empirical data showing increased LC responsiveness to novel events argues for some role in vigilance, while other data suggest some role in stress-related processes. The potential functions for LC adrenergic neurons are large, for example, sleep and waking, reinforcement, memory formation, affective functions and dysfunctions, and anxiety–pain interactions (see Bloom & Kupfer, 1995).

### 1.06.3.3 Dopamine (DA)

Dopamine, also a catecholamine, is an NT involved in three major systems associated with activation of motoric, motivational (reward), and learning processes. These systems are described below. Dysfunction or alteration of any one of these systems can have profound consequences, including integrated movements and cognitions, including persistent and abnormal ideations, movements, and behaviors (see Sections 1.06.6.8, 1.06.6.9, and 1.06.6.11).

#### 1.06.3.3.1 Dopaminergic neurotransmission

Neurons using DA as their NT have both similarities (synthesis and metabolism) and differences (organizational complexity) relative to adrenergic neurons. It has already been stated that the synthesis or production of DA is just like that for NE and Epi. What is different in the DA neuron is the absence of dopamine  $\beta$ -hydroxylase, which would convert DA to NE. In the presynaptic cell, MAO converts DA to dihydroxyphenylacetic acid (DOPAC). After release from the presynaptic terminal, COMT converts DA to 3-MT (3-methoxytyramine) and MAO converts 3-MT to homovanillic acid (HVA). In humans, HVA, the primary DA metabolite, provides a good indicator of DA activity when measured in cerebral spinal fluid. The regulation of DA levels is similar to that for

NE. DA release depends on the presence of calcium ions presynaptically and on the rate and pattern of firing in the presynaptic cell, with burst-firing producing enhanced DA release. Since DA neurons have presynaptic inhibitory autoreceptors, DA agonists inhibit, while DA antagonists enhance DA release. Like NE and Epi, the major way DA is removed from the synaptic cleft is by reuptake into the presynaptic neuron. This process involves a DA transporter. We now know that several DA-specific compounds can selectively block this reuptake process to enhance DA activity. Cocaine and amphetamine are examples of nonspecific reuptake blockers.

#### 1.06.3.3.2 Dopamine pathways

Dopamine neuron cell bodies, largely found in the midbrain and hypothalamus, are three to four times more numerous than NE neurons. Middle-length DA pathways connect areas of the hypothalamus with the pituitary (involved in prolactin secretion), other areas of the hypothalamus, and limbic system and areas of the medulla with other brainstem regions. The long-length DA neurons arise from cell bodies in the ventral tegmental (VTA) and substantia nigra (SN, nigrostriatal system) areas of the midbrain. SN-DA neurons project to the basal ganglia (neostriatum: caudate and putamen areas) and serve to activate and organize motor functions (damage and/or dysfunction of these cells is found in Parkinson's disease). Like most DA projection systems, the nigrostriatal system contains interactive, reciprocal connections, so that innervation of striatal neurons results in a decrease in SN-DA activity. VTA-DA neurons form two projection systems, the mesocortical system projecting to limbic cortical areas and the mesolimbic system projecting to deeper limbic areas (e.g., nucleus accumbens, and the amygdala). Generally, the mesocortical and mesolimbic systems serve affective and motivational functions. These brain areas appear responsive to positive and negative events or states (stress, conditioned fear, pain and pleasure). Further, they modulate the consequences of such events (affective states like anxiety and "cognitive" processes like reinforcement; see Bloom & Kupfer, 1995; Wise, 1990). The DA projections largely function to inhibit their targets.

#### 1.06.3.3.3 Dopamine receptors

Postsynaptic targets use both of the two major DA receptor subtypes, D<sub>1</sub> and D<sub>2</sub>. DA-autoreceptors (located on all parts of the DA neuron) typically are D<sub>2</sub> receptors. Activation of auto-

receptors on the cell body or dendrites slow the firing rate of the DA cell, while those at the axon terminal can inhibit DA release or synthesis. Not surprisingly, autoreceptors located far from the cell body synthesis apparatus (e.g., prefrontal or cingulate cortices) only possess the inhibition of release property. Both  $D_1$  and  $D_2$  receptors are metabotropic, involving the second messenger adenylate cyclase, but  $D_1$  activation stimulates while  $D_2$  activation inhibits adenylate cyclase. Further,  $D_2$  receptors are five to ten times more sensitive than  $D_1$  receptors to DA (or DA agonists like apomorphine). Thus, low doses of DA agonists will first affect these autoreceptors, resulting in a net reduction in DA function. Higher doses, of course, will override this effect and stimulate postsynaptic receptors. Nevertheless, postsynaptically, a maximal DA effect on behavior or brain activity may require activation of both  $D_1$  and  $D_2$  receptors. All DA receptors can develop supersensitivity and desensitization with changes in the chronic level of DA activity. So in Parkinson's disease, the loss of SN input produces an increase in striatal  $D_1$  and  $D_2$  receptor density. Increases in  $D_2$  receptor density also are found in the brains of postmortem schizophrenics. However, the presynaptic autoreceptors appear more likely to show desensitization. This means that chronic DA-mediated drug therapy may fluctuate in its efficacy.

Two other DA receptors are subtypes of the  $D_2$  receptor.  $D_3$  receptors are densest in limbic brain areas and have a much higher affinity for specific DA agonists. In primates, the density of a  $D_4$  receptor subtype is highest in the frontal cortex, midbrain, amygdala, and medulla and lowest in the basal ganglia. Of interest, the  $D_3$  and  $D_4$  subtypes have a higher affinity for the atypical antipsychotic Clozapine, which has few basal ganglia side-effects.

#### 1.06.3.3.4 Dopamine neuropharmacology

The same drugs affect NE and DA synthesis, storage, release, reuptake, and metabolism. All CAs produce end-product inhibition of tyrosine hydroxylase (TH) which converts tyrosine to DOPA. Increased neuronal firing changes TH from a low-affinity to a high-affinity form and increases speed of this conversion.  $\alpha$ -methyltyrosine inhibits TH, thereby reducing the production of DOPA. DOPA is very rapidly metabolized, so administering L-DOPA dramatically increases the production of DA. DA end-product inhibition of DA synthesis and release also occurs when DA binds with presynaptic receptors. DA agonists (apomorphine) can inhibit synthesis and release of DA, and DA antagonists (antipsychotics like haloperidol) can enhance them. However, in certain

pathways (SN), but not others (mesolimbic and mesocortical), tolerance to antipsychotics occurs. A return to baseline levels of DA activity, release, and metabolism after long-term treatment illustrates the latter effect.

Various antipsychotics, such as phenothiazines (e.g., chlorpromazine), thioxanthines, and butyrophenones act as DA antagonists. The resulting reduction of DA function produces extrapyramidal (i.e., found outside the primary pyramidal motor system) motor side-effects and the alleviation of psychotic symptoms. However, these antipsychotics also can increase DA turnover, presumably by blocking autoreceptors, which would normally decrease DA function. On the other hand, several drugs can enhance DA activity. Amphetamine and cocaine block DA reuptake and amphetamine may induce the release of DA. At high doses, amphetamine and cocaine will produce a psychotic state that is reversible with antipsychotics (this has suggested a pharmacological model for schizophrenia). Nomifensine and benzotropine, an anticholinergic drug, also block DA reuptake. Pargyline enhances DA function by MAO inhibition (MAOI). MAO would normally degrade intraneuronal DA. All antidepressive drugs that are MAOIs will increase the amount of DA available for release. Tropolone, which inhibits COMT, can block the degradation of extraneuronal DA.

#### 1.06.3.4 Serotonin (5-hydroxytryptamine, 5-HT)

Serotonin, an indole amine like melatonin, functions as an NT for cells residing in the brainstem. These cells project down into the spinal cord and upwards into many areas of the brain. 5-HT also functions as an NT in the periphery (gut and blood vessels). 5-HT, as described below, apparently has sometimes subtle, regulatory functions for mood, sleep/waking, and motivated behaviors. Clinically, drugs that enhance 5-HT levels have antidepressive effect and may stabilize excessive behavior patterns (see Sections 1.06.6.1 and 1.06.6.5).

##### 1.06.3.4.1 Serotonin neurotransmission

Serotonin (5-HT) synthesis starts with the essential amino acid tryptophan. Tryptophan is converted to 5-hydroxytryptophan, which then is converted into 5-HT. As an interesting side note, 5-HT in the pineal body is converted to melatonin. MAO breaks 5-HT down into 5-hydroxyindoleacetaldehyde, which is further reduced to 5-hydroxyindoleacetic acid (5-HIAA).

Unlike the CAs, 5-HT and 5-HIAA do not inhibit the synthesis of 5-HT and its metabolites. However, blocking their catabolism results in linear increases in both. Several avenues can regulate the production and release of serotonin, including diet.

#### 1.06.3.4.2 5-HT neuropharmacology

5-HT is primarily found in peripheral tissues, with only 1–2% found in the brain. Like the CAs, 5-HT binds to many different receptors, but unlike the CAs, different 5-HT receptors mediate very different functions. We know of four 5-HT<sub>1</sub> receptors, four 5-HT<sub>2</sub> receptors, and five 5-HT<sub>3–7</sub> receptors. The most well-researched receptor is the 5-HT<sub>1a</sub> receptor. Some psychiatrists use buspirone, a possible selective antagonist for this receptor, to treat many anxiety disorders. Ondansetron and granisetron, treatments for chemotherapy-induced nausea and vomiting, antagonize the 5-HT<sub>3</sub> receptor. The atypical antipsychotic Clozapine antagonizes 5-HT<sub>6</sub> and 5-HT<sub>7</sub> receptors, and amitryptaline, a tricyclic antidepressant, also antagonizes the 5-HT<sub>7</sub> receptor. 5-HT functions primarily as an inhibitory NT, causing a decrease in tonic firing. 5-HT activity is highest in states of physiological arousal, lowest in sleep, and completely absent in REM sleep. Therefore, an association between increased motor activity and increased 5-HT may exist. This apparently paradoxical action may occur by increased inhibition of inhibitory neurons.

The administration of *p*-chlorophenylalanine hampers enzymatic conversion, competing with tryptophan for access to the enzyme. After synthesis, 5-HT is stored in and released from presynaptic vesicles, just like the CAs. As a result, reserpine will interfere with the storage mechanism of 5-HT as well. The recreational drug lysergic acid diethylamide (LSD) is a partial agonist on presynaptic and postsynaptic 5-HT receptors. It functions to reduce 5-HT release. As mentioned above, buspirone is a 5-HT<sub>1a</sub> antagonist, which causes reduced binding of 5-HT. MAO inhibitors (MAOI) slow the degradation of 5-HT in the cell, allowing more storage of 5-HT in vesicles. Since the 1980s, fluoxetine, fluvoxamine, sertraline, and paroxetine have been found to act as selective serotonin reuptake inhibitors (SSRI). They serve to increase the amount of 5-HT in the synaptic junction.

#### 1.06.3.4.3 5-HT pathways

5-HT-containing neurons cluster in the raphe nuclei, near the midline of the upper brainstem. Some nuclei project caudally to the medulla and

spinal cord, and others project rostrally to the limbic system, striatum, thalamus, and cerebral cortex. Research has discovered two different types of neurons: fat beaded axons from the median raphe and fine axons from the dorsal raphe. Research reveals that the fine axons are more sensitive to MDMA, taken recreationally under the name “Ecstasy.” MDMA produces a profound loss of these axons that lasts for at least months, if not forever. MDMA users have a measurable loss of function of 5-HT metabolism and catabolism.

#### 1.06.3.5 Amino Acid NTs

Certain amino acids have also been found in the brain and appear to act as NTs. Strictly speaking, however, many amino acid NTs have not yet met all the criteria for being called NTs. Despite this, they are the most widespread of all the NTs. CAs and acetylcholine NTs are better researched, but account for only a small part of neurotransmission in the brain.  $\gamma$ -aminobutyric acid (GABA) and glycine are the primary, inhibitory NTs and glutamate and aspartate are the excitatory NTs. Both excitatory (NMDA) and inhibitory amino acid NT mechanisms appear to function in one or more components of learning and memory processes.

##### 1.06.3.5.1 $\gamma$ -Aminobutyric acid (GABA)

Of the two inhibitory amino acid NTs, GABA has been the subject of more research, possibly because of its primary location in the CNS. GABA's particular interest lies in the benzodiazepine (BZD) receptor, which is part of the receptor complex. Activation of this receptor produces anxiolytic (anxiety-reducing) effects (see Section 1.06.6 on anxiety disorders). Also of interest is the fact that the compounds which block activation of GABA or BZD receptors can produce proconvulsant effects (i.e., increased likelihood of seizures).

The synthesis of GABA begins with glutamine, which is then converted to glutamate (an excitatory NT, also known as glutamic acid). Glutamate is then converted to GABA by glutamic acid decarboxylase (GAD). GABA-transaminase (GABA-T) metabolizes GABA. Both GAD and GABA-T require pyridoxine (a form of vitamin B<sub>6</sub>) as a coenzyme, although in differing affinities. As a result, pyridoxine deficiency causes an increased susceptibility to seizures due to the reduced production of GABA. Pyridoxine deficiency occurs most commonly in infants with a vitamin B<sub>6</sub> deficiency, and the resulting seizures can be fatal.

Research identifies two classes of GABA receptors: GABA<sub>A</sub> and GABA<sub>B</sub>. GABA<sub>A</sub> has a gated chloride (Cl<sup>-</sup>) channel, with binding sites for GABA, BZDs, barbiturates, steroids, and picrotoxin. BZDs increase the frequency of opening of the Cl<sup>-</sup> channel, and barbiturates increase the duration of channel opening, thus potentiating GABA's inhibition of neuronal activity. Both ethanol and certain neurosteroids also appear to increase GABA activity. Inverse agonists like beta-carbolines also act at the BZD receptor site but close the Cl<sup>-</sup> channel. The latter effect is similar to that produced by proconvulsants such as pentylenetetrazol (metrazol), which act at the picrotoxin site. GABA<sub>B</sub> receptors are less common than GABA<sub>A</sub> receptors in the CNS and are not linked to Cl<sup>-</sup> channels or affected by BZDs or barbiturates. Baclofen is a well-known agonist for GABA<sub>B</sub> receptors.

As mentioned above, BZDs and barbiturates potentiate the effects of GABA at postsynaptic receptors, which may explain their common anxiolytic action. Flumazenil, a benzodiazepine antagonist, can block this effect. The GABA agonist muscimol will activate GABA<sub>A</sub> postsynaptic receptors, and bicuculline and picrotoxin will block postsynaptic GABA receptors. One can also inhibit degradation of GABA with gabaculine, which is an inhibitor of GABA-T. GABA travels in two long pathways and in short interneurons of the cortex, hippocampus, and spinal cord. GABA is the primary NT in striatal neurons that project to the substantia nigra, which is the brain site with the highest concentration of GABA.

#### 1.06.3.5.2 Glycine

Glycine is very widespread throughout the human body, in part because it is a necessary component of many metabolic pathways. However, research has discovered evidence that it also functions as an NT in inhibitory interneurons of the spinal cord, lower brain stem, and possibly the retina. Strychnine appears to block the inhibitory effects of glycine.

#### 1.06.3.5.3 Glutamate and aspartate

Glutamate and aspartate occur in high concentrations in the brain and exert powerful excitation on neurons. Thus, it is likely that these compounds act as excitatory NTs. Part of their function may involve the establishment of new functional connections between neurons, but dysregulation of their receptors also may produce hyperexcitability, including seizures and even cell death. Each compound also serves

in metabolic pathways, and glutamate is involved in GABA synthesis. Thus, differentiating between these functions and any NT activity the compounds may have is difficult. However, research has discovered five different types of excitatory amino acid receptors, the best-known of which is the NMDA receptor. The NMDA receptor has binding sites for both glutamate and a particular kind of glycine molecule. Phencyclidine (PCP) is an NMDA channel blocker and a drug of abuse. NMDA receptors appear to affect memory and developmental plasticity, having high densities in hippocampal and cortical areas.

#### 1.06.3.6 Neuroactive Peptides and Hormones

Neuroactive peptides are protein fragments, cleaved from larger compounds. Peptides, known mostly to coexist with other NTs, enrich the effect of the classical NT. For example, a peptide may alter the rate of production of another NT or strengthen or prolong the effect of an NT. Hormones are substances that are released from one location and travel to another location to act on receptors there. These compounds may act both on CNS and peripheral organ systems.

Opioid peptides, also known as endorphins, mimic endogenous morphine-like substances that have antinociceptive (i.e., antipain) properties. The class of opioid peptides includes proopiomelanocortin (POMC) peptides (e.g.,  $\beta$ -endorphin,  $\alpha$ -MSH, ACTH), proenkephalin peptides (e.g., met- & leu-enkephalin), and prodynorphin peptides (e.g., dynorphin A & B). The enkephalin peptides may act as NTS (Carlson, 1994). The four major kinds of opiate receptors are mu, kappa, delta, and sigma. Kappa receptors, largely located in the dorsal horn of the spinal cord, induce analgesia by depressing the initial site of pain transmission. The mu receptors, primarily found in the medial thalamus and brainstem, mediate morphine-induced analgesia and morphine's sedative, respiratory depressant, miotic, euphoric, and physical dependence properties. Delta receptors largely overlap both mu receptor location and, to a lesser extent, its effects. Activation of the opiate sigma receptors, primarily found in the limbic system (hippocampus and amygdala) can result in psychotomimetic, hallucinatory, and dysphoric responses.

Several other neuroactive peptides are equally well-researched. Two, released from the posterior pituitary, vasopressin (AVP, antidiuretic hormone) and oxytocin, respectively, causes the kidneys to retain more water and cause contractions of smooth muscles of

milk ducts and the male and female sexual systems. Substance P, a tachykinin, is thought to act as a sensory (pain) NT or modulator. Neurotensin, colocalized in some DA and NE neurons, can intensify the actions of those NTS. Glucagon-related peptides, like vasoactive intestinal polypeptide (VIP), colocalize with ACh. In parasympathetic systems, particularly those of the gut. Pancreatic-related polypeptides, like neuropeptide Y, colocalize with the NTs, NE and Epi, affecting the action of these NTs in the brain (e.g., limbic system) and smooth muscle in the periphery. Some hormones are peptides, like AVP, oxytocin,  $\beta$ -endorphin, and ACTH, but others are steroids (small fat soluble molecules), like cortisol, corticotropin-releasing hormone (CRH), and sex hormones. Neuroactive peptides and hormones are occasionally, but rarely, administered exogenously. These peptides may mediate psychological or neurological disorders or modulate CNS drug actions (see Cooper et al., 1996).

#### 1.06.3.6.1 Other neuromodulators

Clearly, there are a host of other neuropeptides and neuromodulatory compounds not described in the above discussion. Neuropeptides include those involved in regulatory processes related to water balance and thirst (e.g., angiotensin II), to nutritional balance, to growth, and/or to gastrointestinal function (e.g., somatostatin, cholecystokinin). Other neuromodulators include adenosine (which is found at most synapses) and a large class of second messengers. The second messenger compounds (including cyclic AMP and GMP, calcium/calmodulin, phosphoinositide, and eicosanoids) serve to mediate the initial reaction produced by NT-receptor binding by activating intracellular enzymes called protein kinases.

#### 1.06.3.7 The Neuropharmacology of Normal Behavioral Functions

Many books devote considerable space to the description of the neuropharmacology of psychological or behaviorally related physiological functions (see Carlson, 1994). Chapter 1.05 also discusses some of this information. However, a very brief mention of one function should serve to illustrate the multiple involvement of the various NT and neuromodulatory systems. Reciprocal inhibitory interactions among cholinergic (giant-celled, lateral reticular area of the pons), serotonergic (raphe nuclei in brainstem), and noradrenergic (locus coeruleus) areas of the brainstem have been implicated in the regulation of sleep and waking

and in the various sleep disorder syndromes. Further, the neuromodulator adenosine also may act to inhibit cholinergic activation of the frontal cortex by the midbrain reticular formation. The latter reticular effect thought to be important in arousal functions and in the present context is interesting since caffeine and other xanthene compounds inhibit adenosine's actions.

### 1.06.4 BASIC PHARMACOLOGY

This section reviews three topics relevant to a general understanding of psychopharmacology. Pharmacokinetics refer to the principles and processes governing how the body absorbs, distributes, and eliminates drugs. Pharmacodynamics define the ways in which targets of drugs (i.e., drug receptors) behave. The subsection on interaction between drugs and receptors begins to introduce some functional ways in which drugs can affect the CNS. Pertinent reference works for the latter two topics include those by Smith and Darlington (1996), Hardman and Limbird (1996), Dyskstra (1992), Johanson (1992), and Julien (1997).

#### 1.06.4.1 Pharmacokinetics

##### 1.06.4.1.1 Drug administration and absorption

Bioavailability refers to that fraction of unchanged drug reaching systemic circulation, whatever the route of administration. Drug absorption depends on the drug dose, the drug formulation and the route of administration. Selection of a drug dose, usually calculated in drug weight per body weight, depends on many factors. The initial therapeutic window defines the drug concentration in the body that produces the desired effect without intolerable side-effects. Development of tolerance (requirement of a higher dose for the same effect) or sensitization (requirement of a lower dose for the same effect), respectively, might call for the upward or downward adjustment of the dose. Incomplete absorption or reabsorption of the drug from the bloodstream into the body tissue can occur before or after first-pass metabolism by the liver, thereby reducing the drug concentration in the blood. The time that the drug concentration in the blood is above the minimum effective concentration determines the intensity duration of the drug effect.

Drug formulation or preparation (i.e., capsule, tablet, liquid, injection, vapor) depends on the route of administration and absorption. The latter include administering the drug by oral intake through the mouth, by insertion into the rectum, by injection (parenterally through the



skin), by inhalation through the lungs, or by application and absorption through mucous membranes (nose, mouth, etc.).

Oral drugs must be soluble and stable in the stomach and permeable across stomach or intestinal lining to enter the bloodstream (factors include stomach/intestinal acidity or pH and the drug's lipid or water-soluble properties (see below).

Parenteral administration requires that the drug (*drogue*, French for “a dry powder”) be dissolved or suspended in a vehicle, usually physiological or normal saline (0.9% NaCl, table salt in water). Potential problems arising with drug injections include rapid absorption, the necessity of sterile technique, and lack of “recallability.” Absorption is not a factor with intravenous (IV, into the vein) injections, where the effect can be immediate. Licensed health professionals find this method useful for emergencies, dose control, and large volumes and/or diluted irritating agents. However, the IV route involves potential increased risks for untoward effects, requires slower injection rates, and cannot be used for oily or insoluble agents.

Intramuscular (IM, into the muscle) and subcutaneous (SC, under the skin) injections also can produce an immediate response with aqueous substances, with sustained drug blood levels. IM injections can prove useful for the administration of moderate volumes and of oily and some irritating agents. However, they cannot be used with anticoagulant drugs and can interfere with some medical assays. SC injections can be useful for insoluble agents or pellets, but cannot be used for large volumes and can produce pain and necrosis. Intraperitoneal (IP, into abdominal or peritoneal cavity) injections are rarely used in humans.

Rectal drug administration may be expedient but absorption is variable. Drugs absorbed through mucus membranes (nose, mouth, rectum) generally enter the blood stream directly (e.g., nicotine gum, nasal decongestants). Inhalation of gaseous drugs produces rapid absorption, but little is known about inhalation of particulates (e.g., smoke).

#### 1.06.4.1.2 Drug distribution

The volume of drug distribution refers to the quantity of the drug in the body tissue relative to blood or plasma. This measure depends on the degree to which the drug exists in an ionized or a nonionized, lipid (fat) soluble state, and binds to proteins in blood limits its entry into tissue. These factors determines what tissue a drug can enter. Finally, drugs must be transported across membranes (usually in capillaries). Drugs that are lipid-soluble or small water-soluble mole-

cules can passively diffuse or be transported across membranes. Drugs that are large water-soluble molecules require active transport across membranes. The blood-brain barrier (a capillary-glial cell layer) admits nonionized, lipid-soluble drugs, but excludes ionized, water-soluble drugs. Whether the drug reaches the CNS depends on the extent to which it stays in the blood, bound to proteins and how well it passes the blood-brain barrier. Similarly, lipid-soluble drugs can readily pass the placental barrier. Drug risk for the fetus is greatest both early (teratogenesis) and late (respiratory depression) in pregnancy. The circulatory system rapidly distributes drugs through the body, unless the drug is bound to blood-borne proteins. However, not all water-soluble drugs are bound to plasma, for example, the lithium ion.

#### 1.06.4.1.3 Elimination

Clearance refers to the rate of elimination of the drug in urine relative to its concentration in blood (or any tissue). Some maintenance concentrations will achieve a steady-state drug level when rate of drug administration equals the total rate of clearance. A drug's half-life is the time required for a drug to reach 50% of steady-state levels during administration or to decay 50% from steady-state levels after administration ceases. Time-course versus drug-concentration curves map both absorption and elimination processes.

Elimination mechanisms involve the metabolism (breakdown) of drugs by one of four major metabolic routes: oxidation, reduction, hydrolysis, and conjugation. Liver and blood-borne enzymes convert drugs into ionized and excretable metabolites. First-pass metabolism (FPM) refers to the initial transport of the drug to the liver via the arterial portal system. Here, much of it is broken down by one or more of the several metabolic pathways. The rate of metabolism varies with drug and route of administration, for example, FPM is greater with oral than intravenous routes of administration. Drugs and their metabolites are excreted through the kidney, lungs, or bile, but also in sweat, saliva, and breast milk. The rate of metabolism may depend on several factors. Enzyme induction promotes increased enzyme activity (see Section 1.06.5.2), depression of enzymes produced by drug accumulation (e.g., disulfiram blocks metabolism of the alcohol metabolite acetaldehyde). Other metabolic factors include age, gender, circadian rhythms, nutrition, disease state, and genetics (including ethnicity). Most drug interactions also depend on metabolic factors. (For a description of

metabolic pathways in the CNS, see Cooper et al., 1996). Clearly, metabolism also affects the therapeutic window.

### 1.06.4.2 Pharmacodynamics

#### 1.06.4.2.1 Receptor theory

A receptor is a binding site (a protein membrane component) to which a ligand, the drug, can bind. This event produces an intracellular action that results in some drug effect. Yet drugs can bind to many cellular sites other than NT receptors. For example, the D<sub>2</sub> receptor is the primary target site for chlorpromazine, an antipsychotic, and for endogenous dopamine. However, the enzyme inositol-1-phosphatase may be the receptor for lithium, and a calcium channel the receptor for verapamil.

#### 1.06.4.2.2 Drug receptor properties

Drug receptors have several distinctive properties. Generally, a limited number of receptors exist, and the extent to which the drug molecules occupy these sites varies with its concentration. Saturability defines the size of this effect with maximum saturation occurring with occupation of all the sites. Receptor selectivity refers to the extent to which a receptor binds with a particular drug rather than other molecules. Selectivity depends both on the receptor and on the size, shape, and bioelectrical charge of the drug molecule. Reversibility of drug/receptor binding occurs, since the latter interaction is generally not permanent.

How avidly or “tightly” a receptor binds a given drug molecule is described as its affinity. Receptors with high affinity for a drug require a lower drug concentration for full saturation. For example, agonists and antagonists could bind to the same receptor but differ in their affinity. A high affinity agonist and low affinity antagonist could lead to an insurmountable drug effect.

Efficacy refers to the size of the intracellular or drug effect when the drug and receptor interact. Zero efficacy drugs include receptor antagonists, which generally have no effect other than preventing the receptor from being activated by an agonist drug. A full agonist drug has high efficacy and can produce the maximum effect on receptors at a sufficient concentration. Partial agonist or inverse agonist drugs have a lower efficacy and cannot produce the maximal effect at any drug concentration level.

Allosteric regulation of receptors with multiple binding sites can occur. Allosteric (“other side”) binding sites are usually sites other than that for the endogenous agonist. Activation of these sites can change the shape of the NT

receptor, thereby positively or negatively regulating its affinity for the primary endogenous ligand. For example, at the GABA<sub>A</sub> receptor complex, BZD agonists enhance and inverse agonists retard activation of GABA<sub>A</sub> receptor activation.

#### 1.06.4.2.3 Types of drug-receptor interactions

These interactions determine how drugs affect cell function (see Smith & Darlington, 1996). Activation of extracellular receptors can open an ion channel, for example, the action of BZDs on the GABA<sub>A</sub> receptor Cl<sup>−</sup> channel. Stimulation of extracellular receptors coupled to an intermediary G-protein can produce intracellular enzyme changes (tetrahydrocannabinoids). Triggering of extracellular receptors on a transmembrane protein can effect intracellular enzyme changes. Finally, drugs can be directly transported across membranes to affect receptors in the cell nucleus, for example, steroids.

At least three types of receptor antagonism exist. Competitive antagonists bind to the same site as the agonist and compete with the agonist for that site. Noncompetitive antagonists bind to a site different from that to which the agonist binds but block receptor activation by the agonist. Finally, partial antagonists, or mixed agonists and antagonists have agonist properties with submaximal efficacy.

## 1.06.5 PRINCIPLES OF BEHAVIORAL PHARMACOLOGY AND PSYCHOPHARMACOLOGY

Much of the information described below also applies to most nonbehavioral physiological measures of drug effects, for example, heart rate and endocrine effects. Behavioral pharmacology (usually preclinical, animal studies) or psychopharmacology (usually human, clinical studies) defines the special principles or factors one should address when considering drug actions on behavioral or mental processes. Some references relevant to this topic include those by Bloom and Kupfer (1995), Carlton (1983), Leonard (1992), McKim (1986), and the volumes edited by Goldberg and Stolerman (1986), by Grabowski and VandenBos (1992) and by Thompson, Dews, and Barratt (1987).

### 1.06.5.1 Behavioral Drug Properties

#### 1.06.5.1.1 Dose–effect curves (DEC)

Plotting drug effect against drug dose or concentration conveniently illustrates a variety of drug properties, individual differences in drug action, and changes in drug action over

time. Several standard types of dose–response relationships have been identified. Lawful and constant rates of change in response with increases in the dose describe linear DEC<sub>s</sub> (rarely demonstrated across all doses). With sigmoidal DEC<sub>s</sub>, the typical dose–response relation signifies some threshold dose at which a change in the response begins to occur and an asymptotic dose at which higher doses produce no further response changes. Inverted U-shaped dose–effect functions resemble sigmoidal DEC<sub>s</sub>, except that at doses higher than those producing the maximum response, the size of the response change decreases with further dose increases.

#### **1.06.5.1.2 Drug sensitivity and effectiveness**

Potency refers to the dose or concentration of the drug required to produce some effect, for example, different drugs can have different potencies for a given effect. The ED<sub>50</sub> (effective dose) indexes the dose that produces half the maximal drug effect. For example, barbiturates are more potent (require a lower dose in mg/bodyweight) in producing sedation than alcohol. Similarly, haloperidol produces more potent antipsychotic effects than chlorpromazine. Conjoint treatment with a second drug can alter the behavioral potency of drugs (Dwoskin, Neal, & Sparber, 1987). For example, pretreatment with an antagonist will shift the agonist DEC to the right, resulting in a higher ED<sub>50</sub>, indicating a decreased sensitivity or potency for the agonist drug. Likewise, pretreatment with a similar agonist (#2) could shift the DEC for agonist #1 to the left, resulting in a lower ED<sub>50</sub> and possibly indicating some synergy between the drugs. A related DEC metric, the toxicity index (TD<sub>50</sub>) defines the dose at which 50% of patients experience toxic effects. Finally, the therapeutic index, defined as the ratio of the median toxic dose (TD<sub>50</sub>) to the median effective dose (ED<sub>50</sub>), provides a relative measure of a drug's toxicity or safety. Drugs with a very low therapeutic index, like lithium and Clozapine, require monitoring of serum drug levels.

Efficacy provides a measure of the maximum desired effect, given a sufficient drug dose. In the behavioral economics of psychotropic medications, knowing the cost/benefits of different drugs is important. Drugs must have sufficient efficacy with the fewest side-effects and lowest costs. Note that idiosyncratic drug responses can occur, for example, some patients become quite agitated when given BZDs such as diazepam or atypical anxiolytics such as buspirone. While some individual differences in response to drugs are unpredictable, certain factors may predic-

tably alter drug potency and/or efficacy, for example, age, gender, ethnicity, prior drug history, and current psychiatric status.

### **1.06.5.2 Drug Tolerance**

#### **1.06.5.2.1 Definitions**

This phenomenon is most easily shown with sedative/hypnotic drugs. Two definitions of tolerance differentiate drug history versus individual, predrug history. Baseline tolerance defines those initial individual differences in the potency of a drug to produce some effect in a drug-naïve subject. That is, drug-naïve individuals could be hyporeactive, normoreactive, or hyperreactive to a given drug. The source of this potency difference could stem from pharmacokinetic or pharmacodynamic factors or from factors like genetics, age, gender, disease state, emotional state, environment, and so on. Acquired drug tolerance defines the situation where prior exposure to a drug decreases the potency of that drug to produce a given effect. Stated differently, to produce the same, initial drug effect requires a higher dose (i.e., the dose-effects curve shifts to the right producing a higher ED<sub>50</sub>).

#### **1.06.5.2.2 Kinds of tolerance**

Physiological tolerance describes the body's adaptation to repeated drug exposure. Metabolic or pharmacokinetic tolerance could result from an increase in the rate of drug metabolism after repeated drug exposures. For example, the induction of metabolic enzymes for the drug could produce metabolic tolerance. Functional or pharmacodynamic tolerance results from a decrease in the way CNS receptors respond to the drug after repeated drug exposures. A down-regulation of receptors (i.e., fewer) or a reduction in receptor affinity or efficacy could produce a functional tolerance effect. Finally, cross-tolerance may occur, where the development of tolerance to one drug conveys tolerance to drugs within the same class or acting on similar CNS mechanisms.

Behavioral tolerance belongs to a special class of functional tolerance, involving conditioned or learned changes rather than drug receptor changes (see Goudie & Demellweek, 1986). The opponent-process theory of drug tolerance (Siegel, 1990) relies on the Pavlovian conditioning model. According to this theory, most drugs appear to have immediate alpha effects (e.g., a decrease in activity), followed by delayed-rebound beta effects. The latter are opposite in direction to that of the alpha effect (e.g., an increase in activity). Tolerance results from

conditioning of the beta opponent process. With repeated drug exposures in the same context, the beta effect becomes conditioned to the contextual stimuli. It gradually moves forward in time, reducing the initial alpha drug effect. This kind of learned tolerance is specific to the drug exposure context. The compensatory-response model of behavioral tolerance involves instrumental or operant conditioning (Holloway & King, 1989; Schuster, 1978). According to this model, drug effects on behavior result in a loss of reinforcement. This prompts the acquisition of some strategies to compensate for the drug's effects and restore access to reinforcements. This kind of learned tolerance is specific to the drug's behavioral effects and to the behavioral-reinforcement demand characteristics of the task.

### 1.06.5.3 Sensitization

Sensitization refers to an increased potency of a drug with repeated exposures to that drug (i.e., decrease in the  $ED_{50}$ ). The principal theory for sensitization points to pharmacodynamic changes. According to this viewpoint, an increase in the way CNS receptors respond to the drug occurs after repeated drug exposures. The mechanism of these changes is incompletely understood. Nevertheless, the obvious candidates are an up-regulation of receptors (i.e., more) or an increase in receptor affinity or efficacy. This phenomenon is most readily shown with stimulant drugs like amphetamine or cocaine. Some evidence points to the possibility of behavioral or learned sensitization. A related phenomenon, reverse tolerance, refers to an increase in a drug's potency with repeated exposure due to toxic reactions, disease state, or organ damage. For example, chronic alcoholics with cirrhosis may experience increased alcohol effects due to the loss of the chief organ of alcohol metabolism.

### 1.06.5.4 Physical Dependence

Physical dependence refers to a general physiological adaptation to chronic drug exposure, usually manifested during withdrawal from the drug (see Henningfield, Schuh, & Jarvik, 1995). The chronic withdrawal or abstinence syndrome involves physiological, subjective, and cognitive symptoms. The character of the withdrawal syndrome depends on the drug class. For example, sedative/hypnotics, like alcohol, BZDs, and morphine, generally produce an excitable withdrawal profile. On the other hand, stimulants like cocaine or amphetamine generally produce depressive or lethargic withdrawal symptoms. These data suggest an

opponent-process, rebound pattern of withdrawal symptoms. However, both drug classes can produce subjective effects of anxiety. Acute withdrawal after even a single drug exposure also can occur, for example, alcohol hangover.

## 1.06.5.5 Basic Psychopharmacology Principles

### 1.06.5.5.1 Drug-behavior interactions

The preceding Sections on tolerance and sensitization clearly illustrate the importance of drug history in understanding the current drug effects on behavior. However, other biobehavioral factors related to drug history also can influence current drug effects. For example, experience with a drug's subjective and/or behavioral effect probably removes novelty *per se* as a determinant of drug action. Further, prior use of related drugs may activate the same enzyme or brain systems as the primary drug (cross-tolerance?). Further, one's subjective or behavioral history can influence the potency and/or efficacy of a drug's behavioral effects: For example, prior practice on a behavioral task may reduce the size of the drug's effects on task performance. Also, a drug can affect newly acquired behaviors or skills (e.g., young drivers) more adversely. Finally one must distinguish between behavioral antagonism versus masking by drugs. Sometimes a drug may decrease performance or behavior, not by antagonizing some endogenous receptor, but by producing other effects that interfere with that behavior (Dwoskin et al., 1987).

### 1.06.5.5.2 Baseline effects/rate dependency

The intensity or frequency of behavior can influence both the size and direction of a drug's effect on that behavior. For example, with high rates of responding, even a drug like amphetamine will reduce responding (such as methylphenidate's effects in ADHD). Similarly, with low rates of responding, even a drug like alcohol or a benzodiazepine can increase that behavior. In this regard, related baseline factors can modulate a drug's behavioral action. For example, acute or chronic state variables can influence the size of the drug effect. The latter states include an individual's normal states (e.g., arousal level) and any psychiatric state or trait factors (e.g., anxiety or depression).

### 1.06.5.5.3 Discriminative and state-dependent drug effects

Nearly all psychotropic drugs produce interoceptive stimulus cues that subjects/patients can learn to discriminate or detect (see Colpaert, 1986). These drug stimuli can become

conditioned occasion setters for other behaviors. A related phenomenon, state-dependent retrieval, shows that information acquired under a given drug state is most efficiently recalled only when that drug state is present again.

#### **1.06.5.5.4 Reward and reinforcing effects of drugs**

Drugs produce positive reinforcement when the antecedent behavior ("self-administration") increases in its frequency of occurrence (see Dworkin & Smith, 1987; Henningfield, Lukas, & Bigelow, 1986; and Young & Herling, 1986, for descriptions of animal and human studies respectively). Drugs also act as negative reinforcers, that is, they may be self-administered to alleviate some aversive condition (e.g., taking aspirin for arthritis or consuming alcohol to avoid withdrawal or hangover). This latter situation describes the "self-medication" hypothesis of drug seeking. In addition, drugs can produce reward or aversive effects (i.e., positive or negative hedonic states), as indicated by an attraction to or an avoidance of places associated with such states. Strictly speaking, reward and positive reinforcement are not the same, but the same neural systems may mediate both, that is, the dopaminergic mesolimbic brain areas (n. accumbens and VTA; Wise, 1990).

#### **1.06.5.6 Drug Classes**

Multiple classification schemes for psychotropic drugs exist. Sometimes such drugs are grouped by their mechanism of action (see Cooper et al., 1996), by the pattern of their behavioral effects (e.g., drugs of abuse; see Balfour, 1990; Smith & Darlington, 1996), by the symptoms or disease for which they are used to treat (see Stahl, 1996), or by a mixed set of criteria (see Hardman & Limbird, 1996; Julien, 1998). This chapter will not focus on such classification schemes, but the interested reader can refer to the references just cited. The major problem with such classification schemes is that many psychotropic drugs have profiles showing effects on multiple NTS, on multiple aspects of behavior, and on multiple types of psychiatric symptoms.

#### **1.06.5.7 Drug Side-effects, Interactions, and Overdose**

The particular array of side-effects depends on which NT receptors are blocked, i.e., muscarinic (blurred vision, dry mouth, urinary retention, constipation, sinus tachycardia, and cognitive dysfunction),  $\alpha_1$ -adrenergic (blockade of some antihypertensive drug effects, postural

hypertension, drowsiness, dizziness, and reflex tachycardia),  $\alpha_2$ -adrenergic (blockade of some antihypertensive drug effects),  $D_2$ -dopaminergic (extrapyramidal movement disorders), and  $H_1$ -histaminergic (sedation, weight gain). Drugs can have undesired interactions through pharmacokinetic or pharmacodynamic mechanisms. These interactions can vary widely in their potential to cause serious problems. Tricyclic antidepressive drugs can produce potential hazardous interactions when combined with MAOIs, NE, Epi, or phenothiazines. Drug overdosing could result from certain classes of drug interactions but more commonly results from the patient's deliberate attempt at suicide. See Kaplan and Sadock (1996), Hughes and Pierattini (1992), or Gelenberg and Bassuck (1997) for the diagnosis and treatment guidelines for these drug effects.

#### **1.06.5.8 Drug Regulation**

The federal Food and Drug Administration (FDA) in the United States was given the authority under the federal Food, Drug, and Cosmetic Act (FDC) to approve only those new drugs that were both safe and effective. The sale of such drugs is conditional on truthful labeling and all the pertinent information needed for the drug's safe and effective use. Prescription of a drug for unapproved use is not regulated but this does not exempt the prescriber from liability. The Drug Reinforcement Administration classifies drugs according to various levels of their abuse potential, for example, Schedule I drugs like heroin are deemed to have high abuse potential and low usefulness, while Schedule V drugs like *lomotil* have low abuse potential and high usefulness. Scheduled drugs with higher abuse potential require increased caution when prescribing.

### **1.06.6 THE PSYCHOPHARMACOLOGY OF MENTAL DISORDERS**

Drugs used to treat mental disorders are given for a variety of reasons. Some disorders are better understood than others, and psychopharmacological treatments are based on hypotheses about the biological causes or influences of the disorder. Unfortunately, our understanding of many more disorders is limited. Psychopharmacological treatments for these disorders may be based on theories about the biological systems involved. Stahl (1996) describes at least eight ways that a compromised CNS could affect the manifestation or expression of behavior or mental disease: (i) changes in molecular neurobiology (e.g., the inheritance of an abnormal

gene); (ii) loss of synaptic plasticity (i.e., the branching, pruning, growing, and dying of axons and dendrites); (iii) excitotoxicity (e.g., the overexcitation of a cell by glutamate, resulting in cell death); (iv) no neurotransmission (e.g., the loss of DA input to the basal ganglia in Parkinson's disease); (v) too much neurotransmission (see discussion below on the DA hypothesis for schizophrenia); (vi) an imbalance among NTS; (vii) the wrong rate of neurotransmission, and (viii) the wrong neuronal wiring during development. The interested reader is encouraged to refer to any of a number of reference works for more complete descriptions of theories, models, and data on the psychobiology of neuropsychiatric illness (Bloom & Kupfer, 1995; Kaplan & Sadock, 1996).

Frequently, however, treatment drugs that alleviate symptoms of a disorder are discovered both by serendipitous observations and by systematic screening of candidate drugs. Therefore, we will discuss the current theories about the systems involved in many mental disorders and the drugs used to treat the disorders separately. Where research has not differentiated between the biological system involvement of disorders, disorders will be grouped into classes (e.g., depressive disorders). We will not include mental disorders that have no apparent psychopharmacological treatments (e.g., gender identity disorders). Further, because of space limitations, this section will not address all neuropsychiatric disorders (e.g., somatoform disorders, sleep disorders, seizure disorders, and personality disorders).

Before reviewing the clinical psychopharmacology of psychiatric disorder, a few prefatory remarks must be considered. First, currently these disorders are defined based on symptom profiles with inclusionary and exclusionary criteria, and based on some known biological cause (see American Psychiatric Association, 1994; World Health Organization, 1993). However, rapid progress had been made in developing brain chemistry models of these disorders. Improvements in the clinical psychopharmacological study of therapeutic agents have paralleled this trend (e.g., double-blind techniques, preclinical screening with animal models, blind raters, "active placebos," and sophisticated analyses). All of the earlier sections on basic psychopharmacology are relevant here plus two additional concepts, those of rebound and relapse. Relapse refers to the reemergence of the original disease process after discontinuation of the drug, for example, panic attacks return after stopping benzodiazepine medication. Rebound refers to the recurrence of a more severe version of the disease, for example, more severe and frequent panic attacks.

## **1.06.6.1 Mood Disorders: Depressive Disorders**

### ***1.06.6.1.1 Characteristics of depression disorders***

The cluster of symptoms in depression involves abnormalities of mood, its quality, its direction of change from normal (down indicates depression, up indicates mania), and its frequency and duration (Stahl, 1996). However, other dimensions of depression involve symptoms that may be vegetative (reductions in sleep, appetite, weight, and sex drive), cognitive (lowered attention span, memory function, frustration tolerance, and self-esteem), impulse control (suicide and homicide attempts), behavioral (lowered motivation, pleasure, interest, stamina), and somatic (increased headaches, stomach aches, and muscle tension). The possible presence of other distinguishable symptom clusters such as anxiety (comorbid) further complicates the symptom picture of depression. Finally, depression may result from or accompany any number of organic conditions, including neurological disorders, metabolic or endocrine disorders, infectious diseases, tumors, drugs, medications, or poisons.

### ***1.06.6.1.2 The catecholamine (CA) hypotheses of depression***

Many theories of how depressive disorders (e.g., major depressive disorder, dysthymic disorder) develop have been proposed. The most widely known is the CA hypothesis, first postulated by Schildkraut (1965). That certain drugs that alter CA function also alter depressive symptoms provides the strongest evidence for this hypothesis. For example, the effective antidepressives, MAO inhibitors and tricyclics, work to increase CAs in the synaptic cleft. Additionally, the drug reserpine interferes with the CA storage mechanism and causes massive release of CAs, followed by lower than normal CA levels. Reserpine can induce serious depression that is indistinguishable from endogenous depression and administering L-DOPA, the precursor to DA, reverses reserpine-induced depression. A second area of evidence favoring the CA hypothesis is the connection between depression and the norepinephrine (NE) metabolite, 3-methoxy-4-hydroxyphenylglycol (MHPG). Early studies found urinary levels of the NE metabolite were lower in depressed patients compared with controls. More recently, researchers have found that unipolar patients show a wide range of MHPG levels, and we can probably identify the best treatment based on this measure. Low-MHPG patients generally respond well to NE-active antidepressants (imipramine) or fluoxetine, and high-MHPG

patients respond poorly to NE tricyclic antidepressants and fluoxetine. Thus, high-MHPG patients may have a depression not associated with impaired CA function.

Some unanswered questions about the CA hypothesis persist. First, why do drugs such as cocaine (a CA reuptake inhibitor), that act to increase CAs, fail to work as antidepressants? Secondly, why do other drugs, such as iprandole, work as antidepressants yet do not affect any CA system? Thirdly, why do antidepressants require at least two weeks before they produce a measurable clinical effect, although the pharmacological action is essentially immediate? The fourth question is why antidepressants continue to work in spite of postsynaptic receptor desensitization, occurring after chronic antidepressant administration. It seems clear, then, that the CA hypothesis cannot explain all the phenomena associated with depressive disorders, and other or additional theories are necessary.

#### ***1.06.6.1.3 The serotonin (5-HT) hypothesis of depression***

The inability of the CA hypothesis completely to explain some facets of depression led to the development of other theories. The 5-HT hypothesis was originally suggested by European researchers who were encouraged by findings that depressed patients with low levels of the 5-HT metabolite 5-HIAA were at greater risk for violent suicide attempts. More recent research has found that low 5-HT turnover is related to impulsive violent behavior, although it is not specific to depression.

However, the 5-HT hypothesis led to the development of the currently popular CA and 5-HT hypothesis. This theory relies on the fact that NE and 5-HT systems are known to interact with each other. Selective lesions of 5-HT neurons increase NE receptor density in some regions of the brain, and NE receptors cannot be down-regulated without an intact 5-HT system. The strongest evidence for the CA and 5-HT hypothesis rests on two discoveries: first that well-known antidepressants like the tricyclics affect both NE and 5-HT systems, and second that the 5-HT-specific reuptake inhibitors (SSRIs) have strong antidepressive effects but primarily affect 5-HT systems. The suggestion is that depression disrupts a delicate balance between CA and 5-HT systems that can be restored with medication.

#### ***1.06.6.1.4 The cortisol hypothesis of depression***

The last theory to be discussed does not involve CA or 5-HT systems at all. It is the

hypothalamic-pituitary-adrenocortical (HPA) axis dysregulation hypothesis. This theory is supported by the finding that most people with Cushing's disease have symptoms of depression. Cushing's disease is a disorder of the HPA axis characterized by excessive cortisol secretion. The HPA axis dysregulation hypothesis is based primarily on the usefulness of two endocrine challenge tests, the Dexamethasone Suppression Test (DST) and the Corticotropin Releasing Hormone (CRH) Stimulation Test. Dexamethasone is a glucocorticoid receptor agonist that will drive the negative feedback system for cortisol secretion in a person with a functional HPA axis. In nondepressed people, administering dexamethasone causes a precipitous drop in cortisol production. In approximately 50% of depressed patients, cortisol production drops only a little. These individuals are known as nonsuppressors. Interestingly, the rate of cortisol nonsuppression increases with the severity of the depression (e.g., most people with depression with psychotic features are nonsuppressors). From these findings, it is inferred that the glucocorticoid receptors in depressed people are down-regulated or desensitized, so their cortisol levels would regularly be too high.

Many people with major depression also have high levels of CRH in their corticospinal fluid, which is believed to come from chronic hypersecretion of CRH. In a functional system, CRH is released by the hypothalamus and causes the release of ACTH, which itself causes release of cortisol. Administering CRH to a healthy individual induces a dramatic increase in ACTH and, subsequently, cortisol. This challenge is called the CRH stimulation test. Some depressed patients fail to show as large an increase in ACTH and cortisol, which is thought to be caused by down-regulated or desensitized CRH receptors. If a single depressed patient is given the DST and the CRH stimulation test and age is taken into account, the specificity of diagnosis of endogenous depression is more than 90% (Heuser, Yasouridis, & Holsboer, 1994). However, it is not yet clear whether the high levels of cortisol and CRH are a cause or result of, or even unrelated to, development of depression. In addition, no medication has been found to interact specifically with the HPA axis, although traditional antidepressants will reduce levels of cortisol and CRH.

#### ***1.06.6.1.5 Status of depression theories***

Clearly, no single NT theory accounts for all the data. This situation results from the fact that depression is not one disorder and that the various NT systems interact. Also, dysfunction of any of several NT systems may be sufficient to

produce depression. For example, cholinergic neurons activate muscarinic receptors on the NE neurons of the locus coeruleus. Further, cholinergic agonists are known to induce depressive symptoms and chronically depressed patients are hypersensitive to cholinergic agonists.

#### **1.06.6.1.6 Drug therapies for depression**

Clinical psychopharmacology of depression is very effective. Approximately 60–70% of people with depression respond to pharmacotherapy, and rates go up to 80% if limited to mild or moderate major depressive disorder. Three types of antidepressants are currently used: MAO inhibitors, tricyclics and tetracyclics, and SSRIs. MAO-inhibiting drugs were the first antidepressants, and include phenylzine, tranylcypromine, isocarboxazid, and selegiline. MAO inhibitors inhibit the action of MAO, which metabolizes CAs. The result is that more CA is available in the cleft. Both MAO<sub>A</sub> and MAO<sub>B</sub> inhibitors are useful, as MAO<sub>A</sub> breaks down NE, 5HT, and DA, and MAO<sub>B</sub> breaks down DA. The three drugs listed inhibit the action of MAO irreversibly, which requires that new enzymes be synthesized. European health professionals use reversible MAO inhibitors that are not currently available in the USA.

However, potentially serious side-effects occur with many MAO inhibitors. The most famous side-effect is the “cheese reaction.” Foods containing tyramine can induce hypertension in patients on MAO inhibitors. MAO also metabolizes tyramine, and its inhibition in the gastrointestinal tract allows tyramine to pass into the blood where it causes a sympathomimetic response. Patients on MAO inhibitors should avoid a range of foods, including certain cheeses, some types of alcohol, and some cured meats and sausages. Fortunately, the MAO<sub>B</sub> inhibitor selegiline does not produce the cheese reaction. In addition, because MAO is involved with many systems in the body, patients should not use multiple medications, including many over-the-counter cold or flu remedies. MAO inhibitors should also not be combined with SSRIs or tryptophan treatment. The latter combination poses a great risk of the 5-HT syndrome, which can include tremor, hypertonicity, myoclonus, autonomic symptoms, hallucinations, and fever. MAO inhibitors also commonly induce insomnia. In fact, the alerting properties of MAO inhibitors can be used to advantage in hypersomnic people. A related effect is afternoon fatigue, which can be severe. In part due to the serious side-effects, MAO inhibitors are generally prescribed for treatment of refractory depression, particularly depression with atypical features (hypersomnia, weight

gain), combined depressive and anxious symptoms, or personality disorder symptoms.

Tricyclic and tetracyclic antidepressants, such as imipramine, amitriptyline, clomipramine, doxepin, and maprotiline, are named for their molecular structure. They were first found to alleviate depressive symptoms in the 1950s. Their primary mechanism of action is to block reuptake of NE, 5-HT, and some affect DA reuptake. Although the result is to increase the amount of NE and 5-HT in the synapse, the clinical effect is thought to occur through receptor changes. The prolonged presence of increased, synaptic NE results in desensitization of postsynaptic  $\beta$ -receptors. This state of affairs correlates with the desired clinical effect. In addition, the increased NE also acts to desensitize  $\alpha_2$ -autoreceptors, which results in a further increase of the amount of NE released. Increased 5HT in the synapse seems to increase the sensitivity but reduce the density of postsynaptic receptors. The sensitization time course is also correlated to clinical effect. Tricyclics also affect second-messenger systems and act as cholinergic antagonists, either of which may result in a clinical effect. Tetracyclics may also antagonize postsynaptic 5HT and DA receptors.

Side-effects of tricyclics and tetracyclics are generally due to the anticholinergic and antihistaminergic effects. Common side-effects include dry mouth, constipation, blurry vision, and tachycardia, which are all caused by peripheral anticholinergic properties. Central anticholinergic effects can include memory loss, confusion, and delirium. Sedation is another side-effect and it is due to histamine antagonism. Some people also experience sexual dysfunction and weight gain. More important, the margin of safety for these drugs is relatively small. Doses equivalent to two to three week prescriptions can be lethal. Death usually occurs from cardiac toxicity or seizures. Consequently, tricyclics and tetracyclics are used with extreme care in individuals who are or may become suicidal. Finally, patients must wait two to six weeks to see any clinical effect on symptoms of depression.

The 5-HT selective reuptake inhibitors (SSRIs) currently available in the USA are fluoxetine, paroxetine, and sertraline. Fluoxetine was discovered in the 1970s and was introduced for clinical use in 1988. Since then, it has become the most widely prescribed (and widely researched) antidepressant in the USA. As one might surmise by their name, SSRIs inhibit reuptake of 5-HT. Because they have little to no interaction with cholinergic, histaminergic, or noradrenergic systems, they produce fewer side-effects. Fluoxetine has a long half-life and an active metabolite, which also has a long half-life. In clinical trials, fluoxetine has performed as



well as or better than tricyclic antidepressants in relieving symptoms of depression.

Although fluoxetine has a better side-effect profile, it does produce more nervousness, sleep disturbances, and nausea than tricyclics. Interestingly, it also produces weight loss, which is opposite from the weight gain often seen with tricyclic use and has been useful in treating obesity. Fluoxetine is also sedating, at rates similar to the tricyclic antidepressants. In spite of lay reports, large clinical trials show no greater risk of suicide or suicide attempts over that seen with other antidepressants. Some have suggested that the activating effects (restlessness and agitation) may cause some susceptible people with a history of suicidal ideation to attempt suicide. Therefore, any patient who exhibits increased activity and feelings of restlessness should be monitored carefully. In contrast to the tricyclics, higher doses of SSRIs do not appear to alleviate symptoms of depression better. In fact, some clinical trials suggest that patients may do less well on higher doses. Other antidepressives include mianserin and iprindole. Newer types of antidepressive act to alter the linkage between the adrenergic and/or serotonergic NT and second-messenger systems, for example, Rolipram, which inhibits cAMP phosphodiesterase.

#### 1.06.6.2 Mood Disorders: Bipolar Disorders

Bipolar disorders (both Bipolar I and Bipolar II) have been effectively treated with medication for nearly 50 years. People with bipolar disorder experience alternating periods of depression and mania. Lithium is the most common psychopharmacological treatment, but other drugs have also been found useful. Lithium is effective in approximately 70–80% of patients presenting with acute mania. The mechanism by which lithium exerts its therapeutic effect is not yet clear, but possibly it enhances the activity of the 5-HT system. Some evidence shows that lithium increases the release of NE but also reduces its postsynaptic receptor sensitivity. However, since lithium affects most NT systems and signal transduction pathways, conclusions cannot yet be drawn as to its mechanism of action. Lithium appears to block an enzyme of the intracellular second-messenger system (inositol phosphate), resulting in a reduced responsiveness in those neurons that depend on such second-messenger linkages (Kandel et al., 1991).

Lithium's side-effects include confusion and memory problems and loss of some motor coordination and development of a slight tremor. It can also induce hypothyroidism. It must be

noted that lithium has a small therapeutic window, with high serum levels causing neurotoxicity. Symptoms of lithium's neurotoxic effects include delirium, ataxia, and seizures. Toxicity can be fatal if not treated immediately. Lithium is also teratogenic, so should be used in pregnant women with extreme care.

Lithium is used to treat acute manic episodes, but its effect is not fully realized for five to ten days. As a result, acute episodes are often treated with BZDs or neuroleptics as short-term adjuncts to lithium. Lithium's primary usefulness, however, is in prophylaxis of manic and depressive episodes. Patients who experience mixed episodes or rapid cycling or have comorbid substance abuse are less likely to respond well to lithium. Patients with bipolar disorder also respond well to treatment with the anticonvulsant drugs, valproate and carbamazepine. MAO inhibitors, tricyclics, and SSRIs have also been found useful, particularly to treat the depressive episodes. Antidepressants are used most often as adjunctive treatments or with patients who have not responded well to lithium alone. Further, lithium can produce many side-effects (see Gelenberg & Bassuk, 1997).

#### 1.06.6.3 Anxiety Disorders: Generalized Anxiety Disorder

While depression and its treatment are separated from anxiety and its treatment, most forms of anxiety disorders used to be lumped together under the heading of generalized anxiety disorder (GAD). Anxiety was seen as a normal emotion to some threatening situation (real or imagined). However, since the 1960s, research has principally focused on the anxiety subtypes described below. As happens with social phobia (see below), a clear research focus on the biology of GAD has yet to develop. For many years, GAD has been treated with BZDs, which continue to be the first drug of choice for these patients. However, recent concern about long-term treatment with BZDs has prompted a search for other psychopharmacological interventions. As a result, clinicians welcomed the introduction of buspirone, a non-BZD anxiolytic. Buspirone does not produce dependence and can be withdrawn with relative ease. Although buspirone is effective in treating GAD symptoms, it has a slow onset of action and may not be accepted by patients who want immediate symptom relief. Some studies also show that tricyclic antidepressants and SSRIs may be helpful for patients with GAD. As with depression, many organic conditions can cause anxiety states, including endocrine disorders (e.g., hyperthyroidism), metabolic disorders

(e.g., hypoglycemia), and drugs or medications (e.g., caffeine or withdrawal from addictive drugs).

#### **1.06.6.4 Anxiety Disorders: Panic Disorder**

##### ***1.06.6.4.1 Phenomenology and etiology of panic disorder***

Panic disorder (PD) is the best researched anxiety disorder. Twin studies have found higher concordance in monozygotic twins than dizygotic twins, suggesting that PD is heritable. In truth, we have only limited understanding of PD and its biological basis. One current theory is the respiratory hypothesis, the formulation of which stemmed from the discovery that administration of sodium lactate could induce panic attacks that were identical or very similar to spontaneously occurring panic attacks. Researchers found that lactate induces panic attacks in 50–70% of people with PD, but only 10% of people without PD. Later studies have not found any direct CNS effect of lactate, which is primarily a respiratory stimulant. Also, a correlation exists between hyperventilation produced by lactate and the induction of panic attacks.

Common symptoms of panic attack include shortness of breath, choking feelings, and trouble breathing. Voluntary hyperventilation induces panic attacks in 30–50% of people with PD, and inhalation of carbon dioxide-enriched air induces panic attacks in 50–80% of people with PD. The respiratory hypothesis suggests that PDs stem primarily from the increased sensitivity of the central CO<sub>2</sub> receptors. Activation of these receptors leads to a “false suffocation alarm,” and triggers the psychological symptoms of anxiety. This hypothesis would suggest a purely psychological intervention of breathing retraining, physiological symptom exposure, and cognitive evaluation of the false suffocation alarm. In fact, cognitive-behavioral therapy reduces vulnerability to lactate-induced panic and appears to have short-term and long-term effects on the reduction of spontaneous panic attacks.

##### ***1.06.6.4.2 Neuropharmacology of panic disorder***

However, some NT systems have been implicated in PD. Some researchers have found that patients with PD are hyperreactive to yohimbine (an  $\alpha_2$  NE receptor antagonist), while others have found patients to be hyporeactive to clonidine (an  $\alpha_2$  NE receptor agonist). These findings have led to the NE “dysregulation” hypothesis. Researchers have found reduced  $\alpha_2$  NE receptor binding sites and a blunted growth hormone response to cloni-

dine, which suggests  $\alpha_2$  NE receptor down-regulation. 5-HT has also been suggested as a possible mechanism for PD. Administration of m-chlorophenylpiperazine (m-CPP), which is a 5-HT agonist, in low doses showed a greater rate of panic induction in PD patients than in nonpatients. This might suggest a postsynaptic receptor supersensitivity. When this discovery is coupled with the suggestion that benzodiazepine receptor sensitivity is reduced, 5-HT is thought by some to be the primary route of PD development. However, the topic clearly requires more research.

##### ***1.06.6.4.3 Drug therapies for panic disorder***

Psychopharmacological treatment of PD is common and often combined with psychotherapy. High-potency BZDs such as alprazolam and clonazepam have been used with good success. Although they were originally prescribed simply to relieve anticipatory anxiety, later research has suggested that they may function to reduce the number and intensity of the panic attacks themselves. BZDs generally produce symptom relief within a week, a benefit not shared with other medications. Antidepressants also used include the tricyclic imipramine, the tetracyclic clomipramine, and MAO inhibitors. The SSRI fluvoxamine has been found effective, although it is not currently available in the USA. Antidepressants are thought to exert their action by their 5-HT reuptake inhibition, although research has not yet borne this out. In contrast to the BZDs, antidepressants must be administered for three to six weeks to see any improvement. No psychopharmacological treatment to date seems helpful in alleviating symptoms of agoraphobia.

#### **1.06.6.5 Anxiety Disorders: Obsessive-compulsive Disorder**

For many years, clinicians were frustrated by the lack of effective psychopharmacological treatments for patients with obsessive-compulsive disorder (OCD). Then scientists discovered that clomipramine could decrease OCD symptoms, and it has become the drug of choice. Until then, clinicians had few clues for how OCD developed or what NT systems were involved. Twin and family studies have not supported the heritability theory of OCD. With the discovery of clomipramine's effectiveness, researchers developed the 5-HT hypothesis. This theory is based on the positive results of 5-HT-enhancing drugs, but additional research has found that OCD patients have increased turnover of 5-HT and its metabolites (5-HIAA).

In addition, the 5-HT agonist m-CPP exacerbates symptoms of OCD. Some researchers have found that pretreatment with clomipramine blocks the m-CPP-induced exacerbation, although others have not always been able to replicate this phenomenon.

The 5-HT system dysregulation also supports a second theory that suggests that patients with OCD suffer striatal damage. Clinically, OCD shares certain characteristics with tic disorders (i.e., uncontrollable repetitive behaviors), and tic disorders are believed to be caused by striatal dysfunction. OCD has also developed in patients with Huntington's, which involves lesions in the striatum. Interestingly, the pathway from the striatum to the thalamus and cortex is heavily dependent on 5-HT, which could explain the effectiveness of 5-HT-enhancing drugs. As mentioned above, 5-HT is primarily inhibitory and the neurons in the striatum appear to inhibit activity downstream after the thalamus. The striatal damage hypothesis postulates that the loss of striatal neurons would increase activity in the thalamocortical pathways. In fact, positron-emission tomography (PET) scans show increased glucose utilization in the caudate and orbitofrontal regions of OCD patients. Other studies have shown a reduction in metabolism in those areas after treatment with clomipramine, fluoxetine, or behavior therapy. In severe cases of OCD, psychosurgery lesioning of pathways from the thalamus to the orbitofrontal areas appears to alleviate symptoms. Thus, the evidence suggests a strong influence of 5-HT in the genesis of OCD.

As mentioned above, psychopharmacological treatment of OCD relies heavily on the use of 5-HT-enhancing drugs such as clomipramine and SSRIs. Over the years, clinicians have tried several other medications, but the 5-HT-enhancing drugs are the only ones currently recommended for use with people who have OCD. Doses higher than those used for depression are usually necessary. In a few cases, the disorder does not respond to any treatment. For these individuals, psychosurgery may be recommended to alleviate their symptoms. The most common surgery involves making lesions in the cingulate cortex on both sides of the brain.

#### **1.06.6.6 Anxiety Disorders: Post-traumatic Stress Disorder**

Research on post-traumatic stress disorder (PTSD) has primarily been with individuals who have combat-related PTSD, although other traumas do induce the disorder. The current theory of PTSD states that patients must show autonomic hyperresponsivity. This

hyperresponsivity manifests itself in three ways. First, patients respond to reminders of their trauma (e.g., imagery, sounds, videos, or pictures) with increased blood pressure, heart rate, muscle tension, and other physiological indicators of stress. Second, patients also respond to trauma reminders with increased neuroendocrine (epinephrine and norepinephrine) activity. Third, patients have demonstrable HPA axis dysfunction. Acute stress increases cortisol release, and the chronic stress experienced in PTSD appears to result in high cortisol levels for a long period. High cortisol levels appear to damage the hippocampus, which would suggest that PTSD patients might experience neuroanatomical changes. People with PTSD have increased density of glucocorticoid receptors, which results in exaggerated negative feedback of ACTH and cortisol. PTSD patients also supersuppress in response to the DST, which further suggests an exaggerated negative feedback system. All the evidence to date points to PTSD being a disorder of hyperresponsivity.

Unfortunately, researchers have not yet been able to find a psychopharmacological treatment to correct for the autonomic hyperresponsivity. As a result, clinicians continue to rely on medications to treat the emotional and subjective symptoms of PTSD. The most common drugs prescribed are the tricyclic antidepressants, especially amitriptyline and imipramine. Researchers have had mixed results with these drugs, but they appear to have some effect beyond their antidepressive and anti-anxiety effects. Other drugs, such as the MAO inhibitor phenelzine, SSRIs, and carbamazepine have been used with varying success. Overall, research suggests that the positive symptoms of PTSD (e.g., increased arousal, re-experiencing) often respond to psychopharmacological treatment while negative symptoms (e.g., avoidance) do not.

#### **1.06.6.7 Anxiety Disorders: Social Phobia**

We know little about the biological basis of social phobia (SP). Some researchers have suggested that the sympathetic nervous system may be dysregulated, because people with SP report autonomic activation symptoms, and  $\beta$ -blockers such as propranolol sometimes help. In the late 1990s, however, a sustained research focus in the biology of SP does not exist. Administration of phenelzine, a MAO inhibitor, alleviates nervousness and fear of criticism for some people with SP. BZDs have also been helpful. Nevertheless, in psychopharmacological treatment, as in the etiology of SP, none of

the choices are clearly superior. In fact, some research suggests that cognitive-behavioral treatment may be the best treatment choice.

### 1.06.6.8 Psychotic Disorders: Schizophrenia

#### 1.06.6.8.1 Characteristics of psychosis

Psychosis is one of the oldest psychiatric syndromes (mixture of symptoms) but is not a specific diagnostic category in the *Diagnostic and statistical manual of mental disorders* (4th ed., *DSM-IV*) or *International classification of diseases* (10th ed., *ICD-10*) (Stahl, 1996). Psychotic symptoms include perceptual distortions (delusions, hallucinations), disorganized speech and behavior, severe distortions in testing reality, and motor disturbances (peculiar postures and stereotyped movements and acts). Mania, depression, substance abuse, and cognitive disorders like Alzheimer's dementia may present with psychotic features. Finally, psychoses can be described as paranoid (delusional beliefs and projections, hostile belligerence, and/or grandiose expansiveness), disorganized or excited, and/or depressive (retardation, apathy, self-blame). Finally, one should recognize that many organic conditions may produce or be associated with psychotic symptoms, for example, space-occupying CNS lesions, cerebral hypoxia, neurological disorders like temporal lobe epilepsy, cerebral vascular disorders, CNS infections like meningitis, metabolic and endocrine disorders like thyroid disease, nutritional deficiencies (thiamine in Wernicke-Korsakoff syndrome), and drug, medications or toxic agents like amphetamines or corticosteroids.

Medications are generally administered to any person who exhibits psychotic symptoms, including people with schizophrenia, schizophreniform disorder, schizoaffective disorder, delusional disorder, brief psychotic disorder, and severe depressive disorders with psychotic symptoms. Some newer research examines etiology and treatment of nonschizophrenic psychotic disorders, but little is known yet.

#### 1.06.6.8.2 The phenomenology and biological basis of schizophrenia

Schizophrenia was first described in the nineteenth century by Benedict-Augustin Morel, who termed it *démence précoce* (Kaplan & Sadock, 1996). Schizophrenia is the most common (1% of the population) and, fortunately, the most well-understood psychotic disorder, but studies suggest that other psychotic disorders have similar etiologies. The symptom profile of schizophrenia really does have two faces. On the one side is the array of

positive symptoms suggesting an exacerbation of normal functions (delusions, hallucinations, disorganized speech and behaviors, agitation, and bizarre behaviors). On the other side is the cluster of negative features suggesting a loss of normal function (flattened affect, alogia or reduced fluency of thought and speech, anhedonia, avolition or reduced goal-directed behavior, and poor attention). Finally, schizophrenia is characterized by a significant genetic heritability. Twin studies find monozygotic concordance rates of 30–80%, the highest found in any psychiatric disorder.

Fortunately, research on schizophrenia and theories as to its cause abound. One such theory proposes that schizophrenia may be related to neuroanatomical lesions common to people with the disorder. Neuropsychological tests have revealed deficits in prefrontal cortex and medial temporal lobe function, showing a lack of ability to plan and organize. Many schizophrenics also have more generalized deficits, including enlarged lateral and third ventricles and deeper sulci than people without the disorder. The ventricular enlargement is correlated with greater impairment in functioning and a poorer prognosis, although some have disputed this conclusion.

#### 1.06.6.8.3 The DA hypothesis of schizophrenia

By far the most popular theory of schizophrenia and selected psychotic disorders is the dopaminergic hypothesis. This theory states that schizophrenia is caused by excessive central DA activity. In some people with the disorder, the striatum and nucleus accumbens, which are heavily dependent on DA, exhibit normal volume and area but a reduced neuronal diameter. Further, both areas show increased binding of the DA antagonists haloperidol and spiperone. The substantia nigra does manifest reduced volume, and postmortem studies reveal increased density of D<sub>2</sub> receptors in the caudate and putamen, while the density of the DA transporter and D<sub>1</sub> receptors remain normal. Some evidence suggests the existence of some structural abnormalities in DA receptors and an abnormal linkage between D<sub>1</sub> and D<sub>2</sub> receptors, but these findings are not well-replicated.

Antipsychotics block DA and reduce its function, which supports the dopaminergic hypothesis, but it is not certain that this is the mechanism by which the drugs relieve symptoms. Unmedicated schizophrenics do not show higher levels of HVA, the major metabolite of DA in cerebrospinal fluid, when compared with nonschizophrenics. Medicated schizophrenics do have higher HVA levels, which suggests that antipsychotics actually do work on DA neurons.

Long-term treatment with antipsychotics produces tolerance in nigrostriatal pathways, but not in mesocortical or mesolimbic pathways. In postmortems of schizophrenics on chronic antipsychotics, increased HVA was found in cingulate and frontal cortices (mesocortical and mesolimbic areas), but not in the putamen and nucleus accumbens (nigrostriatal system). Although many studies have found increased numbers of DA receptors in postmortem schizophrenic brains, it is not clear whether these are from the disease or the treatment. Some have suggested that an abnormality in the DA systems projecting to the nucleus accumbens may mediate symptoms like anhedonia, while DA systems projecting to the central nucleus of the amygdala may participate in the generation of paranoid ideation.

#### **1.06.6.8.4 Antipsychotic medications**

The primary psychopharmacological treatment for schizophrenia and other psychotic disorders is the use of DA antagonists, otherwise known as antipsychotics. The older typical antipsychotics and the newer “atypical” antipsychotics differ based on their pharmacological mechanism and on when they were found effective. Chlorpromazine, the first antipsychotic, was discovered in 1952. The class of conventional or typical antipsychotics is butyrophenones and phenothiazines, but this category also includes thioxanthenes, dihydroindolones, and dibenzoxazepines. Their DA antagonist properties were discovered when they were found to reduce the behaviors caused by administering dopamine agonists to both humans and animals. The clinical efficacy of typical antipsychotics is directly correlated to the drugs’ affinity for the D<sub>2</sub> receptor. Later studies show that for an antipsychotic to work, it must effectively bind the receptor and produce a subsequent decrease in DA activity. Nevertheless, both butyrophenones and phenothiazines affect NE and DA systems. Also, both drugs increase DA turnover, presumably because their blockade of both postsynaptic DA receptors and presynaptic DA autoreceptors produces a feedback activation of these neurons.

Antipsychotic drugs are usually administered orally, but can also be given by depot (slow-release) injection. Medications can be given to treat a psychotic episode, but are more commonly used as maintenance therapy to prevent relapses. Antipsychotics are strong medications and can induce a range of side-effects. Medication effects can include sedation and a lack of interest in one’s environment (ataraxia). Antipsychotics also increase prolactin secretion,

which can result in breast development in men and milk secretion and menstrual abnormalities in women.

#### **1.06.6.8.5 Antipsychotic drug side-effects**

All typical antipsychotics also produce extrapyramidal symptoms (EPS) as a side-effect. Extrapyramidal refers to the localization of the motor dysfunction in the spinal tracts. EPS are motor problems that include Parkinsonian-like symptoms (stiffness, tremor, shuffling gait), acute dystonia (abrupt spasms of head and neck), and akathisia (physical restlessness). Unfortunately, the extent to which the drug works as a DA antagonist correlates with the extent of EPS. So anti-Parkinsonian drugs are often administered to help reduce these symptoms. Tardive dyskinesia (TD) develops in 10–20% of patients on antipsychotics for more than one year. TD is a movement disorder characterized by mouth, tongue, and face movements, besides slow, writhing movements of the limbs and trunk. The elderly and those with affective disorders are particularly vulnerable to developing TD. It was once thought that TD was progressive and irreversible, but this is no longer thought to be true.

Neuroleptic malignant syndrome (NMS) is an uncommon, but potentially fatal reaction to antipsychotics. It is characterized by severe muscular rigidity, autonomic instability, high temperature, and changing levels of consciousness. It happens more often when high-potency antipsychotics are given in high doses and escalated rapidly, and is more common in males and younger patients.

#### **1.06.6.8.6 Side-effect profile of newer antipsychotics**

Clozapine is the most common atypical antipsychotic currently used in the United States. Risperidone is also approved and is gaining popularity, and raclopride is still in the clinical trial phase of FDA approval. Atypical antipsychotics are so named because they appear to involve DA receptors other than D<sub>2</sub>. Clozapine seems to affect neurons only in the ventral tegmental area and not in the substantia nigra, which is thought to be the source of the EPS and TD symptoms. This drug also has an affinity for 5HT neurons, which may be involved in its function. In fact, Clozapine has a “cocktail” of NT actions, affecting up to nine different receptors (Stahl, 1996).

Clozapine is noted for the rarity of patients developing EPS and TD while on it. NMS also occurs at lower rates than with typical antipsychotics. However, 1–2% of patients develop

agranulocytosis (a loss of a particular group of white blood cells), usually within the first six months of use. This can be a fatal condition and, to monitor it, white blood cell counts must be checked weekly. Seizures are another significant side-effect. Because of the potentially serious side-effects, Clozapine is currently approved for use in treatment-refractory schizophrenia, patients with unmanageable EPS, and patients with TD.

Other atypical agents (Julien, 1997) include molindone (Moban), which resembles 5-HT and appears effective for schizophrenia with only moderate sedative and EPS profiles. Loxapine, structurally related to Clozaril, has antipsychotic properties but moderate sedative and EPS profiles and lowers convulsion thresholds. Carbamazepine (Tegretol) is not effective as an antischizophrenia drug. The interested reader should refer to Stahl's summary of some current discovery strategies for new antipsychotics (Stahl, 1996).

### 1.06.6.9 Substance Abuse and Dependence Disorders

#### 1.06.6.9.1 Definitions and diagnostic criteria

Stahl (1996) outlines the major *DSM-IV* criteria for substance intoxication, abuse, and dependence. Intoxication refers to a reversible pattern of behavioral or psychological changes produced by the drugs' action on the CNS (e.g., mood, belligerence, impaired cognition, judgment, motor skill performance, and social functioning). Substance abuse refers to a maladaptive pattern of using any drug in "culturally disapproved manner" that leads to adverse consequences. The latter include failure to meet obligations, placing self and others in harmful situations, legal difficulties related to drug taking, and social or interpersonal problems. Substance dependence refers to a physiological state of adaptation produced by a prolonged maladaptive pattern of substance use. The *DSM-IV* definition of substance dependence includes three or more of the following characteristics: the development of tolerance (the need for more drug to get the same effect), a drug withdrawal syndrome (usually a drug rebound state opposite to the general effects of the drug), inability to control the amount consumed or to stop the consumption, the subordination of other life activities to drug taking, and/or the persistence of drug taking despite recurring adverse consequences (see Section 1.06.5.4). Relapse refers to recurrence of drug taking after some effective treatment regimen. Other concepts introduced earlier are relevant to this topic, for example,

cross-tolerance and cross-dependence (substituting a second drug to substitute for or block withdrawal from the first one). The latter effects may in part account for the phenomena of polysubstance abuse. The reader should take note that addiction itself is not defined in the *DSM-IV*. We like the following one: "a behavioral pattern of drug abuse characterized by an overwhelming involvement with the compulsive use of a drug, the securing of its supply, and a high tendency to relapse after its discontinuation" (Stahl, 1996, p. 336).

#### 1.06.6.9.2 Models of addiction

Of all the psychiatric disorders, those in this category are as much socially as physiologically defined. Marlatt (1992) in fact describes four models based on whether individuals are or are not responsible for developing the addiction and for changing their addictive behaviors. The "medical" or "addiction as a disease" model most readily lends itself to a discussion of psychopharmacological interventions. Most addiction researchers suggest that a drug's potential for abuse rests on its potential to act as a reinforcer and/or to induce euphoria (see Section 1.06.5.5.4 on reinforcing and reward properties of drugs). The latter effects derive from the drug's action on certain mesolimbic brain systems (specifically, the DA neurons from the ventral tegmental area [VTA] innervating the nucleus accumbens) that are involved in normal motivational processes (see Kalivas & Samson, 1992; Wise 1990). Proponents of this view cite the multiple NT systems that participate in this brain region (e.g., GABA<sub>A</sub>, nicotinic cholinergic, serotonergic, and opioid systems). A key concept in this model is that it is a psychopharmacological model, that is, both the drug and the drug self-administration are equally important in sustaining the larger pattern of drug-seeking behaviors. Some biological theories of addiction suggest that genetic differences in sensitivity to drugs may reflect the under- or overexpression of the latter NT receptors, among others (see Crabbe & Harris 1991). For example, underexpression of one or more of the key NTs involved in the mesolimbic reward system might promote excessive use of drugs that affect such NTs (self-medication?). This idea is the same as Blum's "reward deficiency syndrome" (Blum, Cull, Braverman, & Comings, 1996).

#### 1.06.6.9.3 Psychopharmacology of and pharmacotherapy for drugs of abuse

The following sections will describe some attempts to treat some kinds of drug abuse with drugs. Of course, a key to this discussion is what

is being treated and what neuropharmacological systems are involved. The treatment of acute drug intoxication and drug withdrawal reactions is straightforward. The approaches to treating the drug-seeking behavior or drug craving may be a little less obvious. A classic strategy would be to arrange for the drug-taking behavior to be punished, for example, disulfiram or Antabuse (alcohol consumption with Antabuse makes one sick). Another strategy involves pharmacologically blocking the drug's CNS actions, for example, using naltraxone (an opiate receptor antagonist) to counteract heroin overdoses. A final strategy would be to substitute another drug or another form of the abused drug, for example, methadone (a synthetic opiate agonist) maintenance for heroin addicts or nicotine gum for addicted smokers. Apart from treatment of the addiction, medications may be used to treat acute intoxication or overdose, withdrawal reactions, or drug-induced psychosis. The interested reader should examine Schuckit's fine clinical guide to the treatment of drug and alcohol abuse (Schuckit, 1995) or the *Practitioner's guide to psychoactive drugs* (Gelenberg & Bassuk, 1997). Both volumes also review the psychopharmacological profile for all the major drugs of abuse.

#### 1.06.6.9.4 Alcoholism

About 70% of the US adult population consumes alcohol. Problem drinkers number about 10 million and half of these may be physically dependent. Risks for developing alcoholism include sociodemographic factors (lower socioeconomic status, ethnicity, being divorced, nationality), genetic factors (being male, family history of alcoholism), and individual history factors (younger age, anti-social personality, school and conduct problems). Chronic heavy drinking, of course, can lead to certain forms of CNS damage, including Wernicke's disease, Korsakoff's psychosis, and Wernicke-Korsakoff syndrome. Postmortem studies of the latter patients show lesions in the mammillary bodies of the hypothalamus. These disorders appear to be produced by a B<sub>1</sub> vitamin deficiency and are treated with thiamine. Further, alcohol can produce profound CNS or other organ systems during development, for example, the fetal alcohol syndrome.

Alcohol (ethyl alcohol or ethanol), when absorbed, uniformly reached all tissues in the body, including the CNS. Besides the CNS damage, many other organ systems may be compromised by chronic alcohol, for example, the liver and heart. While an alcohol receptor may not exist, several NT systems may mediate some direct acute or chronic effects of alcohol

and its acute (hangover) or chronic withdrawal syndrome. Alcohol enhances inhibitory GABA<sub>A</sub> neurotransmission, promotes the release of DA in mesolimbic pathways associated with reinforcement and pleasure, decreases how much 5-HT is stored in neurons of the dorsal raphe nucleus, and appears to reduce excitatory neurotransmission at the NMDA form of the glutamate receptor. The liver converts alcohol to acetaldehyde (enzyme-alcohol dehydrogenase), which may promote the release of and interaction with CAs from stores in the adrenals and in the brain. Apparently acetaldehyde and biogenic amines chemically interact to produce opiate-like substances (tetrahydroisoquinolines, TIQs, and beta-carbolines). TIQs appear to promote DA release in the nucleus accumbens (Robert Myers, personal communication) and the beta-carbolines may act as inverse agonists at the benzodiazepine site on GABA<sub>A</sub> receptors. Such an inverse agonist action would reduce the impact of drugs that enhance GABA<sub>A</sub> activity, for example, BZDs, barbiturates, and alcohol. The latter effect is interesting since animal research suggests that the GABA<sub>A</sub> system of animals more likely to self-administer alcohol is less sensitive to the effects of alcohol (Crabbe & Harris, 1991).

Given the opponent-process model of drug rebound and the array of direct CNS effects of alcohol, it is not surprising that alcohol withdrawal signs include excitation, anxiety, and discomfort (note these signs are opposite from alcohol's direct sedative, calming, and euphoric effects). Other characteristics of the prolonged alcohol withdrawal syndrome in physically dependent individuals include insomnia, tremulousness, muscle tension, facial flushing, and sweating, followed by nausea, vomiting, abdominal cramps, hyperreflexia, and anorexia. Later during withdrawal, tremor, seizures, and hallucinations may develop, followed sometimes by delirium tremens (confusion, hallucinations, disorientation, agitation, and delirium). Sedative-hypnotics, like barbiturates and BZDs, have been traditionally used to treat the withdrawal syndrome, and more recently alpha- and beta-adrenergic blockers. The persistent hallucinations after prolonged drinking bouts probably represent an organic condition that can be treated with antipsychotic drugs like the phenothiazines or butyrophenones.

Until recently, pharmacological treatment for alcoholism itself has been limited to disulfiram (Antabuse). That drug blocks the metabolism of acetaldehyde, producing a buildup of acetaldehyde and a plethora of unpleasant autonomic, somatic, subjective and CNS effects. This treatment is generally ineffective because of poor compliance (about 20% remain alcohol-free for

a year). In cases of dual-diagnosed alcoholics, treatment of the psychiatric component may improve the substance as well, for example, lithium in bipolar alcoholics. Newer developments include the use of naltrexone to reduce alcohol "craving." The latter is thought to work through the endogenous opiate system interactions with the mesolimbic DA reward system. Similarly, drugs that boost brain 5-HT levels (e.g., SSRIs) have a modest effect in reducing alcohol consumption. Alcoholics receiving buspirone, a partial 5-HT<sub>1A</sub> agonist, show reduced consumption and anxiety, but further research is needed. Other compounds that may be useful in reducing drinking and/or craving include (Bloom & Kupfer, 1995): acamprosate (a direct GABA receptor agonist), bromocriptine (a nonspecific dopamine-2 receptor agonist), tiapride (a dopamine-2 receptor antagonist), and  $\gamma$ -hydroxybutyrate (an endogenous sedative that increases DA levels).

#### **1.06.6.9.5 Cocaine and amphetamine dependence**

Besides marijuana, cocaine and amphetamines represent the most widely abused illegal drugs in the USA. The interested reader can consult any number of references for more details (Gelenberg & Bassuk, 1997; Schuckit, 1995). Both are CNS and cardiovascular stimulants (vasoconstriction), acting in different ways to promote the increase of DA levels, and, to a lesser extent, NE and 5-HT levels. Although cocaine is a local anesthetic, its stimulatory effects derive from its blockade of DA and NE reuptake into the presynaptic neuron. Amphetamine acts primarily to release DA or NE from the presynaptic neuron, but also has reuptake blocking properties. Their abuse potential lies in their activation of the brain reward system, that is, the DA receptors of the mesolimbic nucleus accumbens. Both agents at low to moderate doses produce a positive symptom profile: including euphoria; increased wakefulness, concentration, and alertness; and decreases in fatigue and appetite. However, higher doses can produce a host of negative effects, including tremor, irritability, emotional lability, anxiety, motor tremors, paranoia, panic, and repetitive stereotypes in movements and speech. Overdosing can result in stroke, heart failure, and seizures. The withdrawal profile of these drugs also is similar. Intense craving, agitation, and anxiety are followed by fatigue, by increased sleeping and eating, and by depression, then by listlessness, anhedonia (inability to experience pleasure), decreased interest, and increased drug craving. Longtime cocaine addicts also may show sensitization or reverse tolerance where

even low doses can induce bizarre behavioral reactions that appear similar to those of a paranoid psychosis.

The clinical psychopharmacology of amphetamine and cocaine also is similar (see Schatzberg & Nemeroff, 1995). Acute intoxication with either agent can be treated with DA-blocking drugs like chlorpromazine or haloperidol. Both drugs continue to have legitimate medical uses, for example, cocaine is used in ophthalmology and amphetamines are used to treat narcolepsy, ADHD, and exogenous obesity. However, most of the clinical research on agents to counteract stimulant addiction has focused, with uneven success, on cocaine. One "quasi-substitution" strategy has been to use antidepressive drugs like desipramine, which also block DA reuptake. Only modest reductions in the cocaine relapse rates were found. A similar approach using bromocriptine, a DA agonist, looks promising, but double-blind clinical trials are needed. Finally, carbamazepine, an antiseizure medication, has been tried since cocaine appeared to increase "kindled" seizure activity in animal studies. Unfortunately, double-blind studies have yet to show any reduction in relapses back to cocaine use.

#### **1.06.6.9.6 Nicotine and caffeine dependence**

The nicotine delivered through cigarette smoking acts directly on nicotinic cholinergic receptors in the CNS (Schuckit, 1995). The reinforcing and other actions of nicotine (mood elevation, enhancement of cognitions, psychomotor stimulation, and decreased appetite) are similar to those of cocaine and amphetamine in that all activate mesolimbic DA neurons. However, the "high" produced by smoking is shorter acting since nicotine apparently briefly shuts down the nicotinic receptor shortly after binding to it. Smokers may up-regulate their nicotine receptors to compensate for the frequent "shutdowns." Further, this latter neuroadaptation may be the basis for nicotine tolerance, dependence, and withdrawal (intense craving, agitation, increased appetite) phenomena. Nicotine also appears to increase NE activity, but to decrease 5-HT levels and turnover. At high doses, nicotine can produce toxic effects. The best current pharmacological treatment of smoking addiction is a "tapering-off" substitution regimen using nicotine gum, transdermal patches, and the recently developed nasal spray (the latter is not currently available).

The caffeine found in coffee, tea, cocoa, colas and over-the-counter medication is a member of the class of compounds called xanthines, which also include theophylline (tea, respiratory medications) and theobromine (chocolate).



Caffeine acts to produce stimulatory effects in multiple organ systems, for example, cardiovascular, respiratory, kidney, and CNS. In the brain, caffeine may block GABA and adenosine receptors (activation of these normally acts to inhibit other neurons) and may increase turnover in CA and 5-HT systems (Shuckit, 1995). "Caffeinism," produced by a daily consumption greater than 500–600 mg, presents a set of symptoms resembling panic attacks. Caffeine dependence is evidenced by its withdrawal effects, which include headaches, increased muscle tension, fatigue, irritability, anxiety, and poor concentration. No specific pharmacological treatment is available other than gradually tapering-off caffeinated products.

#### 1.06.6.9.7 *Opiate (narcotic) dependence*

Section 1.06.2 described the neuropharmacology of opiate receptors. Different opiate narcotic drugs have different profiles of agonist and/or antagonist actions at mu, kappa, delta, and sigma receptors (see Julien, 1997). The four general categories of abused opiate drugs are: (i) natural alkaloids of opium (opium, morphine, and codeine), (ii) semisynthetic derivatives of morphine (heroin, hydromorphone or Dilaudid, and oxycodone or Percodan), (iii) nonmorphine-derived synthetics (meperidine or Demerol, methadone, and propoxyphene or Darvon, and (iv) opioid-containing preparations (usually with codeine or paregoric). Most of the latter compounds produce their psychotropic effects by acting on mu opiate receptors, which in turn act on other brainstem systems (e.g., the VTA DA reward pathway). All, to a lesser or greater extent, produce analgesia, euphoria, sedation, respiratory depression, cough suppression, pupillary constriction, nausea and vomiting, and a host of other gastrointestinal symptoms.

Nonmedical opioid use of any kind in the USA ranges from 9% in women to 15% in men (for trying heroin, 0.5% and 2%). The acute effects, described as an orgasmic-like rush, last about a minute, followed by a tranquil state lasting for several hours (drowsiness, apathy, motor retardation, mental clouding, depressed respiration, and mood lability). The pupillary constriction and constipation effects of heroin are the most resistant to tolerance development. Commonly, overdosing and death result from the respiratory depression (naloxone and naltrexone, opiate receptor antagonists, are used to treat overdosing). Of course, the opioid withdrawal syndrome, peaking between 48 and 72 hours after last dose, is opposite in character, that is, anxiety, dilated pupils, diarrhea, insomnia, agitation, runny nose and eyes, sweating, and so forth.

Alpha adrenergic agonists like clonidine are sometimes used to treat the overactivation of the sympathetic nervous system during opiate withdrawal. However, more commonly, another opiate agonist like methadone, Darvon, or Demerol or a mixed agonist–antagonist like buprenorphine, is employed to attenuate the withdrawal symptoms. Pharmacological treatment of the opiate addiction itself employs one of three strategies. First, opiate antagonists, like naloxone or naltrexone, have proven effective in the well-motivated addict but generally these agents present problems in compliance. Substitution therapies involve the maintenance on some prescribed opiate agonists like methadone or LAAM (a long-acting agonist). The rationale for this approach is that the regimen blunts the addict's craving, reducing criminal and other risky behaviors (prostitution, HIV-infected needles). The rate of relapse one year after heroin or methadone detoxification can run as high as 80–90%. A newer approach has been to use the mixed agonist–antagonist, buprenorphine. The rationale here is that the agonist effects will blunt the craving and drug-seeking behaviors and the antagonist effects will blunt the heroin high produced by any relapses. Clinical trials of this compound are underway.

#### 1.06.6.9.8 *Sedative-hypnotic and anxiolytic dependence*

These drug abuse problems involve benzodiazepine (BZD) and non-BZD compounds that, with varying potencies, produce CNS depressant effects, ranging from lethargy and sleepiness to anesthesia, and to death from respiratory and heart depression (see Schuckit, 1995). BZDs, commonly used to treat anxiety and/or withdrawal from other drugs, include chlordiazepoxide (Librium), diazepam (Valium), alprazolam (Xanax), and triazolam (Halcion).

Non-BZD CNS depressants include short-acting barbiturates (used as general anesthetics and rarely abused), longer-acting barbiturates like pentobarbital (used as hypnotics to induce sleep and often abused), and long-acting barbiturates like phenobarbital (used to treat epilepsy and other seizure disorders and not usually abused). Other barbiturate-like drugs (e.g., methaqualone or Quaalude) were introduced as hypnotics that lacked the sleep disturbances produced with barbiturate hypnotics. Unfortunately, some, like Quaalude, are widely abused. Other non-BZD CNS depressants include chloral hydrate (used as a short-acting sedative-hypnotic) and meprobamate (used to treat anxiety), and both have high abuse potential.

The specific mechanisms by which all these drugs exert their CNS depressive actions are not

completely clear, but most are known to suppress all excitable tissue. The mechanism by which some of these drugs exert their anxiolytic, muscle relaxant, and anticonvulsant actions lies in their affinity for BZD or barbiturate receptors on the GABA<sub>A</sub> receptor complex. There they act as allosteric modulators of the chloride ion channel and enhanced inhibitory, GABAergic transmission. Via GABAergic input to the nucleus accumbens, the latter drug actions may produce the reinforcing properties of euphoria or tranquility that lead to abuse of these compounds. Pharmacodynamic tolerance to the latter effects develops quickly with both barbiturates and BZDs, probably through some neuroadaptation involving desensitization of the receptors.

With continuous usage of BZDs or barbiturates and then abrupt cessation, a withdrawal profile characterized as rebound hyperexcitability emerges (see Schuckit, 1995). Barbiturate withdrawal frequently produces seizure, while the BZD withdrawal pattern resembles that of alcohol withdrawal. Not surprising, the treatment of depressant withdrawal symptoms is usually a drug from the same class of drug. Of all the drugs of abuse, depressant drugs pose the most serious problems because of their many medical uses, their potential for abuse, and their potential for overdosing or drug interactions. CNS depressive drugs interact with each other, with alcohol, and with other psychiatric drugs (e.g., tricyclic antidepressives) to produce sedation. Thus, these medications are frequently used in suicide attempts. Fortunately, at least for BZDs, a receptor blocker exists (flumazenil) and can be used for acute BZD intoxication or overdose. Since no effective blocker for non-BZDs exists, other medical means must be considered for barbiturate overdoses (e.g., hemodialysis).

Treatment of depressant, and particularly BZD, addiction is complicated by the fact that the drug is often prescribed for anxiety. Thus, besides BZD withdrawal, the possibility of rebound-anxiety exists. An alternative strategy would be to taper off BZDs, slowly and in small steps. However, the latter approach may be problematic when other factors are present, for example, underlying anxiety disorder, any other drug use (including caffeine, nicotine, and alcohol), premenstrual syndrome, life stressors, or other medical conditions (Stahl, 1996).

#### **1.06.6.9.9 Marijuana abuse**

Marijuana and hashish (from the flowering tops of the marijuana plant) derive their effects from the plant's most active ingredient, THC (delta-9-tetrahydrocannabinol). Use of THC

dates back almost three millennia and today it is the most frequently used illegal drug. In the 1970s and 1980s, more than 50% of high school seniors used marijuana at least once. Marijuana usually has both sedative and stimulatory acute effects. These include euphoria (including a sense of well-being, relaxation, and friendliness), changes in one's level of consciousness without hallucinations (loss of temporal awareness), feelings of heightened sexual arousal and hunger, mental slowing and sleepiness, and short-term memory loss. However, at high doses, it can produce panic, delirium, and sometimes psychosis. Chronic marijuana use is associated with an amotivational syndrome (decreased drive and ambition), impairment of social and/or performance skills (i.e., poor communication, ineffective interpersonal interactions, and introversion, and/or poor judgment, inattention, and distractibility), and a decline in personal habits and insight (Schuckit, 1995; Stahl, 1996).

Tolerance may develop to THC, and sudden cessation of THC after chronic use may produce withdrawal symptoms, including: a hyperarousal profile (insomnia, anxiety, restlessness, photophobia, and irritability), somatic and autonomic symptoms (myalgia, chills, sweating, diarrhea, yawning), and a mixed depressive profile (depression, craving, anergy, and mental confusion) (Gelenberg & Bassuk, 1997).

THC can be administered any number of ways (though usually smoked). However, its complete elimination may be slow (up to a week or more) since both THC and its metabolites are sequestered in fat deposits for later release. The mechanism by which THC produces its effects is still poorly understood, but some data suggest activation of the septal area of the brain's limbic system (known for its involvement in emotion). The acute memory impairment and long-lasting EEG changes also suggest clear CNS actions. Some evidence suggests that the brain contains THC receptors and perhaps endogenous THC-like ligands. In any event, no clear medication treatment has been tried for THC abuse, other than detoxification and long-term abstinence. Treatment of symptoms includes anxiolytics for anxiety, panic, or flashbacks and antipsychotics for the occasional toxic delirium, psychotic reactions, or hallucinations (Schuckit, 1995).

#### **1.06.6.9.10 Abuse of hallucinogens and related drugs**

Hallucinogens include a group of drugs that produce intoxicating effects, described as "highs," characterized by enhanced sensory and perceptual experiences (both exteroceptive and interoceptive) (Stahl, 1996). Sometimes,

these effects result in visual illusions and hallucinations. The hallucinations may be psychedelic (i.e., enhanced sensory awareness, “mind expansion,” and feelings of oneness with a larger “other”) or psychotomimetic (i.e., superficially resembling a psychosis). These drugs produce visual hallucinations and strong emotional reactions, whereas schizophrenia is more typically associated with auditory hallucinations and flat affect (Schuckit, 1995). Initially, the latter symptoms can occur without consciousness changes or confusion. However, the intoxication can progress from a confusional state (disorientation and agitation) to anxiety, depression, and panic, and to toxic delirium and the psychotic symptoms of delusions and paranoia. The panic-like state can include physiological and subjective effects like anxiety and feelings of mind loss. Even after cessation of use, flashbacks may occur.

Schuckit (1995) described four general categories of hallucinogenic agents. Indolealkamines like LSD or lysergic acid diethylamide (a synthetic drug) and Psilocybin (an extract of a mushroom plant) appear to act as partial agonists at 5-HT receptors, particularly 5-HT<sub>2</sub>. Phenylethylamine drugs, like mescaline or peyote, and phenylisopropylamine drugs (so called “designer drugs”), like methylene dioxymethamphetamine (MDMA or “ecstasy”), have structures and actions more similar to biogenic amines and amphetamine. Finally, a group of related drugs also can produce hallucinations; these include phencyclidine (PCP or “angel dust”) and inhalants (e.g., amyl or butyl nitrites). PCP appears to have a specific mechanism of action, that is, it acts allosterically to block the NMDA type of glutamate receptor by reducing calcium ion influx into the cell. The latter action disrupts normal and necessary excitatory neurotransmission, thereby producing skeletomuscular effects (besides other effects described above) that include catatonia (excitement alternating with catalepsy and stupor), motor seizures, and muscle breakdown. Other commonly abused inhalants include glues, solvents, aerosols, cleaning solutions, and gasoline. While these substances can produce euphoria and lightheadedness, overdosing can produce loss of normal cardiac function, hypoxia, seizures, and death. Tolerance to the effects of inhalants develops rapidly, and hallucinations can occur with abrupt discontinuation after prolonged use. Chronic inhalant abuse is thought to produce brain damage associated with impairment of higher cognitive functions.

Medical management of hallucinations is generally the same as for psychosis, that is, use of antipsychotics. None of the drugs in this

class have any current medical use. Some patterns of abuse may be short-lived because of the unpleasant, dysphoric effects of the drugs. However, some drugs, like PCP, are associated with a very treatment-resistant pattern of drug use.

#### ***1.06.6.9.11 Over-the-counter and prescribed drugs***

We conclude this section by indicating that many legal drugs can be abused. These include analgesics, antihistamines, cold and cough remedies, sleep medications, and caffeinated products. Some research shows that certain combinations of these products produced subjective effects similar to those produced by cocaine, amphetamine, and morphine.

#### **1.06.6.10 Eating Disorders**

There are many theories to explain the etiology of anorexia nervosa and bulimia nervosa, ranging from cultural to psychological to biological, but none have emerged as dominant in the field. Most of the biologically based theories hypothesize that people with eating disorders lack certain mechanisms related to feeding, such as appetite, satiety, and desire for balanced nutritional intake. Because depression is so often associated with eating disorders, however, the first medications used were antidepressants.

Antidepressants continue to be the psychopharmacological treatment of choice. Both the traditional tricyclics, such as imipramine and desipramine, and the SSRIs, particularly fluoxetine, have been useful. Recent studies suggest that both types of antidepressants relieve depressive symptoms and eating disorder symptoms in both anorexia and bulimia. The primary outcome measure in people with anorexia is weight gain. Although tricyclics may cause weight gain in other patients and SSRIs may cause weight loss, anorectics taking either class of drug gain weight at equivalent rates. Bulimics also respond well to either drug, although as measured by different outcomes, such as bingeing and purging behaviors. It is not clear how two classes of drugs so dissimilar in their mechanism of action can produce such similar results.

Studies of other medications are underway. Some researchers who postulate that eating disorders are similar to opiate dependence have used naltrexone with some success. Others are investigating endocrine responses in eating disordered patients, with plans to develop and test new medications in the future.

### 1.06.6.11 Neurological Disorders

Because of limitations of space, not all neurological disorders can be covered in this section. However, the reader should be aware that many disorders are age-related. Further, medication in elderly individuals presents a unique set of problems and concerns. The reader is directed to a chapter on geriatric psychopharmacology (Gelenberg & Bassuk, 1997).

#### 1.06.6.11.1 Parkinson's disease

Parkinson's disease, a neurodegenerative disorder characterized by resting tremor, muscular rigidity, and difficulty initiating motor activity, is the most common movement disorder, occurring in approximately 1% of the population. The best current theory for PD rests on evidence of the degeneration of pigmented DA cells in the substantia nigra area of the brainstem (see Kandel et al., 1991). These DA neurons normally project to the striatum of the basal ganglia (part of the extrapyramidal motor control system), primarily in the nigrostriatal pathway. This loss or injury to DA neurons results in an associated loss of striatal DA. In Parkinson patients, striatal DA depletion can range from 70 to more than 90% with lesser losses in other DA and non-DA systems. The loss of midbrain DA is associated with the loss of DA transporters in the striatum. Thus, any DA remaining in the synapse is metabolized rather than being taken back up into the terminal. Persistent lowered levels of DA results in an increase in D<sub>1</sub> and D<sub>2</sub> receptor density (up-regulation) in the caudate and putamen areas of unmedicated Parkinson patients. Treatment with L-DOPA (see below) returns receptor densities to normal levels. Other data are consistent with this DA loss account of Parkinson's disease. In 1983, heroin addicts, attempting to synthesize their own drug, accidentally made MPTP. This compound, which selectively destroys nigrostriatal DA neurons, produced a Parkinson-like syndrome in the addicts and provided researchers with excellent support of the DA theory.

The best current pharmacotherapy for Parkinson's disease is the DA precursor L-DOPA, usually coadministered with an inhibitor of enzymes that could destroy L-DOPA. Unlike DA itself, L-DOPA can pass the blood-brain barrier and can be converted into DA by the few remaining neurons. In particularly severe cases, DA agonists may prove effective, most commonly, bromocriptine, a D<sub>2</sub> agonist. Other drugs that act to increase DA levels (MAOIs and DA uptake inhibitors) also have been tried. One novel approach uses the drug selegiline,

which is thought to work not through inhibition of DA breakdown but as an antioxidant to prevent neuronal death. Other, more experimental approaches, involve the transplantation of fetal adrenal cells directly into the basal ganglia (see Kandel et al., 1991).

#### 1.06.6.11.2 Alzheimer's disease and other dementias

Alzheimer's disease is characterized by severe dysregulation of the acetylcholine pathways in the brain and development of neuritic plaques and neurofibrillary tangles. As with most other neuropsychiatric disorders, its etiology is not clear. Although the plaques and tangles are diagnostic, the cognitive deficits may be caused by the loss of cholinergic function, particularly that generated in the nucleus basalis. This disease causes degeneration of cholinergic neurons in the nucleus basalis, which is the primary ACh input to the cortex. People with the disorder also show reduced ACh activity in the hippocampus, which is thought to be the cause of the memory deficits. Researchers have also found reduced levels of somatostatin, neuropeptide Y, and corticotropin-releasing factor and reduced numbers of NMDA receptors and locus coeruleus neurons. However, the significance of these changes is not yet known. Alzheimer's is associated with Down's syndrome, as most individuals with Down's who live to the upper reaches of the age range develop Alzheimer's. The meaning of this correlation is also unclear. Research is also underway into the cause and meaning of the excessive amounts of  $\beta$ -amyloid protein found in the neurofibrillary tangles of Alzheimer's patients.

Psychopharmacological treatments have focused on the ACh deficit. Researchers have tried giving choline, physostigmine (to inhibit acetylcholinesterase), and the muscarinic agonist arecoline, trying to boost the production or effectiveness of the patient's natural ACh. In the late 1990s, the treatment of choice is tacrine, an anticholinesterase. Tacrine inhibits the breakdown of ACh that increases the amount available for use. However, this only works if enough ACh is being released. Currently, no treatment to stop the degradation of ACh neurons exists. Researchers outside the USA have tried to transplant fetal neural tissue to replace the ACh neurons destroyed by Alzheimer's. Although it is not a psychopharmacological treatment, it is mentioned here because it is designed to treat the disorder directly. However, results have been equivocal and not adequately replicated. Fetal neural transplants are still very controversial, despite the US government's decision to allow fetal tissue use in research.

The interested reader is referred to an excellent chapter by Stahl (1996), which summarizes strategies and data regarding cognitive enhancers and neuroprotective agents.

Vascular dementia is differentiated from other dementias by its sudden onset and stepwise deterioration. Treatment is similar to that for a single stroke. Persons with the disorder may be prescribed cognition enhancers (e.g., free-radical scavengers like vitamin E) and anticoagulants (e.g., aspirin) to relieve symptoms and reduce the chance of another stroke.

#### 1.06.6.11.3 Huntington's disease (HD)

HD, formerly known as Huntington's chorea, is included here primarily because, although defined as a movement disorder, it entails progressive degeneration of cognition and emotion. In some people, the disorder may manifest itself first as a psychiatric disorder or dementia. It is a genetic disorder, transmitted by a gene on the short arm of chromosome 4. The disorder causes a particular type of cholinergic interneuron in the striatum to degenerate. Although researchers do not know why these neurons die, some have suggested that they die of the excitotoxic effects of glutamate or one of the other excitatory amino acid NTs. This results in the disinhibition of the DA neurons in the substantia nigra, which causes the involuntary movements known as chorea.

Unfortunately, a curative treatment for Huntington's disease is not available, but progress should be more rapid now that the gene locus has been identified. Psychopharmacological treatments typically attempt to correct the striatal system imbalance. Treatments include medications such as physostigmine, which is an acetylcholinesterase inhibitor, to enhance the ACh available for use. In addition, DA antagonists like the typical antipsychotics can help to inhibit the overactive DA systems and partially relieve the symptoms. To protect the cholinergic interneurons, NMDA receptor antagonists (to block glutamate) like amantadine and calcium-channel blockers like nimodipine have also been tried, although they are still quite experimental. As with the other movement disorders, fetal striatal transplants have been suggested to replace the neurons lost to the disorder.

### 1.06.6.12 Developmental Disorders Originating in Childhood

#### 1.06.6.12.1 Attention-deficit hyperactivity disorder (ADHD)

ADHD occurs in approximately 5% of the school-age population and is characterized by

inattention, impulsivity/distractibility, and hyperactivity. Researchers do not have a clear theory of its etiology. The most popular current theory is that children with ADHD have some deficits in right hemispheric function, particularly in the prefrontal-striatal circuitry (Heilman, Voeller, & Nadeau, 1991). Several neuropsychological and imaging studies have supported this hypothesis.

However, psychopharmacological treatments of ADHD do not address the hypothesized right hemisphere dysfunction directly. Amphetamine was first used in 1937 to treat ADHD, and it and amphetamine-like drugs continue to be the most popular medications. Medications used in the late 1990s include dextroamphetamine (Dexedrine), methylphenidate (Ritalin), and pemoline (Cylert), all of which improve attention and reduce hyperactivity in children with ADHD. Side-effects include decreased appetite, increased heart rate and blood pressure, insomnia, and tics. ADHD is often comorbid with Tourette's syndrome, and stimulants may worsen or reveal Tourette's-like vocal and motor tics. Pemoline can cause liver dysfunction, and a child on pemoline should have liver function tests every few months to rule it out. Children with ADHD who do not respond to stimulants may need a trial of tricyclic antidepressants such as imipramine and desipramine. Antidepressants are also recommended for children with a comorbid anxiety or depressive disorder.

#### 1.06.6.12.2 Other disorders

For reasons of space and lack of a clear-cut psychopharmacological strategy, most other developmental disorders will not be addressed in this chapter (e.g., mental retardation, autism, Rett's disorder, childhood disintegrative disorder, and Asperger's disorder). However, the reader should be aware that data on pediatric psychopharmacology is limited relative to that for adults (see Gelenberg & Bassuk, 1997, for a review of pediatric psychopharmacology).

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# 1.07

## Animal Models of Psychopathology: Depression, Anxiety, Schizophrenia, Substance Abuse

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### 1.07.1 INTRODUCTION

The concept of modeling psychopathology in animals is as old as the use of animals in psychological investigations; its roots may be found in the work of Pavlov, Watson, and

Rayner (1920) and even earlier (Abramson & Seligman, 1977; Weiner, 1973). However, for many years, practical attempts to devise animal models were sporadic, *ad hoc*, and unconvincing (see Abramson & Seligman, 1977; Keehn, 1979). As a result, animal models of psychopathology

were until recently viewed with justified suspicion. This situation has now changed, with the recognition that animal models can provide a means of investigating the neurobiological mechanisms underlying psychopathology: indeed, given the limitations of the investigational techniques currently available for use in human subjects, animal models represent the only means of asking many of the important questions. Animal models can also be of great value in the process of psychotropic drug development, and again, frequently represent the only viable method of predicting novel therapeutic actions. The development and acceptance of animal models may thus be seen as an adjunct to the concurrent growth of psychopharmacology and biological psychiatry.

For the purposes of this chapter, animal models may be defined as representing

experimental preparations developed in one species for the purpose of studying phenomena occurring in another species. In the case of animal models of human psychopathology one seeks to develop syndromes in animals which resemble those in humans in certain ways, in order to study selected aspects of human psychopathology. (McKinney, 1984)

However, it should be noted in passing that this definition presents a rather limited view of the way in which animal models of psychopathology are actually used. Although the earlier animal models were developed primarily to elucidate psychological processes, animal models are used increasingly, and in some cases, exclusively, within the context of psychopharmacology. Within this context, animal models are used for three distinct purposes (Willner, 1990). In one class of model, which may be termed a behavioral bioassay, behavioral tests are used as a convenient means of measuring the status of an underlying brain mechanism: for example, the bizarre set of drug-induced behaviors known as the serotonin syndrome may be used to measure the responsiveness of serotonin receptors to agonist drugs. For the purposes of this chapter, we simply note the existence of behavioral bioassays, but consider them no further. Behavioral models are also used as predictive screening tests within the pharmaceutical industry. In this context, pharmacological considerations are paramount, and the behavioral features of the model are of less importance. The present review will, out of necessity, make some reference to pharmacological studies, but they will not be emphasized. Finally, animal models are used as experimental tools to investigate or illuminate aspects of psychopathology. It is to this use of animal

models, in which an attempt is made to simulate the disorder, that the above definition applies. This chapter is primarily concerned with simulation models, and presents an overview of animal models in the four areas most relevant to clinical psychopharmacology: depression, anxiety, schizophrenia, and substance abuse. First, we consider some general issues pertaining to the validity of animal models.

### **1.07.1.1 Validation of Animal Models**

The systematic validation of an animal model is no different in principle from that of any other psychological device, such as a psychometric test (Vernon, 1963) or a psychiatric diagnosis (Carroll, 1989), and the same general approaches to validation are applicable: predictive validity means that performance in the test predicts performance in the condition being modelled; face validity means that there are phenomenological similarities between the two; and construct validity means that the model has a sound theoretical rationale (Willner, 1986, 1991). The systematic assessment of animal models against each of these sets of criteria allows an unbiased comparison of their strengths and weaknesses. In the paper that first considered the problem of validating animal models, and so formed a starting point for subsequent research, McKinney and Bunney (1969) suggested that to be valid, a model should resemble the disorder modeled in four respects: etiology, symptomatology, treatment, and physiological basis. The first two of these domains refer primarily to face validity and the third to predictive validity, while the fourth reflects aspects of construct validity. However, any of these substantive domains could in principle contribute to an evaluation of validity on each of the three dimensions. Before reviewing animal models of psychopathology, it is appropriate to discuss briefly the general principles underlying the concepts of face, predictive, and construct validity as applied to animal models.

The simple comparison of the features of the model with the features of the disorder is the starting point for an evaluation of face validity. Ideally, a model should resemble the disorder in a variety of respects, and the *DSM* checklist approach to psychiatric diagnosis (American Psychiatric Association, 1994) provides one useful starting point for enumerating the areas of potential comparison. If several points of similarity are demonstrable, then it is necessary to ask whether the cluster of symptoms identified forms a coherent grouping that might realistically be seen in a single patient, or whether they are drawn from a variety of



diagnostic subgroups. Frequently, animal models focus on a single behavior. In that case, it is essential to assess the importance in the total clinical picture of the symptom modeled. If the behavior in the model consists simply of a change in locomotor activity, for example, this is likely to be of peripheral relevance. Similarly, the face validity of the model is less strongly supported if the symptom modeled is common to several different psychiatric disorders. A further requirement for face validity is that the similarity between behavior in the model and the clinical symptom modeled be demonstrated rather than assumed (Abramson & Seligman, 1977). As Abramson and Seligman observed, animals subjected to uncontrollable electric shocks are undoubtedly stressed, but if severe shock is the only stressor that can be shown to generate a particular set of behavioral or neurochemical changes, then the model has little relevance to the concept of stress as used in human contexts. The demonstration of similarity requires a thorough experimental analysis, which, sadly, is often lacking.

The concept of predictive validity implies that manipulations known to influence the pathological state should have similar effects in the model: thus, manipulations known to precipitate or exacerbate the disorder should precipitate or exacerbate the abnormalities displayed in the model, while manipulations known to relieve the disorder should normalize behavior in the model. In principle, therefore, questions of predictive validity can be addressed to a number of features of simulations, including their etiology and physiological basis. In practice, the predictive validity of animal models relevant to psychopathology is determined largely by their response to therapeutic drugs. In this context, a valid test should be sensitive and specific, it should respond to effective therapeutic agents, and should fail to respond to ineffective agents. Positive responses should occur at sensible doses, and should be demonstrable with a range of structurally diverse compounds, and where applicable, to nonpharmacological treatment modalities; it is sometimes possible to demonstrate a correlation between potency in the model and the clinical dose. Negative responses should be demonstrable with agents that cause behavioral changes similar to the therapeutic effect but by nonspecific actions (e.g., by changing locomotor activity).

A number of other factors that need to be considered in relation to drug effects in animal models are discussed elsewhere (Willner, 1991). However, one important fact that is sometimes overlooked, with misleading consequences, is that the clinical classification of drugs as active

or inactive may sometimes be incorrect. Drugs thought to be active on the basis of early open trials are frequently found to be inactive in later well-controlled tests; conversely, a drug may appear to be inactive because the emergence of side effects prevents its administration at adequate dosages, a problem that is less likely to arise in an animal model. It follows that the failure of an animal model to predict accurately will tend to weigh against the model, but may sometimes call instead for a re-evaluation of the clinical wisdom.

In order to evaluate the theoretical rationale of an animal model (construct validity), we require a theoretical account of the disordered behavior in the model, a theoretical account of the disorder itself, and a means of bringing the two theories into alignment. This can only be done if the clinical theory occupies an appropriate framework, which uses terms and concepts applicable also to subhuman species. Clearly, the subjective dimension of psychopathology cannot be central to such a theory, since subjective phenomena in animals are for most practical purposes outside the realm of scientific discourse (Willer, 1991). However, at the level of the cognitive processes underlying psychopathology (Brewin, 1988), and the physiological and biochemical mechanisms that underly those cognitive processes, the possibility exists of constructing parallel theories (Willner, 1986, 1991). It follows from this analysis that the assessment of construct validity involves a number of relatively independent steps. On the one hand, the theoretical account of behavior in the animal model requires evaluation. For example, if an animal model of depression is conceptualized as a decreased ability to respond to rewards, then at the very least, it must be convincingly demonstrated that the decrease in rewarded behavior cannot be explained by, for example, nonspecific sedative effects. Similarly, an animal model of dementia must demonstrate that performance failures result from a disorder of learning or memory, rather than from nonspecific causes, and further work should seek to characterize the specific memory processes involved. Equally important, however, is the evaluation of the account offered of the clinical disorder, both at the level of cognitive analysis and at the level of the relationship between cognitive changes and subjective experience (Brewin, 1988; Willner, 1991). It will be clear that a detailed consideration of the human disorder forms an essential step in the evaluation of animal models, and that the relatively poor state of theoretical understanding of most psychopathologies places an upper limit on construct validity.

## 1.07.2 ANIMAL MODELS OF DEPRESSION

### 1.07.2.1 Clinical Considerations

Depression is a heterogeneous disorder, which presents with varying patterns of symptomatology, a fact often overlooked by clinical psychologists. Perhaps the most important distinction is between endogenous and nonendogenous depressions, which differ in their responsiveness to psychological intervention (not, as frequently assumed, in the presence or absence of psychological precipitants: see e.g., Lewinsohn, Zeiss, Zeiss, & Haller, 1977; Paykel, 1979). Endogenous depression, which corresponds to the *DSM-IV* diagnosis of melancholia, is defined by an inability to experience pleasure (anhedonia), and may be conceptualized as a decrease in the sensitivity of brain reward systems (Klein, 1974). Melancholia is characterized by psychomotor retardation and a number of biological markers, responds well to antidepressants, and is a particular target for animal models.

Inevitably, animal models must focus on behavioral and physiological symptoms. Thus, anhedonia (decreased response to rewards), loss of appetite, and sex drive are readily modeled, and loss of interest may be modeled as a decrease in motivation, but such symptoms as feelings of worthlessness and guilt, or thoughts of suicide, fall outside the scope of animal models. Although psychomotor change, which is the most characteristic symptom of severe depression (Nelson & Charney, 1981), is typically modeled as a decrease (retardation) or increase (agitation) in locomotor activity, the clinical phenomena are actually considerably more complex, and there must be serious doubt about the face validity of models that rely exclusively on gross changes in locomotor activity. Depression is effectively treated by a wide variety of chemically and functionally distinct drugs, and it is therefore inevitable that pharmacological considerations also play an important role in the validation of animal models of depression. In addition to questions of generality and specificity of drug effects in an animal model, two features of antidepressant drug action are particularly important: that these drugs have a slow onset of action, typically requiring 2–5 weeks of treatment, and that they fail to elevate mood in nondepressed individuals.

### 1.07.2.2 Stress Models

Animal models of depression have been reviewed by Katz (1981), Jesberger and Richardson (1985), and Willner (1984, 1990). The largest group of animal models of depression

consists of paradigms based on the application of stress. In a simple and widely-used model, sometimes known as the “behavioral despair” test, rats or mice are forced to swim in a confined space; antidepressants are found to increase the duration of active struggling, or conversely, to decrease the duration of immobility which develops later in the test (Porsolt, 1981; Porsolt, LePichon, & Jalfre, 1977). This model has been used extensively in drug development, and functions with reasonable accuracy as an antidepressant screening test (Porsolt, 1981; Borsini & Meli, 1988). However, many workers prefer to use the theoretically-neutral term “forced swim,” rather than the theoretically-loaded term “behavioral despair.” This more restrained position is endorsed by studies suggesting that immobility during forced swimming may be better characterized as a successful energy-saving coping response (West, 1990).

The importance of proper behavioral analysis is nowhere more evident than in relation to the original stress model of depression, learned helplessness. This procedure was based on the observation that animals exposed to uncontrollable stress (usually electric shocks) were subsequently impaired in learning to escape from shock, a deficit not seen in animals exposed to controllable shock (Seligman, 1975); these learning difficulties are reversed with reasonable selectivity by subchronic (3–7 days) treatment with antidepressant drugs (Martin, Laporte, Soubrie, El Mestikawy, & Hamon, 1989; Sherman, Saquitne, & Petty, 1982). It was proposed that exposure to uncontrollable (but not controllable) stress provides the basis, in animals as in people, for learning that stress is uncontrollable (helplessness), and that this learning has a number of debilitating consequences, including depression (Seligman, 1975). This hypothesis has been immensely influential within clinical psychology, probably more so than any other hypothesis derived from animal models. However, interpretation of the “learned helplessness” effect has been the subject of considerable controversy in both the human (Abramson, Metalsky, & Alloy, 1989; Abramson, Seligman, & Teasdale, 1978) and animal literature.

The term “learned helplessness” implies that the animals perform poorly because they have learned that their responses are ineffective in controlling their environment (Seligman, 1975). However, inescapable shock has a variety of other simpler effects that could also explain many of the behavioral impairments such as decreased locomotor activity (Glazer & Weiss, 1976) and analgesia (Lewis, Cannon, & Liebeskind, 1980). In order to demonstrate that inescapable shock does, additionally, cause

“cognitive” impairments, performance accuracy was assessed using a maze task, in which performance would be independent of factors influencing motor speed. As predicted, accuracy was reduced in animals previously subjected to unavoidable shock, confirming the presence of a “cognitive” component to the pattern of impairment (Jackson, Maier, & Rapoport, 1978). However, subsequent work showed that this “cognitive” impairment arose from an increase in distractibility rather than from a learning disability (Minor, Jackson, & Maier 1984). So inescapable shock does cause “cognitive” impairment, but at the level of attentional processes rather than “helplessness.” Furthermore, in the typical experimental paradigm, inescapable shock is not only uncontrollable but also unpredictable in onset and/or offset. It was demonstrated many years ago that simply providing a feedback signal to accompany shock offset conferred protection against the ulcerogenic effect of inescapable (and therefore, uncontrollable) shock (Weiss, 1970). More recently, signals at the onset or offset of uncontrollable shock have been shown to protect against subsequent behavioral suppression. It was hypothesized that unpredictability generates high levels of fear, which are maintained within manageable limits by signals denoting the presence of danger or safety (Jackson & Minor, 1988). This reconceptualization explains some hitherto puzzling observations, such as the fact that inescapably shocked animals are easily distractible (Minor et al., 1984), which is exactly as would be expected in a state of fearful hypervigilance, but is difficult to explain in terms of loss of control (though even this is not the whole story: Maier, 1994). From this perspective, learned helplessness may be more valid as a model of anxiety rather than depression. However, while stress-induced response suppression may be prevented by anxiolytic drugs, they fail to reverse an already-established effect (in contrast to antidepressants, which are effective both before and after stress) (Drugan, Ryan, Minor, & Maier, 1984). This suggests that the relevance of these paradigms may be to a form of anxiety that does not respond to benzodiazepines. Phobia is one possibility, particularly if the conditioned fear interpretation of learned helplessness effects is correct: phobias do not respond to treatment with conventional anxiolytic drugs, but there is evidence that some phobic states do respond to antidepressants (Liebowitz, 1992).

Another problem in attempting to relate the learned helplessness model to depression is that the “core symptom” of the learned helplessness paradigm—an impairment of shock avoidance learning—does not reflect in any obvious way

the symptomatology of depression, as captured, for example, by the *DSM-IV* diagnostic system (American Psychiatric Association, 1994). However, the learning impairment is only one among a wide range of consequences of uncontrollable shock, and in fact, the breadth of symptomatic parallels to severe depression has led to the suggestion that rodents subjected to uncontrollable shock could meet the *DSM* criteria for major depression (Weiss et al., 1982). Among these behavioral abnormalities is a poor performance of rewarded behavior, which, as noted above, may be relevant to melancholia. One manifestation of this effect is a long-lasting decrease in responding for brain stimulation reward, which is specific to certain electrode placements, and therefore suggests a subsensitivity within part of the brain mechanism of reward, rather than, for example, a motor impairment (Zacharko, Bowers, Kokkinidis, & Anisman, 1983; Zacharko, Lalonde, Kasian, & Anisman, 1987). However, this subsensitivity to reward is long-lasting only if the animals are tested in the immediate aftermath of stress; otherwise the effect dissipates rapidly (Zacharko et al., 1983). A related finding is that following an initial exposure to severe stress behavioral deficits may be reinstated by mild stressors that are without effect in normal animals (Anisman & Zacharko, 1982). These studies suggest that it may be possible to develop conditioning models to explain how the risk of depression is elevated for several months in the aftermath of a stressful life event: the predisposing influence of life events in depression is well established, but is less well understood (Brown, 1993). Another important observation is that uncontrollable electric shock has variable behavioral effects (most of which are antidepressant-reversible) in different inbred mouse strains. To take an extreme example, in the C57BL/67 strain, uncontrollable shock severely impaired subsequent learning to escape shock, but had no effect on responding for brain stimulation reward, while the DBA/2J strain showed exactly the opposite pattern of deficits (Shanks & Anisman, 1988; Zacharko et al., 1987). These studies may provide a starting point for investigation of the physiological mechanisms underlying individual differences in responses to stress.

Subsensitivity to reward, an operational definition of anhedonia, the defining symptom of the melancholic subtype of major depression (Klein, 1974), has been most extensively characterized and studied in the chronic mild stress procedure. In this model, chronic, sequential exposure of rats or mice to a variety of mild stressors causes a decrease in their responsiveness to rewards, which is typically

monitored as a decrease in the consumption of dilute sucrose solutions. This and related effects may be maintained over a period of weeks or months by continued application of the stress regime (Willner, Towell, Sampson, Muscat, & Sophokleous, 1987; Willner, Muscat, & Papp, 1992; Willner & Papp, 1997). The chronic mild stress paradigm has been extensively validated as a model of anhedonia. Initial studies showed that, in contrast to the decreased intake of dilute solutions of sucrose or saccharin, chronic mild stress did not decrease intake of plain water, food pellets, or concentrated sucrose solutions; thus the effects are not simply nonspecific changes in consummatory behavior (Muscat & Willner, 1992). Neither are the effects on consumption of sweet fluids secondary to loss of body weight, which is another consequence of chronic mild stress (and itself a symptom of depression): weight loss alone does not alter sweet fluid consumption, and in rats or mice subjected to chronic mild stress, decreases in sweet fluid consumption are still present after "correcting" for changes in body weight (Willner, Moreau, Nielsen, Papp, & Sluzewska, 1996).

Convergent evidence to support the hypothesis that changes in sweet fluids reflect an underlying change in sensitivity to rewards comes from studies using place conditioning to measure rewarding effects. In this paradigm, animals display a preference for a distinctive environment in which they have previously received rewards (Carr, Phillips, & Fibiger, 1989). Chronic mild stress attenuated or abolished place preferences established using a variety of natural or drug rewards. By contrast, drug-induced place aversions were unaffected by chronic mild stress, indicating that the effects on place preference conditioning are unlikely to result from a nonspecific motivational impairment or a failure of associative learning (Willner et al., 1992). Finally, chronic mild stress has also been shown to cause an increase in the threshold for brain stimulation reward (Moreau, Jenck, Martin, Mortas, & Haefely, 1992). The convergent evidence from all of these techniques supports the hypothesis that chronic mild stress causes anhedonia: while any one of these effects is susceptible of a variety of interpretations, anhedonia, a generalized decrease in sensitivity to rewards, is the most parsimonious hypothesis to account for all of the data.

Like the learned helplessness procedure, chronic mild stress causes a range of behavioral and physiological changes relevant to depression, in addition to the "core symptom" of the model, in this case, anhedonia. For example, chronic mild stress causes decreases in affective (e.g., sexual) and locomotor behaviors, and

changes in the sleeping electroencephalogram characteristic of depression, such as a decrease in the latency to enter the first period of rapid eye movement sleep. Indeed, all of the behavioral symptoms of major depressive disorders listed in *DSM-IV* have now been demonstrated in animals exposed to chronic mild stress, along with most of the biological symptoms characteristic of severe depressions (Willner et al., 1992; Willner & Papp, 1997). The relevance of this model to depression is further supported by the evidence that the decreases in sensitivity to reward are reversed by all of the major classes of antidepressant drugs, and by electroconvulsive shock, but not by a variety of nonantidepressant drugs. Furthermore, the effects of antidepressants in this paradigm resemble the clinical situation, in two important respects: in both cases, treatment typically requires chronic (2–5 weeks) drug administration; and the effects are specific to the stressed/depressed state, since antidepressant treatment does not increase hedonic behavior either in nonstressed control animals or in nondepressed human volunteers (Willner et al., 1987, 1992; Willner & Papp, 1997). In view of these parallels with clinic, the chronic mild stress model has been used extensively to study the mechanisms of action of antidepressant drugs (Willner et al., 1992; Willner & Papp, 1997).

### 1.07.2.3 Other Approaches

The presumed etiological role in depression of loss events, and particularly, loss of a loved one, has led to the development of a number of animal models of depression based on separation phenomena. The most familiar of these models involves nonhuman primates, either infants isolated from their parents or juveniles isolated from their peer group. These studies originated from the work of Harlow (1958). Harlow's initial studies were designed to investigate mothering, and showed that infant rhesus monkeys reared with surrogate mothers preferred soft, cuddly surrogates to wire models containing a feeding bottle, demonstrating the importance of sensory contact. However, protest responses in infant monkeys separated from real or surrogate mothers were also noted, and these became the main focus of attention. The separation response consists of an initial stage of "protest," characterized by agitation, sleeplessness, and distress calls, followed by "despair," characterized by a decrease in activity, appetite, play and social interaction, and the assumption of a hunched posture and "sad" facial expression (Henn & McKinney, 1987; Suomi, 1976). These symptoms are strikingly similar to those

of “anaclitic depression” in institutionalized children (Robertson & Bowlby, 1956). The relationship to adult depression remains uncertain, though recent life event research has confirmed that “loss events” such as bereavement are particularly likely to precipitate depression (Brown & Harris, 1989). Although the evolutionary proximity of primates has led some authors to consider these models to be of particular importance (e.g., Everitt & Keverne, 1979), their contribution has been rather modest. Because of the expense of using primates, the size of experimental groups in most studies has been too small to provide reliable data, and the few pharmacological studies using these models have not been impressive (Willner, 1990).

Separation phenomena of “protest” followed by “despair” are present to some extent in many other species, including cats, dogs, rodent, and precocial birds (Katz, 1981; McKinney & Bunney, 1969), and several of these phenomena have also been used as the basis for the development of animal models of depression. One of these, the reactivation of distress calling in one-week-old chicks appears to perform relatively well as an antidepressant screening test (Lehr, 1989). Chronic (4–6 weeks) isolation of adult rats has been found to cause a disruption of cooperative social behavior (Berger & Schuster, 1982) reminiscent of the poor social performance of depressed people (Lewinsohn, 1974), and this impairment of social cooperation is reversed by chronic antidepressant treatment (Willner et al., 1989). Thus, the investigation of complex phenomena associated with social isolation does not rely on the use of primates.

There are many other animal models of depression, which will not be discussed in detail because they have been studied largely for their pharmacology, and have received little psychological characterization. These include a number of inbred rat or mouse strains that display certain depression-like behaviors (Overstreet, 1993), and could in principle be used to investigate individual differences in vulnerability to depression, if validated. There is also a quite widely-used model based on the destruction of the olfactory bulb in rats, which differs from most other models in that the animals are hyperactive (Cairncross, Cox, Forster, & Wren, 1979; Van Riezen & Leonard, 1990). Thus, this model may reflect different aspects of depression, and may be relevant to agitated states.

This example highlights a significant limitation of animal models of depression, which have tended to focus either on an undifferentiated depressive state or on melancholia, while largely ignoring some other well-defined subtypes of

depression. Delusional depression, for example, is differentiated behaviorally from nondelusional depression only by a greater association with psychomotor agitation (Nelson & Charney, 1981), but is pharmacologically distinct, being unresponsive to tricyclic antidepressants but responsive to electroconvulsive therapy (ECT) or to tricyclic/neuroleptic combinations (Nelson, 1987). Bipolar disorder is another well-defined diagnostic category for which there are no animal models. In this case, the depressive episodes appear to be identical to those of unipolar endogenous depression, but episodes of mania are interspersed. While there are a number of animal models of mania (Lyon, 1991; Robbins & Sahakian, 1980), the alternation of depressive-like and manic-like behaviors in an animal model has not yet been systematically addressed. Indeed, the episodic nature of unipolar depression (Angst et al., 1973) is itself another area that has not been explored in animal models.

### 1.07.3 ANIMAL MODELS OF ANXIETY

#### 1.07.3.1 Clinical Considerations

Animal models of anxiety, which have been reviewed by Gray (1982), Treit (1985), and Green and Hodges (1991), have a long history, deriving from the early work of Pavlov and Masserman on “experimental neurosis.” In these early studies, regressive, abnormal behaviors were produced by exposing animals to conflict: in Pavlov’s studies, difficult discrimination problems in a conditioning procedure, and in Masserman’s studies, approach-avoidance conflict (Abramson & Seligman, 1977). This set the stage for later work, most of which is based squarely on the assumption that conflict procedures are, almost by definition, animal models of anxiety.

A second early precursor to present-day animal models of anxiety is found in Watson’s description of conditioned fear in the child “Little Albert” (Watson & Rayner, 1920). This observation, together with the formal demonstration by Miller (1960) that fear could be conditioned in laboratory animals, and suppressed by counterconditioning procedures, led directly to the development of systematic desensitization procedures for phobic anxiety (Wolpe, 1958), which for many years remained the jewel in the crown of behavioral psychotherapies (albeit that we now know that conditioning principles provide a rather incomplete account of the processes underlying systematic desensitization: Wilkins, 1971). Another early contribution of fear conditioning research to the treatment of phobias came from

the observation that avoidance responses, once learned, are extremely long-lasting. Though initially perplexing, it soon emerged that this resistance to extinction occurs because an animal that makes a successful avoidance response has no opportunity to learn that the response is no longer necessary. If the animal was prevented from making the avoidance response, and so forcibly exposed to the information that a failure to respond did not lead to adverse consequences, the avoidance response extinguished rapidly. The treatment of phobic anxiety by flooding is based upon this observation (Solomon, Kamin, & Wynne, 1953). Although studies of fear conditioning led directly to the development of two successful treatments, it was later realized that this approach had some serious limitations, notably in failing to address the specificity of phobias to certain classes of objects (e.g., animal phobias are common, knife phobias are not). This feature is found in certain types of animal learning, which, it has been claimed, are preprogrammed by evolution to be selective and long-lasting—one much-discussed example is the conditioned taste aversion, in which novel foods are later avoided if their ingestion is followed by illness (Seligman, 1971). However, while conditioned taste aversions may display some of the characteristics of phobic anxiety, a more recent social learning model, in which monkeys acquire a fear of snakes by observation of already fearful animals (Mineka & Cook, 1986; Mineka, Davidson, Cook, & Keir, 1984), is perhaps more persuasive.

Alongside the concept of “conflict,” the second major principle underlying the construction of animal models of anxiety has been the assumption that a model of anxiety should respond specifically to benzodiazepines and functionally related drugs. However, while benzodiazepines are effective in generalized anxiety disorder, they are actually ineffective in most forms of anxiety, such as phobia, obsessive-compulsive disorder and panic (Rickels, 1985; Rickels & Schweizer, 1987). Thus, to the extent that animal models of anxiety have relied on a response to benzodiazepines, they are primarily models of generalized anxiety disorder. Unfortunately, of all forms of anxiety, the symptomatology of generalized anxiety disorder is the least amenable to behavioral modeling, as it consists primarily of autonomic hyperactivity and a feeling of apprehension. In many animal models of anxiety, the behavior expressed appears to have face validity, in the sense that it appears reasonable that an anxious animal might behave in that way. However, the similarities to generalized anxiety disorder are more intuitive than demonstrable. There is also

the problem that “virtually any test devised by experimental psychopharmacologists can induce mild or severe fear” (Green & Hodges, 1991). Thus, the appeal to the construct of fear to validate an animal model of anxiety is not particularly persuasive.

Furthermore, benzodiazepines are not necessarily the drugs of choice for anxiety. Antidepressants are commonly prescribed for anxiety—they are clearly more effective than benzodiazepines in panic disorders (Kahn, McNair, & Frankenthaler, 1986; Robinson, Nies, Ravaris, Ives, & Bartlett, 1978), and it is unclear whether there are any conditions, including generalized anxiety disorder, in which benzodiazepines are demonstrably superior to antidepressants in their clinical efficacy. With certain limited exceptions, antidepressants are not effective in traditional animal models of anxiety, and neither are a number of recently developed novel anxiolytics. This has led recently to a certain disillusionment with the traditional conflict models.

### **1.07.3.2 Conflict Models**

Conflict models are based on both unconditioned and conditioned behaviors, and vary considerably in their behavioral sophistication. The simplest test is the examination of general behavior in a novel arena: locomotor activity is usually increased by anxiolytic drugs (e.g., Blumstein & Crawley, 1983). However, this test is of limited value, given the multitude of factors that can influence open-field activity the difficulty of subjecting open-field behavior to behavioral analysis. Some derivatives of the open field such as the hole-board test (File & Wardill, 1975) and the staircase test (Simiand, Keane, & Morre, 1984) are more complex, but share the same limitations. Emergence tests, from a presumed “safe” to a presumed “dangerous” area, are somewhat more sophisticated. A simple test of this type involves measuring crossings between the light and the dark half of a two-compartment chamber (Crawley, Skolnick, & Paul, 1984). Again, however, it is difficult to distinguish conflict behavior from general locomotor activity (Carey & Fry, 1988). The best known model of this group is the elevated plus-maze, a plus-shaped set of runways which is raised off the ground and has two enclosed and two open and exposed arms. Most, but not all, anxiolytic drugs tend to increase the time spent on the open arms, while other classes of drugs, such as stimulants, neuroleptics, and antidepressants, do not. The validity of this test is supported by the demonstration that levels of defaecation and freezing, as well as blood corticosterone are

higher in animals confined in the open arms (Critchley & Handley, 1987; Pellow, 1986; Pellow, Chopin, File, & Briley, 1985). A number of animal models of anxiety are based on social interactions of various kinds, though there seems little reason to see any of these tests as being particularly relevant to social phobias. The most popular is a test in which social interaction between pairs of male rats is suppressed by using a novel environment and bright lighting; anxiolytics increase interaction levels under these conditions. Almost alone among animal models of anxiety, the social interaction test requires chronic drug administration resulting in tolerance to sedative effects, which are apparent as a decrease in locomotor activity on acute administration (File & Hyde, 1978).

In conflict models based on conditioned behavior, conflict is explicitly programmed into the experimental paradigm by punishing the animal for performing a response that it has been trained to emit in order to obtain a reward. A simple, and rapidly learned procedure of this kind is the Vogel water-lick conflict test where thirsty animals are first trained to lick from a drinking tube and electric shocks are then delivered through the tube or the floor when the animal licks, causing a suppression of drinking. Nonspecific effects are assessed either by including nonpunished drinking periods or by measuring drug effects on home-cage drinking (Vogel, Beer, & Clody, 1971). A more complex model involves training rats to consume a very sweet (32%) sucrose solution, then switching to a weak (4%) solution. When the switch occurs, the rate of licking falls, and only gradually, over the course of several days, returns to that of a rat maintained throughout on the weaker solution. Benzodiazepine treatment speeds the return to normal behavior (Flaherty, Grigson, Demetrikopoulos, & Demetrikopoulos, 1987; Flaherty, Grigson, & Rowan, 1986; see also Green & Hodges, 1991). Unlike other conflict models, this model is based on frustration rather than fear. Its relevance to anxiety is thus less obvious, though there is considerable evidence for a commonality between the brain mechanisms of fear and frustration (Gray, 1982).

The classic and most widely used conflict model is the Geller–Seifter operant conflict procedure. In this model, animals (rats or pigeons) respond in an operant chamber for food rewards; interspersed into the experimental session are periods (typically signaled by a light or tone) during which responding results in both an increased availability of food and a mild electric shock to the feet. Anxiolytic drugs increase responding during the conflict period, and response rates during the nonconflict

period serve as a control for nonspecific drug influences. If necessary, the contingencies may be manipulated so as to ensure equivalent rates of responding in the two components; alternatively, the response rate during the conflict period may be set low to maximize the chance of detecting an increase, or high, to allow the detection of anxiogenic effects (Geller & Seifter, 1960; Sepinwall & Cook, 1978; see also Green & Hodges, 1991). Clearly, this model has manifest advantages over the unconditioned procedures described above in terms of the degree to which the behavioral parameters are under experimental control. However, the procedure is extremely time-consuming, and may require parametric changes during an experiment to maintain stable response rates. The Geller–Seifter procedure appears to have reasonably good face validity. However, the situation is not entirely straightforward. Following the prolonged training necessary to establish stable baselines of responding, animals may no longer display physical signs of fear or autonomic arousal (e.g., Stephens & Andrews, 1991). It is therefore possible that benzodiazepines may increase punished responding by mechanisms other than anxiety reduction, and in fact, there is evidence that they act at least in part by reducing the animal's ability to discriminate between punished and nonpunished components (Hodges, Baum, Taylor, & Green, 1986). It should be noted, however, that this critique of the Geller–Seifter procedure arises precisely because it is amenable to experimental analysis. Other simpler procedures, in which hypotheses concerning behavioral mechanisms are less readily testable, thereby escape criticism.

The construct validity of conflict models, in which behavior is suppressed by “fear” and reinstated by anxiolytic drug treatment, derives in large part from the hypothesis that anxiety results from activity in a forebrain “behavioral inhibition system” (Gray, 1982). This hypothesis, and with it most of the traditional anxiety models, has received a serious challenge from the development of nonbenzodiazepine anxiolytics. The anxiolytic action of buspirone, and related compounds acting at the 1A subtype of serotonin (5HT) receptors, is now well established; however, these drugs are ineffective, or minimally effective, in traditional anxiety models (Griebel, 1995).

### 1.07.3.3 Ethological Models

An alternative approach views anxiety as arising from threatening situations which elicit defensive behaviors. Defensive behaviors, involving characteristic submissive postures,

are seen in an intruder rat or mouse introduced into the home cage of a resident conspecific; these defensive behaviors are suppressed by anxiolytic drugs (Krsiak et al. 1984; Shepherd & Rodgers, 1989). Another model elicits defensive behavior by shocking a rat or mouse through an electric prod; subsequently, the animal will bury the prod in sawdust if given the opportunity to do so. Again, defensive burying is suppressed with reasonable specificity by anxiolytics (Treit, 1985). However, the basic dependent variable in this model, the depth of sawdust piled up, is difficult to quantify.

A more complex approach, derived from studies that were initially conducted largely in wild rats, is based on an ethological analysis of behavior in the presence of, and following exposure to, a predator (a cat). These studies led to the description of a pattern of "risk assessment" behaviors, including visual "scanning" of the area in which the predator was encountered and "stretch approach" alternating with retreat (Blanchard, Rodgers, & Blanchard, 1994; Blanchard, Yudko, Rodgers, & Blanchard, 1993). It has been argued that risk assessment behaviors bear a striking resemblance to the behavioral features of general anxiety disorder: apprehensive expectation, vigilance, hyperattentiveness, and scanning (Blanchard et al., 1994). A number of simpler tests have been developed for evaluating drug effects on risk assessment, including a test in which rats are exposed to cat odor (Blanchard et al., 1993), and observational techniques applied to the elevated plus maze (Rodgers & Cole, 1993). The potentiated startle model is a very simple model of hypervigilance. In this procedure, the startle response to a loud noise, which is very readily quantifiable, is increased by the simultaneous presentation of a light that has previously been paired with electric shock. The potentiation of startle by the fear-inducing signal is suppressed by anxiolytics, which do not, however, affect the basal (unconditioned) response to the loud noise (Davis 1979).

In contrast to the traditional conflict models, in these ethologically-derived tests, both benzodiazepines and 5HT<sub>1A</sub> agonists are anxiolytic (Blanchard et al., 1993, 1994). The effects of benzodiazepines and 5HT<sub>1A</sub> agonists on defensive behavior are not, however, identical. In response to an approaching predator, animals display a characteristic repertoire of defensive behaviors. Benzodiazepines increase flight responses in wild rats to contact by a human experimenter; however, the 5HT<sub>1A</sub> agonists buspirone and gepirone decrease flight responses to such an extent that the rats allow themselves to be touched (Blanchard, Hori, Rodgers, Hendrie, & Blanchard, 1989; Blan-

chard, Rodgers, Hendrie, & Hori, 1988). Flight has been proposed as a potential model of panic (Deakin & Graeff, 1991), which unlike general anxiety disorder, does not respond to benzodiazepines. While buspirone appears to be ineffective in panic disorder (Sheehan, Raj, Trehan, & Knapp, 1993), positive effects of another 5HT<sub>1A</sub> agonist, gepirone, have been reported (Pecknold, Luthe, Scott-Fleury, & Jenkins, 1993), and the efficacy of specific serotonin uptake inhibitors is reasonably well established (Sheehan et al., 1993). Thus, different psychopathologies may reflect different components of the defensive behavioral repertoire. It is important to add that one of the defining features of psychopathology is that the behaviors displayed are disproportionate, and this to some extent limits the validity of models that relate anxiety and panic disorders to defensive behaviors resulting from the recent or actual presence of a predator. Nevertheless, these models serve as a rich source of hypotheses concerning the nature of, and the relationship between, anxiety and panic.

## **1.07.4 ANIMAL MODELS OF SCHIZOPHRENIA**

### **1.07.4.1 Clinical Considerations**

As in the case of animal models of anxiety, models of schizophrenia are dominated by its pharmacotherapy. The evidence that neuroleptic drugs act by antagonizing dopamine receptors (Creese, Burt, & Snyder, 1976) is so strong as to be indisputable, and despite many attempts to break out of the dopamine trap, there is as yet no clear evidence that drugs acting at other sites are effective (Rifkin & Siris, 1987). Nevertheless, a substantial proportion of schizophrenic patients do not benefit from neuroleptics, particularly, but not exclusively, those patients with predominantly negative symptoms, such as poverty of speech or social withdrawal. This fact not only stimulates the continuing search for other modes of treatment, but also casts doubt on the necessity of neuroleptic responsiveness in an animal model of schizophrenia. It is important to note that neuroleptics cause significant motor impairments (via their action on dopamine receptors in the caudate nucleus), and that many of the behavioral actions in animals are probably related more closely to these side effects than to their therapeutic action (see Iversen, 1987). The newer "atypical" neuroleptics are thought to minimize side effects by virtue of additional actions on other neurotransmitter systems (Tamminga & Gerlach, 1987) and/or specific actions within "limbic" areas (White & Wang, 1983). The



development of these drugs has not, however, challenged the basic premise that neuroleptics act by antagonizing dopamine transmission.

A number of reviews have argued in principle against animal models of schizophrenia on the grounds that the symptoms of this disorder are uniquely human (e.g., Kumar, 1976). In fact, this problem is common to all areas of psychopathology, and arguably is not especially more serious in this case. Most of the symptoms of schizophrenia could in principle have counterparts in animals, particularly so if attention is paid to the structure of behavior rather than its content (Lyon, 1991). Hallucinatory behavior, for example, has been defined operationally as "sequences of well defined behaviors that could apparently only be accounted for by the reaction of the animal to non-existent stimuli" (Nielsen, Lyon, & Ellison, 1983), though it should be added that behavior meeting this criterion in animals reflects visual phenomena rather than the auditory hallucinations that are far more prevalent in schizophrenia. Lyon (1991) has suggested that for the purposes of animal models, the *DSM-III* criteria for schizophrenia should be augmented by two additional symptoms that featured prominently in Bleuler's (1950) account of dementia praecox, excessive alternation or switching between behaviors, and repetitive stereotyped responding. With these additions, Lyon (1991) has argued that the symptoms of schizophrenia may be reduced to four classes of abnormal behaviors: response switching, focusing, fragmentation, and stereotypy, which appear in sequence as the disorder progresses. These behaviors are clearly more relevant to the positive symptoms of schizophrenia (hallucinations, delusions, thought disorder), rather than the negative symptoms (affective flattening, poverty of speech, loss of drive, anhedonia), and this reflects the state of the field, which consists largely of models of positive symptomatology.

#### 1.07.4.2 Dopamine Hyperactivity Models

Animal models of schizophrenia have been reviewed by McKinney and Moran (1981), Iversen (1987), and Lyon (1991). As expected from the predominance of dopamine in theoretical accounts of schizophrenia, the majority of animal models involve pharmacological treatments that increase dopamine receptor stimulation, primarily by using directly acting dopamine agonists, such as apomorphine, or by using amphetamine to release dopamine from presynaptic stores. This strategy is greatly encouraged by the close phenomenological similarities between paranoid schizophrenia

and the psychosis that develops in people after prolonged amphetamine intoxication (Connell, 1958). Single, high doses of amphetamine can also elicit the amphetamine psychosis (Griffith, Cavanaugh, Held, & Oates, 1972). In animals high doses of amphetamine cause a species-characteristic pattern of repetitive stereotyped behavior (Randrup & Munkvad, 1971), and a number of paradigms have been described in which amphetamine elicits a repetitive perseveration either of responses, or more interestingly, of complex programs of behavior, such as switching between two responses, which are continued irrespective of their outcome. These effects are antagonized by neuroleptics (Even-den & Robbins, 1983; Ridley & Baker, 1983). Similar patterns of stereotyped switching have been observed in schizophrenic patients (Frith & Dore, 1983; Lyon, Mejsholm, & Lyon, 1986).

It is frequently difficult to decide whether phenomena of this kind are more relevant to schizophrenia or to mania, which in many respects displays a superficially similar pattern of symptoms. However, manic behavior, while increased in rate, and frequently stereotyped and repetitive, usually retains a goal-directed organization, unlike the fragmentation of behavior characteristic of schizophrenia. Behavior on this criterion is more obviously related to schizophrenia, rather than mania, and is seen following chronic administration of dopamine agonists to rats, cats, or monkeys. This has been achieved either by repeated daily injections of amphetamine or cocaine, typically for a period of many weeks (Ellinwood & Kilbey, 1977, 1980), or by the use of an implanted slow-release amphetamine pellet, which lasts for one or two weeks (Ellison & Eison, 1983; Nielsen et al., 1983). Repeated injection regimes cause, eventually, a wide range of behavioral abnormalities, including hyper-reactivity, abortive or fragmented behaviors, and a large number of motor disorders, including dystonias, ataxia, akathisia, and facial dyskinesias. Motor disturbances are not uncommon in schizophrenics. While these are often ascribed to neuroleptic therapy, postural and oculomotor disturbances were described long before the neuroleptic era (Kraepelin, 1919), and evidence suggests that tardive dyskinesias may also be an intrinsic part of the underlying disorder (Barnes, 1988). An important feature of the chronic stimulant syndrome, particularly apparent in the continuous slow-release model, is the presence of apparent hallucinatory behavior. Nielsen et al. (1983), using stringent operational criteria, described four types of hallucinatory behavior in monkeys, all directed at nonexistent objects, resulting in flight, attack, eating, or other behaviors such as threat responses. These

behaviors, as well as motor stereotypies, were suppressed by the neuroleptic haloperidol (Lyon, 1991).

The release of dopamine in subcortical structures is increased in rats reared in social isolation (Jones, Hernandez, Marsden, & Robbins, 1988). Thus, the pathological effects of isolation rearing may to some extent be mediated by dopamine hyperactivity, though clearly, social isolation also has many other effects. Goosen (1981) has described a "social deprivation syndrome" in female rhesus monkeys, consisting of stereotyped behaviors of various kinds, and bizarre and/or self-directed behaviors. These abnormalities resemble some of the behaviors observed following chronic stimulant drug treatment, but the more florid hallucinatory behaviors are far less conspicuous. As some of the effects of social isolation are reversed by antidepressants, isolation rearing provides at best an approximation to a model of schizophrenia. However, the demonstration that prolonged changes in dopamine activity can be elicited environmentally is encouraging.

In addition to causing motor stereotypies that resemble positive symptoms of schizophrenia, a dramatic loss of social interaction is seen following chronic stimulant treatment in rats (Nielsen & Lyon, 1982) or monkeys (Ridley, Baker, & Scragg, 1979), or acute treatment with high doses of amphetamine in monkeys (Ellenbroek & Cools, 1990). These effects in monkeys are seen as an increase in interindividual distance, a decrease in the number of interactions initiated, and an increase in the number of interactions terminated, all leading to an increase in social isolation, which may represent a model for negative symptoms (Ellenbroek & Cools, 1990).

#### **1.07.4.3 Attentional Models**

Returning to positive symptoms, it has long been known that schizophrenic patients suffer attentional disorders which have been considered to underly their cognitive deficits (Frith, 1987). This defect is usually characterized as a broadening of attention, leading to an increased response to irrelevant stimuli that would normally be filtered out; a more specific formulation has suggested that the abnormality could be considered to represent a reduction in the influence of past regularities on current perception (Hemsley, 1987). These considerations have led to a number of attempts to model attentional dysfunction; the two most widely used paradigms are the prepulse inhibition and latent inhibition models.

Prepulse inhibition refers to the ability of a warning signal to decrease the amplitude of the startle response to a loud noise. Prepulse inhibition is impaired in schizophrenic patients (Braff, Stone, Callaway, Geyer, & Ball 1978), and is also impaired by dopamine agonist drugs (amphetamine or apomorphine) in animals (Mansbach, Geyer, & Braff, 1988), and by isolation rearing (Wilkinson et al., 1994). While a rather simple behavior, the prepulse inhibition paradigm is of interest because the neural mechanisms underlying the acoustic startle reflex are very well understood, and appear to be very similar in rats and humans (Davis, 1984). This model is used increasingly within the pharmaceutical industry, largely on account of its simplicity. Latent inhibition is a more complex form of habituation, in which pre-exposure to a stimulus reduces the ability to learn about its consequences when it is later paired with a significant event. This ability is impaired in acute schizophrenics, though not in chronic schizophrenics (Baruch, Hemsley, & Gray, 1988). In rats, where the significant event is an electric shock, latent inhibition is disrupted by amphetamine and potentiated by neuroleptics (Joseph & Jones, 1991). An attractive feature of this procedure is that a conditioned fear paradigm is used. Thus, the conditioned stimulus suppresses operant behavior, latent inhibition lifts the suppression, and amphetamine reverses latent inhibition to reinstate the suppression. In other words, amphetamine causes a decrease in responding, so the effect on latent inhibition cannot be an artefact of psychostimulation. However, the significance of this model is uncertain, as an increased reactivity to preexposed stimuli could reflect a nonspecific state of arousal. This interpretation is supported by a study suggesting that the effect of amphetamine in this model results from an increase in the perceived intensity of the electric shock rather than an altered response to the conditioned stimulus (Killcross et al., 1994).

While animal models of schizophrenia are dominated by dopaminergic drugs, there are a number of models based on the use of drugs interacting with other neurotransmitter systems. The most notable of these are lysergic acid diethylamide (LSD) and phencyclidine (PCP). Jacobs, Trulson, and Stern (1977) described a range of bizarre behaviors in LSD-treated cats that resemble the effects of chronic psychostimulant treatment, though at low doses, limb flicks, which are not seen either in the stimulant models or in schizophrenia, were the most prominent effect of LSD. Despite the popularity in the 1960s of the LSD experience as a model of schizophrenia in humans, it is clear that both clinicians and patients perceive the effects of

LSD as different from schizophrenia (Hollister, 1962), unlike amphetamine, which appears to exacerbate existing symptomatology. PCP, on the other hand, does appear to induce the full range of positive and negative symptoms, including thought disorder, in volunteers and in schizophrenics (Davies & Beech, 1960; Greifenstein, Yoshitake, De Valut, & Gajewski, 1958). In both animal and human subjects, PCP produces stereotyped behaviors as well as deficits in social behavior that are comparable to the social withdrawal and isolation that form an important part of the negative symptom complex. Both sets of effects could be reversed in rats by clozapine which is almost alone among neuroleptic drugs in its activity against negative symptoms; in contrast, the typical neuroleptic haloperidol suppressed PCP-induced stereotypies, but failed to normalize social behavior (Sams-Dodd, 1996). It is interesting that PCP also disrupts prepulse inhibition, and the characteristics of this response actually resemble the schizophrenic deficit more closely than the effects of amphetamine (Mansbach & Geyer, 1989).

Finally, there have been a number of attempts to model schizophrenia using brain lesions. Lesions of the hippocampus have been shown to impair performance in a foraging task, using a holeboard in which food was located below four of the 16 holes, which has been suggested to provide a possible model of thought disorder (Oades & Isaacson, 1978). The effects of hippocampal lesions are similar in many respects to those of amphetamine, and may in general be antagonized by neuroleptics (Schmajuk, 1987), though the neuroleptic reversal of the holeboard impairment was only partial (Oades & Isaacson, 1978). More recently, hippocampal lesions have been shown to cause an increase in sensitivity to the disruptive effect of amphetamine in the prepulse inhibition model (Swerdlow et al., 1995). Significantly, neuropathological abnormalities have been described postmortem in the hippocampus of schizophrenic patients (Kovelman & Scheibel, 1984), suggesting that hippocampal damage may be at least in part responsible for an apparent dopamine hyperactivity in schizophrenia. However, while the effects of hippocampal damage do resemble those of acute amphetamine treatment, the hallucinatory and bizarre behaviors that emerge following chronic amphetamine administration are absent. A particularly interesting variant of this model is based on a neonatal hippocampal lesion. Animals prepared in this way appear normal as juveniles, but when adults are hyperactive, hyper-reactive to amphetamine, and show defective prepulse inhibition (Lipska et al.,

1995; Lipska, Jaskiw, & Weinberger, 1993). This is the only model to capture the developmental aspect of schizophrenia, which typically first appears in late adolescence.

### **1.07.5 ANIMAL MODELS OF SUBSTANCE ABUSE**

#### **1.07.5.1 Drug Self-administration**

It is clear from the earlier discussion of depression, anxiety, and schizophrenia that the validity of animal models can often be difficult to determine, in part because of the considerable differences in the methodologies used to study animal behavior on the one hand, and human psychopathology, on the other. However, in the case of animal models of addiction, at least superficially, these difficulties appear less acute: just like humans, animals voluntarily self-administer drugs (Bozarth, 1987; Goudie, 1991; Koob, 1995). Before discussing the self-administration model, a second parallel should be mentioned in passing: animals are also able to report on their perceptions of a drug. In drug discrimination procedures, animals receive food rewards for pressing one lever if they have been administered the drug and a different lever if they have received saline. After training, their response to a novel agent reflects the extent to which it is perceived as similar to the training drug. This model is used extensively in mechanistic studies of psychotropic drug action (Stolerman, Samele, Kamien, Mariathasan, & Hague, 1995), and is considered to represent a model of the subjective effects of drugs (Preston & Bigelow, 1991).

In drug self-administration experiments, the animal, typically a rat or a monkey, is implanted with a chronic in-dwelling intravenous catheter, which is connected through plastic tubing to a reservoir located outside the experimental chamber, which places minimal restraints on the animal's freedom of movement. Provided suitable precautions are taken against infection and blood clots, catheters can remain patent for periods of up to several months, allowing for experiments involving complex and lengthy training procedures and/or chronic drug administration. Drug delivery is made contingent on the performance of an operant response, typically lever pressing, but sometimes a less motorically demanding response, such as nose poking. If access to the drug is unlimited, a pattern of self-administration may develop, consisting of periods of drug intake alternating with periods of abstinence, during which other activities such as eating, drinking, and sleeping occur. This binge pattern of drug consumption has been described in animals self-administering

a wide variety of psychomotor stimulants and with alcohol; binges are also characteristic of problematic use of both of these drug classes in humans. Opioid self-administration is more constant over time, as it is in human intravenous opiate users (Brady, Griffiths, Hienz, Ator, Lukas, & Lamb, 1987; Yokel, 1987). However, unlimited access to self-administered drugs can lead to fatalities, and therefore drugs are rarely made available for self-administration on an unlimited basis (Yokel, 1987). When drug availability is limited by scheduling daily sessions of several hours duration, responding typically follows an inverted U-shaped dose-response function. Above a minimum dose, responding and drug intake rise with drug dose; this ascending portion of the curve may be relatively steep and difficult to study. Above a certain dose, responding falls with further increases in dose, and this has the effect of maintaining the blood concentration of the drug at a relatively—in some cases, remarkably—constant level, which remains stable over prolonged periods of time (Koob, 1995; Yokel, 1987).

In general there is close agreement between the drugs intravenously self-administered by animals and those which are abused by people. Thus, animals readily learn to self-administer opioids (e.g., morphine, heroin), psychostimulants (e.g., amphetamine, cocaine), barbiturates, dissociative anesthetics (e.g., phencyclidine), and alcohol. Also self-administered, though less reliably, are nicotine, caffeine, and benzodiazepines. Conversely, various drugs that are not abused do not typically support self-administration; these include antidepressants, neuroleptics, and serotonin agonists (e.g., fenfluramine, buspirone). However, there are some anomalies. The most obvious is that animals do not reliably self-administer either hallucinogens (e.g., LSD) or cannabinoids; on the other hand, they do reliably self-administer certain drugs which are not abused, such as kappa opioid agonists and local anesthetics (Yokel, 1987; Young & Herling, 1986). Thus, the agreement between self-administered drugs and abused drugs is not perfect; nevertheless, drug self-administration procedures are routinely used within the pharmaceutical industry to predict whether novel compounds have “abuse liability.”

Self-administration procedures can also estimate the relative reinforcing efficacy (relative abuse liability) of different agents. This issue must be approached with caution, since under many circumstances (e.g., cocaine self-administration under fixed ratio schedules of reinforcement and limited daily access), an increase in unit dose of the drug results in a compensatory decrease in response rates, and vice versa. Thus,

a higher response rate may not denote a higher efficacy. This problem has been addressed in a number of ways, the most popular being the progressive ratio schedule. In progressive ratio schedules the response requirement progressively increases, during the session or across session, up to the point at which the subject stops responding; at this “breakpoint,” it is assumed that the work requirement exceeds the reinforcing effect of the drug. Thus, different drugs can be compared directly according to the work that animals are prepared to perform to earn them. Studies using this procedure have shown that cocaine supports higher breakpoints than a variety of other agents, supporting the popular view of cocaine as a highly addictive drug (Katz 1990; Young & Herling, 1986). Motivation to work for drug, as revealed by performance in a progressive ratio schedule, has also been proposed as a method of measuring drug craving in animals (Markou et al., 1993). This approach is supported by a study of human volunteers reinforced under a progressive ratio schedule by puffs on a cigarette, in which breakpoints correlated significantly with a questionnaire measure of cigarette craving (Willner, Harding, & Eaton, 1995).

The importance of measuring motivation, rather than simple consumption of a drug, is well illustrated by studies of alcohol self-administration. While rats or monkeys readily self-administer alcohol by intravenous injection, the majority of animals will not drink alcohol in sufficient quantities to produce significant pharmacological effects, and certainly insufficient to cause physical dependence. The need to model oral alcohol consumption in animals has stimulated selective breeding programs that have succeeded in producing several rat strains characterized by high alcohol consumption, a preference for alcohol over water in two-bottle tests, and in some cases, the development of physical dependence (Crabbe & Li, 1995). However, when alcohol-drinking rats are required to work for alcohol, differences between the strains emerge. Only one strain, the P (alcohol-preferring) line, works harder under a progressive ratio schedule than its nonpreferring (NP) control strain; other high alcohol-drinking strains (HAD, AA) do not (Ritz, Garcia, Protz, Rael, & George, 1995). While the greater proclivity of P rats to consume alcohol suggests that the P and NP rat strains may prove useful in investigating possible genetic bases for alcoholism, it is equally important to note that the difference between the strains is not absolute. Moderate or high levels of alcohol intake can be induced in outbred animals by introducing alcohol in a sweetened solution and gradually fading out the sucrose component—a

progression reminiscent of the process by which many teenagers acquire the taste for alcohol. After initiation of alcohol drinking by means of the sucrose fading technique, alcohol consumption in NP rats is comparable to that of P rats (Samson, Tolliver, Lumeng, & Li, 1989).

#### 1.07.5.2 Drugs as Positive and Negative Reinforcers

The most striking feature of the early literature utilizing drug self-administration models is that drugs function as reinforcers, in operant procedures, in a manner highly reminiscent of conventional reinforcers. In particular, the characteristic patterns of behavior associated with different schedules of reinforcement are maintained as effectively by drugs as by conventional reinforcers, such as food delivery or shock termination (Schuster & Johanson, 1981). These similarities raise the question of whether drug reinforcement has unique characteristics. One potential difference is that with conventional reinforcers, increasing the magnitude of the reinforcer leads to increased responding, whereas with drug reinforcers, the dose-response function has an inverted U-shape, such that increases in dose cause compensatory decreases in response rate (Wise, 1987). However, the same effect can, in fact, be observed with conventional reinforcement: variations in the sweetness of a food reinforcer also lead to changes in responding that follow an inverted U-shaped function (Willner, Phillips, & Muscat, 1991). Another question is whether drugs are more efficacious than conventional reinforcers. Early studies suggested that cocaine might be more reinforcing than food, insofar as cocaine maintained higher response rates than food in second-order schedules of reinforcement (in which long chains of behavior are required to achieve delivery of a reinforcer). However, this difference in responding is only seen when reinforcers are presented at intervals during the session: if the session terminates upon delivery of the first reinforcer, cocaine and food maintain equivalent rates of responding. Similarly if a session run under these conditions is preceded by an injection of cocaine, this results in faster responding in both cocaine and food reinforcement conditions. These studies suggest strongly that cocaine is not uniquely reinforcing; rather, the apparent differences between cocaine- and food-maintained behavior result from stimulant effects of the cocaine delivered during experimental sessions (Katz & Goldberg, 1987).

Studies have begun to examine the relationship between drug-reinforced and

conventionally-reinforced behaviors in more complex settings, where both types of reinforcer are concurrently available. It is clear from studies using progressive ratio procedures that drug-reinforced responding is "price sensitive" (i.e., drug intake is suppressed when the work requirement is too high). This leads to variations in drug taking as a function of the relative price and availability of drugs and alternative reinforcers, which can be described with some precision using concepts derived from economics, such as elasticity of demand (Bickel, Hughes, DeGrandpre, Higgins, & Rizzuto, 1992; Hursh, 1980). For example, in an early study, two baboons were found to respond equally to heroin and food when both were in plentiful supply, but as resources became scarcer, heroin showed greater elasticity: food intake was maintained but heroin intake decreased (Elsmore, Fletcher, Conrad, & Soderetz, 1980). Behavioral economics may provide a useful framework for analyzing human drug taking (Heyman, 1996).

The extensive parallels between drug reinforcement and conventional reinforcement in nondependent animals, together with the fact that the construct of dependence is of very limited value for understanding human drug taking (Sanger, 1991), has led some workers to argue that self-administered drugs function primarily as incentives, which are sought for their rewarding properties (Stewart, de Wit, & Eikelboom, 1984). The incentive properties of drugs of abuse are demonstrated by the so-called "priming" effect: in animals trained to self-administer a drug, and then extinguished so that responding decreases substantially, a single drug administration is sufficient to reinstate responding, in a dose-related fashion, despite the fact that responding leads to no further drug injections. Priming effects have been consistently demonstrated with intravenously self-administered stimulants and opiates (Stewart & de Wit, 1987), as well as oral alcohol (Chiamulera, Valerio, & Tesari, 1995), and can be seen following multiple nonreinforced extinction sessions (Shaham, Rodaros, & Stewart, 1994; Chiamulera et al., 1995). Priming effects are seen not only with the training drug, but are also elicited by other drugs, which are effective in reinstating responding to the extent that they resemble the training drug in drug discrimination assays (Stewart & de Wit, 1987).

Furthermore, priming effects can also be elicited by environmental stimuli previously associated with drug infusions (Stewart et al., 1984). Consistent with these findings, in human drug users, stimuli associated with cocaine have been found to elicit cocaine craving in cocaine users, but were without effect in opiate users or

in nondrug users (Ehrman, Robbins, Childress, & O'Brien, 1992). These studies are interpreted as meaning that stimuli associated with abused drugs can acquire conditioned incentive properties. For example, following conditioning sessions in which neutral stimuli are paired with drug infusions, nondependent animals will learn a new response that is reinforced by presentation of the conditioned stimulus (Davis & Smith, 1987). Conditioned reinforcing effects have been studied most extensively in the place-conditioning model (Carr et al., 1989). In this paradigm, animals are typically exposed to one of two distinctive environments in the presence of a drug, and to a different environment in its absence. In a subsequent drug-free choice trial, animals display a preference for environments associated with self-administered drugs, with few exceptions (notably, alcohol), while avoiding environments paired with drugs that are presumed to have aversive effects, such as lithium chloride or the opiate antagonist, naloxone (Goudie, 1991). A major methodological advantage of these conditioning procedures is that reinforcing effects are studied in the absence of the drug, which avoids problems associated with motor or other nonspecific drug effects.

In contrast to this focus on the euphoric properties of abused drugs, the traditional view of drug dependence has emphasized the importance of drug tolerance, physical dependence, and the avoidance of withdrawal distress as a major fact in the maintenance of continued drug use. It is well established that the induction of physical dependence leads to an increase in drug self-administration in animal and human subjects (Cappell et al., 1987; Griffiths, Bigelow, & Henningfield, 1980; Griffiths, Bigelow, & Liebson, 1986). There is also some evidence from animal models that the induction of physical dependence increases not only the quantity of drug consumed, but also the reinforcing efficacy of drug reinforcers in self-administration paradigms. However, the evidence is equivocal. For example, in studies using a progressive ratio schedule to assess relative reinforcing efficacy, induction of physical dependence increased responding maintained by codeine or morphine, but not by another opioid, loperamide, which has minimal abuse potential in humans, or by alcohol (Yanagita, 1987).

Other effects of withdrawal can be readily demonstrated within the self-administration paradigm. For example, in a classic study, rats were maintained in a procedure in which periods of morphine availability alternated with periods in which lever pressing resulted in food availability or in avoidance of electric shock.

The withdrawal of morphine led to pronounced decreases in both food deliveries and successful avoidances (Thompson & Schuster, 1964). This phenomenon, known as behavioral dependence, is a sensitive index of withdrawal, since the disruption of operant behavior maintained by conventional reinforcers can be observed following withdrawal from treatment regimes, with a variety of drugs, that produce few if any overt signs of distress (Balster, 1985). Indeed, experiments of this kind provide strong evidence for a brief cocaine withdrawal syndrome, which is still unrecognized by many clinicians. For example, the threshold for brain-stimulation reward is markedly elevated, indicating anhedonia, following withdrawal from as little as 12 hours exposure to cocaine self-administration (Markou & Koob, 1991).

A number of lines of evidence confirm the aversive nature of drug-withdrawal states. The most direct evidence is that morphine-dependent monkeys learn to respond to a conditioned stimulus so as to avoid or delay infusions of an opiate antagonist (Goldberg, Hoffmeister, Schlichting, & Wuttke, 1971). Indeed, stimuli associated with drug withdrawal in dependent subjects, which, typically, have been repeatedly precipitated, in the presence of the conditioned stimulus, by administration of a pharmacological antagonist, can acquire a powerful control over behavior. For example, stimuli or places paired with opiate antagonists can elicit signs of physical withdrawal in morphine-dependent rats (Wikler, 1973), monkeys (Goldberg, 1976), and humans (O'Brien, 1975), which can be seen many weeks after drug withdrawal, and long after offset of the withdrawal period. Like withdrawal itself, such stimuli are aversive, as demonstrated, for example, in place conditioning experiments (Hand, Koob, Stinus, & leMoal, 1988).

### **1.07.5.3 Limitations**

It is clear from this brief review that there have been significant advances in understanding the factors that control the initiation and maintenance of drug self-administration, and many of these factors are of proven relevance to human drug taking. However, a significant limitation on the face validity of animal models of addiction arises from the impoverished environments in which animals typically self-administer drugs. There is evidence that drug taking is decreased by testing animals in social groups (Alexander, Beyerstein, Hadaway, & Coombs, 1981), and as discussed above, it is clear that another potential source of enrichment, the availability, and conditions of availability, of alternative sources of reinforcement,

can markedly influence drug taking. Human drug taking typically involves a substantial element of social ritual and cultural conventions, and a high degree of choice (Hartnoll, 1991). These factors may be relatively unimportant in studies of the biological bases of drug action, but may be crucial if the object is to understand the environmental controls on drug-taking behavior. It is tempting to draw the comparison between the environmental impoverishment typically imposed on experimental animals and the conditions of social deprivation that characterize many communities in which drug-taking is rife; however, these parallels require detailed evaluation, and should be viewed with caution.

Another area that has received little attention is the process of change in patterns of drug self-administration. For good and obvious reasons, drug self-administration is usually studied under steady-state conditions, in which drug taking is constant over sessions, and well-regulated within sessions. Much of the literature discussed above describes factors that influence drug self-administration under these conditions. Much human drug taking is also controlled and well-regulated, and one of the major challenges is to understand the conditions in which regulation breaks down, leading to uncontrolled, heavy, and typically, problematic substance use. As discussed above, uncontrolled use, including a characteristic human pattern in which drug or alcohol binges alternate with periods of abstinence, can also be observed in animal models, under appropriate experimental conditions, and studies carried out under these conditions are most encouraging (Koob, 1995). However, there has been little attempt to use animal models to study the evolution of controlled into uncontrolled use, and the factors that control those changes.

As noted earlier, the evaluation of construct validity requires a good theoretical understanding of both the model and the condition modeled (Willner, 1991). Unfortunately, both sides of this equation are rarely met; in the present case, drug intake by animals is now well understood (at least under steady-state conditions), but a coherent theoretical understanding of substance abuse by people remains elusive. The motivational framework outlined above provides an excellent, if limited, account of many aspects of drug taking in animal models. This framework is of great heuristic value in guiding the rapid development of an understanding of the neurobiological substrates mediating the rewarding effects of drugs and the aversive effects of withdrawal (Altman et al., 1996; Koob & Goeders, 1989). However, the importance of these constructs for understand-

ing human substance abuse, and the relevance of the neurobiological insights arising from animal models of drug self-administration, remains to be confirmed

#### 1.07.6 OVERVIEW: VALIDITY AND UTILITY OF ANIMAL MODELS

Having surveyed animal models relevant to four areas of psychopathology, it is of interest now to reconsider the general issue of the validity of animal models in relation to the criteria outlined at the start of this chapter, and to summarize their uses. McKinney and Bunney (1969) suggested that an animal model should resemble the clinical disorder in its etiology, treatment, symptomatology, and physiological basis, and these categories provide a convenient framework within which to examine the contribution of animal models to our understanding of the clinical disorders.

As noted earlier, the orientation of animal models towards psychopharmacology has meant that the predictive validity of the models is determined largely by their pharmacological responsiveness, which has not been discussed in detail in this chapter. In general, however, predictive validity is reasonably high for each class of model, though there are exceptions to this general rule at the level of individual models. Animal models of depression can usually be relied upon to differentiate established antidepressants from known inactive agents, though there are some well-known exceptions which are difficult to interpret (e.g., anticholinergic drugs often appear antidepressant-like: see Willner, 1990). However, a multiplicity of novel agents are currently undergoing clinical trials in depression, and it will be some years before the predictive validity of animal models of depression can be assessed in relation to these novel agents. By contrast, animal models of anxiety, traditionally the jewel in the crown among animal models when assessed by their response to benzodiazepines, are currently in the unfavorable position of coming to terms with the anxiolytic and putatively anxiolytic actions of novel serotonergic agents and, rather belatedly, with the anxiolytic actions of antidepressants.

Animal models of schizophrenia remain for the moment unchallenged as regards their predictive validity, as all drug development in this area has involved variations on the theme of dopamine blockade. However, a comparison with the position of anxiety models a few short years ago suggests caution: we do not know how well the current models would cope with the discovery of novel neuroleptics acting through

mechanisms unrelated to dopamine. Animal models of substance abuse perform well in discriminating abused from nonabused substances, but less well when asked to rank different substances for their relative abuse liability.

Given the intrinsic limitation that animal models must be based on behavioral rather than subjective symptomatology, in those cases where efforts have been made to establish face validity, the position is again reasonably encouraging. Core symptoms of the disorder are demonstrable not only in models of depression (e.g., subsensitivity to rewards), anxiety (responses to aversive stimuli), and substance abuse (drug self-administration), but also in models of schizophrenia (bizarre and hallucinatory behaviors). However, a recognition of the diagnostic heterogeneity of the clinical disorders is now becoming a high priority. In the case of schizophrenia, the need for an acceptable model of negative symptomatology has long been explicitly recognized. However, the heterogeneity of anxiety disorders has until recently had little impact on animal models: in comparison to the multitude of tests addressing generalized anxiety disorder, very little thought has been given to the development of animal models of phobic anxiety or panic, and virtually none to obsessive-compulsive disorder. In part, this narrow view reflects the degree to which the field of animal modeling is drug-driven: it is difficult to see any other reason for the scarcity of work on models of phobic anxiety, which is not drug-responsive but lends itself superbly well to the development of models for which face validity may be established. However, domination by drugs is not the whole story: the pharmacological distinction between delusional and nondelusional depressions is well established clinically but has been overlooked in the development of animal models of depression. Rather, the preoccupation with generic disorders of depression and anxiety reflects an unwillingness to look outside the ordered confines of the laboratory at the chaotic richness of the clinical world. This attitude is understandable, but perhaps, short-sighted.

Animal models are at their weakest in the area of construct validity. However, this reflects to a considerable extent the inadequacies of current clinical theorizing. Theories of psychopathology tend to be either strongly psychological or strongly biological in nature, with little attempt to unify the two approaches. Psychologically based theories are well developed in relation to aspects of depression and anxiety, though rudimentary in their account of schizophrenia (see Brewin, 1988). These theories are frequently

couched in language difficult to transport into animal models, though where the possibility exists (as, for example in the learned helplessness model of depression), the response has been enthusiastic. Irrespective of the extent to which the terminology of psychological theories of depression or anxiety may or may not be applied to animals, such theories must be capable of explaining the efficacy of antidepressant and anxiolytic drugs. The fact that cognitive theories of psychopathology cannot explain drug effects is a measure of their current limitations. Biologically based theories of psychopathology take the actions of therapeutic agents as one of their starting points, but beyond this, have made relatively little progress. Few biological abnormalities have been determined with any certainty in the brains of psychiatric patients, and as a result, biologically based theories of depression (Willner, 1985), anxiety (Gray, 1982) or schizophrenia (Frith, 1987) are derived in large part from research using animal models (an uncomfortable fact of life that clinicians tend to overlook). While there is an unacceptable degree of circularity in appealing to these theories as support for the construct validity of animal models, it is certainly arguable that the construct validity of animal models in all three areas approaches the limits set by clinical theory. Much the same can be said of substance abuse: the animal literature provides a clear and powerful theoretical framework for understanding drug self-administration, but the human literature is marked by an equally striking lack of theoretical consensus (e.g., Altman et al., 1996).

Perhaps surprisingly, animal models have little to say about the etiology of anxiety. Potentially important insights about the etiology of phobias arise from the studies of observational conditioning of fear in monkeys (Mineka & Cook, 1986), but as far as generalized anxiety disorder and panic are concerned, animal models of anxiety provide no insights into why some people respond normally in situations that generate pathological anxiety in others. Models of depression are more promising, and provide strong support for the view that stress and social isolation are of etiological significance, as well as some pointers to the mechanisms mediating these effects. It may be important to reflect that this research provides clear evidence against the distinction between "psychological" and "biological" depressions, in demonstrating that environmental precipitants can cause profound physiological consequences. The major contribution of animal models to the etiology of schizophrenia has been the demonstration that neuropathological features of the schizophrenic brain, such as



hippocampal abnormalities or ventricular widening, could indeed be of etiological importance. Animal models of substance abuse have focused to a certain extent on factors determining individual differences in susceptibility to drug or alcohol self-administration, and have identified clear genetic influences and hormonal variables that could be relevant to human drug taking.

It seems likely that animal models may play an important role in the elucidation of relationships between etiology and symptomatology. In particular, the demonstration that stress can cause insensitivity to rewards may be of great significance for understanding melancholia; there is little in the clinical literature to predict this relationship, which clearly deserves further investigation. The generation of positive, but not negative, schizophrenic-like symptoms by hippocampal lesions similarly suggests that specific etiological factors may relate to specific symptom patterns. In addition to their use in the experimental investigation of problems related to etiology, animal models may also be of value in defining diagnostic boundaries. The current tendency to obscure the border between depression and anxiety has arisen primarily from clinical observations (for example, that antidepressants are also anxiolytic), but has long been prefigured in the involvement of serotonin in animal models of both disorders. Work with animal models has led to a unifying concept that serotonergic systems function to suppress the generation of impulsive behavior (Soubrie, 1986; Willner, 1989), which is of major importance for understanding a spectrum of disorders including depression, anxiety, bulimia, alcoholism, and obesity, in which serotonergic transmission is abnormal and specific serotonin reuptake inhibitors are therapeutically effective.

Animal models were initially introduced, during the behaviorist era, as a means of developing behavioral therapies for psychological disorders, and this remains the arena in which animal models are best known to many clinical psychologists. However, this use of animal models is very little evident in recent or current work. Indeed, it is difficult to see any contribution to behavioral psychotherapy arising out of the current models surveyed in this chapter in the areas of depression, anxiety or schizophrenia. The exception is in the area of addiction, where the demonstration that stimuli conditioned by association with abused substances can acquire motivational properties in their own right has led to attempts to treat addictions by cue exposure therapy, which attempts to extinguish responses to drug-associated cues (Drummond, Tiffany, Reming-

ton, & Glautier, 1995). Nevertheless, animal models do contribute in major ways to the treatment of psychopathology. These contributions arise from their use in drug screening programs and their role in determining the mechanisms of action of psychotherapeutic drugs. This does, however, represent a significant change of direction: animal models were initially located within mainstream psychology, but now serve primarily as one of the major foundations of psychopharmacology.

In the future, animal models will have their greatest impact in relation to the physiological mechanisms underlying psychopathology. This is a rich seam of research, which derives not simply from the models themselves but also from the ways in which research in animal models relates to the broad spectrum of research in behavioral neuroscience. In effect, animal models form a critical interface between psychiatry and basic research in behavioral neuroscience; they are the channel through which developments at the basic level are brought into clinical perspective. Animal models serve both as theories of psychopathology and as practical tools for its investigation. Their development is an iterative process: the investigation of an animal model provides hypotheses concerning the clinical condition, and the outcome of the clinical investigations provides the basis for a more refined assessment of the validity of the model. Viewed in this light, the major limitation of animal models, their preoccupation with the objective aspects of psychopathology rather than the subjective, may actually be something of an advantage; it forces the development of a linguistic framework within which the psychological and biological dimensions of psychopathology may ultimately be reconciled.

### 1.07.7 SUMMARY

This chapter reviews animal models of depression, anxiety, schizophrenia, and substance abuse. Although much of the review, of necessity, deals with drug effects in animal models, the focus throughout is on animal models as simulations of psychopathology, rather than on their utility as drug development tools. Even in this psychological context, the models in current use relate primarily to psychopharmacology rather than to mainstream psychology, and their principal use is in investigations of the physiological mechanisms underlying psychopathology. In contrast to their historical roots, animal models of psychopathology now make relatively little contribution to the development of psychotherapies. For

each of the disorders reviewed, models, or classes of model, are assessed for the extent to which they meet the criteria of predictive validity (ability to predict from the model to the clinic), face validity (extent of phenomenological similarity), and construct validity (theoretical rationale). In general, the results of this assessment are quite encouraging.

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# 1.08

## Cultural Dimensions

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## 1.08.1 INTRODUCTION

### 1.08.1.1 Anthropology and Psychology

The collaboration between anthropology and psychology is a recent chapter in the brief history of the behavioral sciences. The interest of anthropologists in the problem of mental illness originated in their concern to understand the relationship between culture and personality. In the early 1930s Margaret Mead, Edward Sapir, and Ruth Benedict were raising questions of how anthropology could inform the understanding of the genesis of the human personality.

From 1936 to 1940 the psychiatrist Abraham Kardiner with a group of anthropologists, the most prominent of whom were Cora DuBois, Ralph Linton, Edward Sapir, and Ruth Benedict, were conducting seminars in culture and personality. A spinoff of this seminar was the first comprehensive cross-cultural research program conducted on the interface between anthropology and psychology.

The investigation was carried out on the Indonesian island of Alor under the leadership of DuBois. "We had talked ourselves out, and only field work could test the procedure," she wrote in her introduction to *The people of Alor* (DuBois, 1944), a landmark in cross-cultural psychology. The scope of this investigation was, in her own words, to find out if there was a "demonstrable" relationship between the personalities of adults within a group and the sociocultural milieu in which they lived. If such a relationship were found to exist, its explanation was presumed to lie in the consistency of life experiences ranging from the earliest child-rearing practices and relationships to the reinforcing effects of adult institutions and social roles (DuBois, 1944).

The interest of these pioneers in cross-cultural anthropology, however, was not primarily clinical. In the school of thought initiated by DuBois and her collaborators which flourished from the 1930s and well into the 1950s, the question of the "normal" and "abnormal" and the definition of illness in various societies was not the main issue. The Holy Grail of their investigations was "basic personality structure"—a term coined by Abraham Kardiner or "modal personality" in the language of DuBois. Yet it is these early investigations that have been the main source of inspiration for the current interest in the relationship.

Although the term "clinical psychology" was introduced by Lightner Witmer in 1896, several decades had to pass before the role of the clinical psychologist gradually emerged. The participa-

tion of psychologists in mental institutions and clinical work was restricted to psychometrics and mental tests in a team which, in addition to the psychologist, consisted of a psychiatrist and a social worker. The psychologist had an academic training which in the beginning did not include training in psychotherapy and clinical methods, although the Department of Psychology at Columbia University established a curriculum for clinical psychology in 1936. But formal, systematic training of clinical psychologists in the USA did not get off the ground before the 1940s when the psychological damage caused by wartime experiences primarily among soldiers became a major social problem among the more than 16 million World War II veterans which were added to the 4 million from the previous war.

### 1.08.1.2 The Birth of Clinical Psychology

The time was ripe for clinical psychology, but not yet for a closer collaboration between anthropology and clinical psychology. In the landmark event in the history of clinical psychology which took place in Boulder, Colorado, in the summer of 1949 where the "Boulder model" of clinical training was formulated, neither anthropology nor cross-cultural studies were mentioned in the core curriculum proposed by the conference.

### 1.08.1.3 The Construction of Social Reality

The relevance of anthropology for clinical psychology thus has only recently been recognized by the community of scholars, and a consistent training program has yet to be formulated. The most obvious potential of cultural understanding to the field of clinical knowledge is in the realm of *social reality*. A few epistemological comments are called for in order to clarify this concept which is essential to the postmodern understanding of culture which is at the cutting edge of anthropological theory today.

There is an unresolved disagreement between proponents of the idea that all phenomena can be studied by the same scientific methods and scholars who maintain that there are phenomena of different orders which call for special methods. The first group subscribes to a theory of unity of science and is sometimes, in colloquial discourse, referred to as positivists or logical empiricists. In the social sciences in general and in psychology in particular the positivist approach is referred to as behaviorism.

The second group maintains that there are certain phenomena which cannot be studied by

positivistic methods but must be approached through a special form of understanding colloquially referred to as hermeneutics. Psychoanalysis is an example of a system of understanding based on the hermeneutic method.

The idea of social reality is based on the observation that social life is not of the same order as the physical universe which has an existence independent of human socializing. Social life on the other hand is created or acted out in a social process. In the words of Berger and Luckman (1969) it is "constructed." Social reality is constructed in the process of everyday life through role play following an intricate system of rules. A pioneering study of human interaction was carried out by Goffman (1959).

The anthropological understanding of culture has been strongly influenced by the constructionist approach. The concept of culture developed by the Columbia group under the leadership, of among others, Margaret Mead and Ruth Benedict reflected taxonomic ideas developed in the study of species. These taxonomic ideas are based on the fact that, in the fauna and flora, distinct species can be recognized as isolates with clear boundaries. Although the classification of cultures was not explicitly organized according to the taxonomic paradigm of species, cultures were to a certain degree, regarded as isolates with their distinct and recognizable patterns.

This taxonomic thinking strongly influenced the culture and personality school of thought which flourished in the 1940s in the study of national character. These studies clearly demonstrated that the social sciences are involved in political agendas. The national character studies started as part of the war effort and were designed to understand, and to a certain degree, predict the behavior of the main adversaries—the Germans and the Japanese, and later during the cold war, the Russians. In spite of the political agenda, some of the studies testified to a high quality of scientific craftsmanship. An example of this is Ruth Benedict's *The chrysanthemum and the sword* (1974) in which she presents a study of Japanese culture and personality.

Although cultures are no longer regarded as species, there are definite relationships between culture and personality. Distinct cultures can be recognized, but the focus is now on the nature of their boundaries. The boundaries between species are genetic, a fact which makes them clear isolates. The boundaries between cultures are maintained by entirely different processes. The boundaries are not natural entities, but constructions maintained by complex symbolic efforts of self-ascription, rituals, and social sanctions. The focus is now on ethnic groups

and boundaries rather than cultures. This approach was launched with the publication of the influential work *Ethnic groups and boundaries* by Fredrik Barth (1969). The question of ethnic groups articulates with the problems of personal identity which is an important issue in clinical psychology. One of the important interfaces between anthropology and clinical psychology is located in the symbolic space of identity management.

## 1.08.2 THEORIES OF ILLNESS AND DESTINY

Every human group that has been studied by anthropologists has developed its own form of knowledge to cope with the everpresent adversaries of our existence: disease and misfortune. Two of the pioneers of medical anthropology George M. Foster and Barbara Gallatin Anderson classified theories of illness in technologically simple societies into "personalistic" and "naturalistic" theories (Foster & Anderson, 1978). This classification corresponds to the classification later suggested by George Peter Murdock in his last comprehensive cross-cultural investigation, *Theories of illness. A world survey* (1980). Murdock's investigation is based on the human relations area files at Yale University. On the basis of these files Murdock surveyed theories of illness in 186 human groups or cultures in the language of the 1980s.

### 1.08.2.1 Theories of Natural Causation

Murdock identified four main theories of natural causation:

(i) *Infection*. Defined as the invasion of the victim's body by noxious microorganisms. It refers particularly to the germ theory of disease which seems to be unique to Western medicine. But some groups have knowledge and understanding of the fact that small organisms like worms and insects may cause disease.

(ii) *Stress*. From the point of view of clinical psychology it is interesting to observe that exposure of victims to psychic strain such as worry, fear, and emotional distress is recognized as a major cause of illness in a large number of groups. Among the Javanese and the Pawnee anxiety is recognized as the major threat to health.

(iii) *Theories of deterioration*. Murdock observes, not without irony, that physical deterioration through old age is not generally recognized as a major cause of illness among human beings. According to Murdock, most of mankind considers itself potentially immortal

and is unable to conceive of the infirmities of old or middle age as being caused by the process of aging itself rather than the interference of some hostile agent or force.

(iv) *Accident*. The fact that accidents are rarely regarded as the causes of illness and misfortune indicates that the very notion of accident is a recent philosophical idea in human understanding. The general tendency is to personalize the causation of what we in the modern Western world term accident and to attribute it to supernatural intervention.

### 1.08.2.2 Theories of Supernatural Causation

Through his comparative analysis Murdock came up with three basic groups of supernatural causation: theories of mystical causation, theories of animistic causation, and theories of magical causation. Of the three, the theories of mystical causation may be regarded as the most sophisticated, entailing as they do impersonal agents as the culprits. In certain ways they are closest to modern theories; particularly the belief in contagion is very close to our notion of infection as if the mystic theories by some sort of intuition forecast the breakthrough of modern medicine. Since the mystical theories are less concerned with social forces than the other explanations, they are at first glance, of less interest to the anthropologist and have received much less attention and are also comparatively rare—but not of less theoretical interest as Mary Douglas has shown in her fascinating study *Purity and danger* (1966).

More widespread, however, are the theories of animistic causation, which imply the existence of personalized supernatural entities. In accordance with this kind of explanation, disease may be caused by the occasional departure of a person's soul, as in the cases of soul loss, but much more frequently through spirit-aggression. According to Murdock's investigation, this is the most frequently encountered type of explanation.

Theories of magical causation are also found in a large number of different cultures and are manifest in two main forms: witchcraft and sorcery. For several reasons, not all of them strictly professional, these twin phenomena have been the favorite of anthropology. One reason is possibly that witchcraft and sorcery loom with sordid prominence in more recent Western history. The topic appeals to the peculiar kind of black fascination which human tragedy and catastrophe are singularly capable of arousing. It provides its audience with the combined advantages of participation and immunity, which never fails to rally the crowds also in our age. It gives modern people that

sense of intellectual achievement which sustains the interest in the follies of the recent past. Clearly supernatural ideas also have a special appeal to anthropological field-workers because it was a prominent theme in the heroic age of discovery at a time when anthropologists regarded technologically simple communities as primitive. Although "primitive" has developed a semantic of its own, and today is regarded as a pejorative term, its lexical origin is the Latin term *primus*, which means first. The term primitive indicated that the scholars in the days when words like primitive and savages did not raise an eyebrow even among the most enlightened, were of the opinion that they were studying humankind in its pristine or original state of being. In a way the opinion was that they were investigating our common past.

### 1.08.2.3 Types of Mystical Causation

#### 1.08.2.3.1 Fate

In the realm of mystical causation fate is of particular interest. It was regarded as the major determinant of illness among the ancient Romans. Fate may be regarded as the precursor of our modern idea of accident or chance. In Roman times fate was understood as the influence of astrological influence, individual predestination, or ill luck. In our modern idea of accident the connection to astrology and occult forces has been suppressed although it is still alive in many folk beliefs which can be ascertained by anyone who cares to consult the horoscopes of popular magazines. It is a sophisticated idea which is primarily found in comparatively complex societies such as Turkey, Burma, Thailand, and Java.

#### 1.08.2.3.2 Ominous sensations

This type of theory is of particular interest because it gives prominence to the impact of potent dreams which has been recognized by modern psychoanalysis. In addition to dreams, impressive sights and sounds may cause illness. Dreaming of a recently deceased relative is an ominous sign among the Massa. Depending on the circumstances such dreams may be important events during psychoanalytic treatment and are another example of the intuitive wisdom sometimes encountered in knowledge systems of technologically simple societies.

#### 1.08.2.3.3 Contagion

The word suggests an incipient understanding of the microbiological transfer of disease. But the mystical concept of contagion which is fairly

widespread and attested to by 49 societies in the Murdock sample is of a different order altogether. The mystical contagion refers to fear of the ritually impure, like menstrual blood and corpses. Although the fear of corpses could have a rational dimension in terms of microbiology, the same does not hold for menstrual blood. It is a question of pollution rather than infection and is cognitively related to the purportedly polluting categories of people, such as the “untouchables” of India.

This widespread theory of disease is of considerable interest to clinical psychology because it testifies to a human, cognitive disposition which has been explored by Douglas (1966).

Douglas observes that the impure in cognitive terms is matter out of place. In the physical world order it is recognized by everything in its proper place; in the moral order, impurity is equal to the lack of observance and respect for categories. The classical example is the taboo on pigs’ meat which according to Douglas has nothing to do with an assumed archaic knowledge of microbiological hazards, which is the common folk explanation, but with the categorization of animals into ruminating and nonruminating types. This is written quite explicitly in the Old Testament where it is maintained that the pig is unclean because it has split hooves but does not ruminate. This is an important distinction to people of archaic animal husbandry.

Another example is the fear of homosexuality in societies with rigid sex-role segregation. To regard the homosexuals as unclean is not in accordance with modern rationality, but is of the same order as the impurity of untouchables.

Although Westerners, at first sight, regard the contagion theories of other societies as “superstitious” and “irrational,” an understanding of the cognitive disposition revealed by Douglas demonstrates that some of our Western theories of illness are merely posing as rational. The fact that homosexuality is no longer regarded as a disease or pathological condition in the USA demonstrates that some of our own theories of illness, at least until quite recently, were based on ideas of irrational impurity.

#### **1.08.2.3.4 Mystical retribution**

The idea that disease is a retribution for the violation of moral injunction or taboos is fairly widespread. The most common of the taboos are food, sex, as in the case of incest, and verbal.

Again it is possible to observe that ideas of mystical retribution are not entirely absent in Western society. It is, for example, being maintained by some religious leaders that AIDS

is a divine retribution for the violation of sexual taboos.

Food taboos are common among certain ethnic groups in modern Western societies. The origin of these taboos is generally not known and poorly understood. Yet they are observed with care by people who subscribe to these taboos and great discomfort is to be expected in case of violations. It is important for therapists to understand the power of taboos and beliefs. These beliefs and their effects clearly demonstrate that social reality is a construction and reminds us of the fact that human beings are under the spell of different constructions.

Verbal taboos are recognized in our rules against swearing. But, the fear of swearing has lost most of its mystical force and among modern people is regarded merely as bad manners whereas in some societies the breach of verbal taboos may be regarded as the cause of illness.

### **1.08.2.4 Theories of Animistic Causation**

#### **1.08.2.4.1 Soul loss**

A common idea among human beings is the existence of an insubstantial double or soul which inhabits the body, but is independent of the physical and territorial restrictions which apply to the physical body. This soul may depart from the body during sleep and permanently at death to lead a separate existence in afterlife. If the soul departs from the body on more than minor excursions during sleep, it may cause illness.

The belief in soul loss is often combined with the institution of shamanism, which has recently attracted some attention among psychotherapists as pointed out by Strathern (1995). Strathern points out that in recent developments in psychotherapy altered states of consciousness have become a main topic of interest. He observes that:

Trance facilitates such a reframing (of experience), as does spirit possession. At one end we may be dealing with a new image presented to a patient, at the other with a whole new persona, an alter ego juxtaposed to the self. Either a metaphor or a historical figure may be in focus: in either case the result is an altered state of consciousness which whether trance induced or not conduces towards a patient’s recovery, a process in which health and identity are brought into consonance with each other. (Strathern, 1995, p. 128)

The term shaman is of Siberian Tungus origin, but the shaman is a well-known character in “anthropological” societies. He or she is a curer who has a special relationship with the

spirit world. During consultations, the shamans fall into trance in which they communicate with their tutelary spirits in order to understand the cause of the illness.

The recent interest in shamanism and trance among anthropologists is caused by a major paradigmatic change in the field of cultural understanding. In the classical tradition in the study and interpretation of the personality, the personality was regarded as a reflection in the individual mind of the society and its institution, or in other words: its culture. With the constructionist approach the self was introduced as a concept. Whereas the personality is imposed on the individual by society, the self is an active agent reacting to and sometimes even rebelling against the demands of society. The concept is difficult to operationalize and seems rather nebulous to ardent empiricists. Nevertheless it has acquired some respectability in mainstream anthropology.

#### 1.08.2.4.2 *Spirit aggression*

This is the most widespread of all the theories of supernatural causation. The major types of supernatural aggressors are nature spirits, disease demons, or lesser divinities. Among the aggressors are also found departed ancestors or kinsmen, ghosts and in some instances deities or gods. The thought is not alien to Christian civilization as is testified in the Bible by the book of Job.

#### 1.08.2.4.3 *Magical causation*

As pointed out above, witchcraft and sorcery looms with sordid prominence among the theories of illness and destiny in comparatively recent Western history. In the case of sorcery an active human agent uses various techniques to hurt another human being. The main techniques applied by sorcerers are supposed to be verbal spells, prayers, and curses; object intrusions; ritual manipulation on exuvias and belongings of the intended victim; and administration of poisons.

The distinction between rational procedures known to be effective within a scientific paradigm of understanding and irrational procedures is not always clearcut. This is particularly the case with the application of poison. Sorcery has an instrumental dimension which may be reconciled with the modern world view. The difference between the biochemical effects of certain medicines and the occult effect of the medicines of sorcerers is not always obvious to people with no scientific training. Beliefs in sorcery may therefore be quite resilient.

#### 1.08.2.4.4 *Witchcraft*

In witchcraft beliefs it is assumed that some human beings possess an evil force that may cause disease and misfortune. The force is supposed to be activated automatically, sometimes even without the conscious knowledge of the witch, as demonstrated by Evans-Prichard in his classical monograph *Witchcraft, oracles and magic among the Azande* (1937).

Sorcery on the other hand is a technology of evil. A sorcerer can apply an assortment of procedures based on what we may refer to as irrational knowledge to harm people. A sorcerer is a specialist and may learn his craft.

Witchcraft and sorcery are often combined and, one is tempted to say, confused with one another. In strict anthropological terms, the witches of popular belief in Europe were really sorcerers, who sometimes were supposed to have acquired their evil powers from the Devil himself.

The belief in witchcraft is practically universal in the Circum-Mediterranean region, but is comparatively rare elsewhere in the world except Africa. The prominence of witchcraft in European and American history and thinking is due to the fact that witch craze is a phenomenon primarily of Western culture.

Belief in witchcraft does not always produce witch crazes. In many African societies, witchcraft is regarded as a normal phenomenon, as among the Azande, and does not produce the kinds of moral panic which is characteristic of early modern Europe.

Although popular opinion has it that witch-craze was a social phenomenon of the Middle Ages, its heyday was in the Renaissance. As the British historian Trevor-Roper has pointed out, belief in witchcraft and sorcery in the sixteenth and seventeenth centuries, a period which we imagine as the age of reason, was not

lingering ancient superstition, only waiting to dissolve. It was a new explosive force, constant and fearfully expanding with the passage of time. It was forewarned by the cultivated popes of the Renaissance, by the great Protestant reformers, by the saints of the Counter-Reformation, by the scholars, lawyers, and churchmen of the age of Scalinger and Lipsius, Bacon Grotius, Bernalle and Pascal. (Trevor-Roper, 1970, p. 121)

The craze also hit the USA in Salem, Massachusetts, in the seventeenth century and many completely innocent people were executed.

Witchcraze is of particular interest to clinical psychology as an example of a collective paranoia. Contrary to popular opinion the days of moral panics are not over and it is important that psychologists are aware of the

background and dynamics of moral panics because they are sometimes called to the assistance of victims of irrational or pseudorational persecutions.

Although we have not yet found an uncontroversial understanding of moral panics and the rise of the witchcraze in the Renaissance, we have gained some insight into the relationship between social systems and types of explanation of disease and misfortune.

### 1.08.3 SOCIAL SYSTEMS AND TYPES OF EXPLANATION

Lewis has pointed out that it may sometimes be more illuminating to discard the culturally grounded expressions "spirit-possession" and "witchcraft" altogether and think in terms of oblique and direct mystical attacks (Lewis, 1971). It has been demonstrated by Brogger (1992) that the nature of oblique and direct mystical attacks clearly relate to the nature of dependency between an individual and the community. The crucial dimension seems to be ascribed vs. acquired relationships and statuses on the one hand and dyadic vs. corporate relationships on the other. Ascribed relationships, of course, are those an individual acquires at birth and as a member of his or her community. In small, technologically simple societies, there is little scope for choice and contractual relationships are few. The dominant mode of the traditional village is the ascribed relationship, that of the urban society the acquired form. Dyadic relationships are limited to *one* other person, corporate relationships to "corporations" such as a lineage, an age grade in technologically simple societies, or in modern societies to trade unions or political parties. These dimensions are represented in Table 1.

Acquired dyadic relationship is characteristic of the modern urban situation. In societies where the essential necessities of life are acquired through ascribed, dyadic relationships, witchcraft beliefs will tend to prevail. This does not exclude sorcery, which may be found as a residual category. Where ascribed, corporate relationships dominate, spirit aggression seems to prevail. In cases where acquired corporate relationships dominate, (as in the

modern, bureaucratic state), political demonology may develop as part of the belief-system.

Although the table does not cover all the possible explanations of illness and misfortune its focus on the experiential dimension in exchange relationships adds a comparatively unexplored dimension to the role of economics in the patterning of culture and society. It directs our attention to the cognitive repercussions of the rise of bureaucracy and the introduction of money and markets.

These institutions may have been a prerequisite for a rational understanding in the field of human relationships and therefore the main force behind the breakthrough of scientific thinking in general and of a rational theory of illness in particular.

In systems in which the majority of transactions are sustained within a framework of clearly personal relations, the human intellect easily jumps to the conclusion that people or clearly recognized groups of people not only have economic power, but power over destiny in general and thus over diseases and indeed over life and death. On the basis of a general experience of social life under these conditions, ideas of witchcraft, sorcery, and even spirits do not seem far-fetched.

The general breakthrough of the scientific theory of illness therefore owes as much to the development of market economy and bureaucratic management as to knowledge itself. The fact that many, even when exposed to the ideas of modern medicine, do not resign their beliefs in witchcraft and sorcery clearly demonstrates that knowledge which does not articulate with the experiences of everyday life remains esoteric (Brogger, 1971).

Dependency on the depersonalized forces of bureaucracy, money, and markets, however, does not irreversibly guarantee rational explanation of disease and misfortune. In spite of many impressive achievements, it is as true as ever that when struck by misfortunes and disease, human beings look for someone to blame. As in former times, one's perspective is determined by the nature of one's dependencies. The age of demonology thus is not behind us, only its vocabulary has changed.

Although the modern human being's freedom of fancy with regard to health is restrained by the insights of modern medicine, one is as free as ever to speculate on the social causes of destiny. In tune with the nature of one's dependency, demonology does not operate on the matrix of face-to-face relationships, but on the level of institutions and social categories like race and class. The demonological qualities of national socialism (Nazism) are universally recognized, but this is not the only example. Whether

**Table 1** Corporate and dyadic relationships

	<i>Dyadic</i>	<i>Corporate</i>
Ascribed	witchcraft	spirit-aggression
Contractual	sorcery	political demonology

modern witchhunts are based on the assumed conspiracies of “world Jewry” or the “bourgeois capitalist” the effect on its victims is the same: suppression, persecution, and often extermination. Although an anthropology of knowledge cannot promise to free the world from the savagery performed in the name of irrational ideologies, it may provide knowledge which, biding its time, may deprive aggressive ideologies of some of their demonic allure.

These anthropological insights have an important message to clinical psychology because they are a reminder of the forces of irrationality in mental life. It is of particular importance to appreciate that modern rationality is a world view or *Weltanschauung* which has developed under particular social circumstances. Although the majority of people in the Western world regard the scientific world view as true in an absolute sense, anthropology reminds us of the great variety of world views developed in different societies as demonstrated in the varieties of explanations of destiny and illness.

At the same time social science demonstrates that even scientific rationality is merely “skin deep.” Behind a front of rationality, human passions often subvert the conquests of scientific reason. It is often the task of the therapist to clarify how egocentric irrationality is the cause of psychological disturbance, and it is sometimes the duty of social scientists to represent the voice of reason in the public domain in situations where moral panics threaten justice and human dignity. Anthropological insights may also threaten our somewhat ethnocentric convictions of having found the master key to truth by raising the question of whether our world view is merely a belief system among many others developed by human civilizations.

### 1.08.3.1 Non-Western Systems of Understanding

#### 1.08.3.1.1 Complex societies

The various theories of illness presented in the global overview of Murdock testify that all human groups develop disease theories no matter how simple. In the evolution of knowledge, which in an anthropological perspective cannot be understood without attention to the development of political systems, more complex systems of understanding have developed and gained acceptance and influence in wide areas.

The interdependence between knowledge and social institutions is often overlooked as if knowledge has an independent existence outside the sphere of human organization. The fact is that the crystallization of medical knowledge into comprehensive systems has

followed in the wake of political empire building. Medical knowledge of course is only a special case, knowledge in general is dependent upon the power and communication structures of societies.

No matter how unpopular empires are in the political thinking of everyday life in the Western world, their importance for the development and management of knowledge is enormous.

The consequences of the Arab expansion after the seventh century AD is today common knowledge and therefore a convenient example. Through the medium of the Arab language and lines of communications created by the Arab rulers, the fund of knowledge accumulated in three of the great civilizations of the Occident and the Near East—The Byzantine, the Greco-Roman, and the Persian—were brought together. In the wake of these events was written an important chapter in the evolution of Western medicine. The knowledge reached Europe primarily through Moorish Spain. At the universities in Europe the medical text, *Canon of medicine*, by the Persian scholar Ibn Sina (980–1037), known by his latinized name Avicenna, was part of the curriculum for several hundred years and helped to set the stage for the development of modern Western medicine.

The great medical traditions of Greek, Indian, Arab, Chinese, and American societies will be explored in the following pages.

#### 1.08.3.1.2 Early Greek medicine

One of the basic ideas in Greek medicine is the role of the bodily “humors.” The theory of illness based on the so-called humoral pathology is known to us through the writings of Hippocrates (born around 460 BC), the father of Western medicine. According to Hippocrates, health is a state of equilibrium between the four humors with their associated qualities: blood (hot and moist); phlegm (cold and moist); black bile, also known as *melancholy* (cold and dry), and yellow bile or *choler* (hot and dry).

In the text attributed to Hippocrates we are told that

The human body contains blood, phlegm, yellow bile and black bile. These are the things that make up its constitution and cause its pains and health. Health is primarily the state in which these constituent substances are in the correct proportion to each other, both in strength and in quantity, and are well mixed. Pain occurs when one of the substances presents either a deficiency or an excess, or is separated in the body and not mixed with the others . . . The four humors have specific and different names because there are essential differences in their appearance. They are dissimilar in their qualities of heat, cold, dryness

and moisture ... Phlegm increases in the winter because as the coldest humor, it is most in keeping with the winter. During the spring the quantity of blood increases, taking charge of the body during the summer and autumn. The hot and cold summer weather is conducive to yellow bile, but, as the cool and dry autumn comes on the bile is cooled, and the black bile preponderates ... The physician must bear in mind that each disease is prominent during the season most in keeping with its nature. (Chadwick & Mann, 1950, pp. 204, 206, 207, 208)

Hippocrates also specifies cures.

Diseases caused by over-eating are cured by fasting; those caused by starvation are cured by feeding up. Diseases caused by exertion are cured by rest; those caused by indolence are cured by exertion. To put it briefly, the physician should treat disease by the *principle of opposition* to the cause of the disease according to its form its seasonal and age incidence, countering tenseness by relaxation and vice versa. This will bring the patient most relief and seems to me the principle of healing. (Chadwick & Mann, 1950, p. 208)

The most important organs of the body according to early Greek medicine were the heart, the brain, and the liver. These organs were respectively dry and hot, moist and cold, and hot and moist—the healthy body was thought to have an excess of heat and moisture. But each person had his or her particular temperament according to the personal prominence of bodily humors: The “sanguine,” cheerful and optimistic; the “phlegmatic,” calm and somewhat slow and sluggish; The “bilious,” ill-tempered and indeed choleric; and the “melancholic,” sad with a tendency for depression and in fact melancholic.

The resilience of the Hippocratic medicine is immediately obvious to us as we recognize that the term “temperament” relates to the notion of temperature in the theory of humor pathology and the categories of temperament are still maintained in everyday language in the West.

The therapies were also logically derived from the theory and were directed against a re-establishment of an humoral balance by means of diet, purging, vomiting, bleeding, and cupping—therapies which are still part of folk medicine in the Mediterranean.

The theory of humor pathology foreshadowed or may be influenced the direction of thought in Western medicine where today the biochemistry of the body is a promising field of investigation and therapy.

In the field of psychiatry, the German psychiatrist Ernst Kretschmer developed a typology of temperaments where somatic characteristics of the body take the place of

the Greek humors. Kretschmer recognized three main physical types: pyknic, athletic, and asthenic (leptosome) and added a fourth “mixed” type, the dysplastic. The pyknic type is round in form and has a greater quantity of fatty tissue compared to the leptosome who tend to be relatively thin and of linear build. The term athletic speaks for itself. Kretschmer identified two main types of psychotic dispositions: the schizoid (expressed in schizophrenia) and the cycloid (expressed in manic-depressive psychosis). In the normal population he identified nonpathological dispositions which he referred to as schizothymia, characterized by introversion, sensitivity, and seriousness; and cyclothymia with a more extrovert disposition, affectivity, and a tendency towards mood swings. According to Kretschmer, pyknic individuals tend to be cyclothymic, whereas asthenic, athletic and dysplastic individuals tend to be schizothymic (Kretschmer, 1925).

In the USA William Sheldon developed a typology according to the same paradigm, although he discarded the strict categorizations of Kretschmer and introduced a taxonomic system based on a systematic study of thousands of college students. He concluded that human beings could be classified under three primary components of body build: endomorphy, mesomorphy, and ectomorphy. The endomorph is reminiscent of the pyknic type characterized by softness, roundness, and a large digestive system. The mesomorph has strong bones and muscles, in other words is athletic. The ectomorph is thin and has a large skin area and nervous system. He recognized three essential dimensions of temperament: viscerotonic, somatotonic and cerebrotonic, assuming that temperament was determined by the somatic disposition (Sheldon & Stevens, 1942).

In this context we are not primarily concerned with the scientific merit of the theories of temperament and personality but in demonstrating the historical background of scientific thought.

A crucial institution for the influence of Greek medicine was the library of Alexandria which was founded in the third century BC. With the manuscripts of this library at his disposal, Galen (130–200 AD) gave the theory of humors its final shape. Although he mainly practiced his medical knowledge in Rome, Galen was an authentic Greek and through his writings Greek medicine conquered oriental Christianity and Islam.

Through the East-Roman orbit, Byzantium, the teachings of Galen reached eastern Christianity and Islam. A crucial role in this transmission of knowledge was played by the



Nestorian sect of Christianity in Edessa in Mesopotamia where Galen's work was translated into Arabic. Near the end of the fifth century the Nestorians had to flee Mesopotamia for political reasons and settled in Gund-Shapur in Persia, present day Iran. A center of medical knowledge was established at Gund-Shapur which strongly influenced Islamic medicine and the Greek theory of humor pathology became part of the Arabic medical tradition. Of particular interest is Gund-Shapur as a mediator between Greek and Indian medicine, the Ayurvedic school.

### 1.08.3.1.3 The Indian medical system

As one of the great civilizations India has developed its own medical system which is part of the health care of modern India. The roots of Indian medicine can be traced to the Vedic writings from the latter part of the second millennium BC. In the *Rig Veda* we encounter the term *bhisaj* a word which later became synonymous with *vaydya* which still today is the Indian term for the traditional curer. In the early period disease was believed to be due to punishing gods and evil demons. We recognize the interpretation of disease in Murdock's theory of *spirit aggression*. The god Varuna was associated with moral ideas and punished those who transgressed his commands.

The idea of healing is associated with the divine twin gods, the Asvins. In the *Atharva Veda* demons are presented as the responsible agents and part of the text is in fact made up of spells to cure disease and promote longevity.

As pointed out by Basham (1976) in the centuries following the *Atharva Veda*, the last six centuries BC, the traditional Indian medical system developed into something like its present day form. What is today known as *Ayurvedic* medicine has come down from Sanskrit documents from the first century BC. The most important of these is *Caraka Samitha*. Caraka is the supposed author of the text, and he may be regarded as an Indian Hippocrates.

By the first century AD a naturalistic system had developed in India. Although beliefs in personalistic causation prevail until this day on the sub-continent, Ayurvedic medicine is a system with scientific and empirical merit. The term may be translated as "the science of living to a ripe age."

According to Ayurvedic theory, the universe is composed of four elements: earth, water, fire, and air. The human body has three fluids or humors, in Indian *dosha*: phlegm, bile and gall, and wind. In a healthy individual the three doshas are in balance or equilibrium, this notion is consequently referred to as the *tridosha*

theory. The doshas relate to the age and the seasons: phlegm with youth and the growing season, bile with middle age and the rainy season, and the wind with old age and winter.

The ideas of temperature are also found in the Ayurvedic circle of ideas. Many foods are thought to have heating and cooling qualities. Hot food, *garam*, include eggs, meat, milk, honey, and sugar; cold foods, *tonda*, include fruit juices, yogurt, rice, and water. These notions are reflected in the theory in which diet has an important part to play.

It is interesting to appreciate that during the period when Ayurvedic medicine reached its mature state, a strict system of socio-religious taboos regulating the menus of the Hindus evolved. There is no indication to the effect that these taboos, which support the hierarchy of castes according to degrees of ritual purity, were developed with the aim of promoting health or avoiding disease. But as Basham (1976) points out, it is surprising how many of the instructions in the *Atharva Veda* would tend to minimize the dangers of infection and food poisoning. "Indian society seems unconsciously to have found a means of remaining healthy as far as possible in a subtropical climate, in its efforts to preserve its ritual purity" (Basham, 1976, p. 19).

This statement recalls the discussion of the religious food taboos in the Western orbit. In our age of reason, we tend to project our rational understanding back to the times when the taboos were codified, times when there was no rational understanding to sustain the dietary habits. The results of the dietary and also hygienic taboos sometimes appear to have had practical effects as if they were based on rational understanding of germs and infection. It is tempting to hypothesize that there is an intuitive wisdom in religious taboos, an intuition which antedates the reflexive understanding of modern science.

The ideas of purity and danger explored by Mary Douglas (1966) demonstrate that the dangers of pollution emerge when essential categories are insulted. The orderliness which these notions promote have obvious prophylactic effects, as is the case when the dead body and things affiliated with it are shunned. The Jewish taboos and hygienic rules definitely served to promote health. Although not the explicit intension of the taboos, they helped to increase the hygienic standards. The Jews shunned stagnant waters, like the wells in the medieval cities, which were sometimes infected. The fact that the Jews made a special effort to secure running water was from our point of view sensible enough. But it cost them dearly because some Christians spread the rumor that the wells had been poisoned by the Jews.

Although too simplistic, ideas of evolution are regarded with suspicion by cosmopolitan anthropology. It is a historical fact that the development of the medical systems both of the Greeks and the Indians followed a trend from the personalistic to naturalistic types of explanation. As we shall see, Chinese and Arabic medicine followed the same developmental trend. It is an empirical fact that naturalist and rational explanations emerged with the development of states and institutions. This does not in itself demonstrate a causal connection between development of complex societies and rationality, but invites a hypothesis to that effect. The secret is most likely hidden in the social organization of knowledge and the rationalization of economy and administration.

The great medical systems of the Orient stopped short of developing the reflexive empirically based medical science of the West. They did, however, develop prophylactic procedures, curing techniques, and mental training programs which are now gradually being recognized as valuable by Western medical science.

Of particular interest is yoga which does not strictly belong to the Ayurvedic curriculum. The term "yoga" has two Sanskrit roots, the first means "to meditate," the second "to join". By joining is understood the connection of the human nature with cosmic sources. The development of yoga as a technique of psychological healing may be regarded as an evolution of early magico-religious beliefs and practices into a rational system. Seen in the light of a theory of knowledge this development is of the same nature as the development of empirical, medical theories on the basis of early, personalistic ideas exemplified by Greek and Indian humoral theories.

Yoga has definitely developed on the basis of religious ideas (Hindu and Buddhist) but is not in itself a religion and is not based on faith, dogma, and sacred texts. Yoga represents a system of mental training developed over millennia by practicing yogis.

The yoga philosophy clearly testifies to its religious parentage. It is based on a rejection of worldly life, an idea shared by Christianity. Its aim is an altered state of consciousness. In the West a general scepticism to yoga has prevailed until quite recently. But, empirical studies suggest that relatively subtle effects produced by different yoga practices can be detected by brain-wave studies (Emerson, 1972).

Underlying yogic practice is the idea of the "subtle body," which consists of a series of interconnected centers called "chacras." The chacras have the ability to process energy from the environment and refine it to a higher level.

Seven major centers are recognized. The third-eye center, for example, is located an inch above the meeting of the eyebrows and relates to insight and understanding. These centers are normally closed and it requires conscious effort on the part of the meditator to open them.

The yogic terminology and the sometimes extravagant claims made on its behalf has made it too easy for Western science to disregard the knowledge as obscure. Its promoters in the West also sometimes have proved to be authentic charlatans who have exploited the existential crises of modern men and women to make money. This, and a tendency towards cultural arrogance, has made it too easy for Western science to disregard insights from Asian psychology. But recently, mental health professionals have started an exploration of the Asian techniques on a personal basis. Studies of altered states of consciousness have revealed the complexity of consciousness which for a long time was ignored by behaviorist psychology in spite of the influence of, for example, William James. He demonstrated an open-minded approach:

Our normal waking consciousness is but one special type of consciousness, whilst all about it, parted from it by the flimsiest of screens, there lie potential forms of consciousness entirely different. We may go through life without suspecting their existence, but apply the requisite stimulus, and at a touch they are there in all their completeness. No account of the universe in its totality can be final which leaves these other forms of consciousness quite disregarded. How to regard them is the question. At any rate they forbid a premature closing of our accounts with reality. (James, 1914, p. 298)

Recent advances in Western science seem to facilitate a more open-minded attitude to Asian psychology and medical practice and theory (Walsh & Vaughan, 1980).

#### 1.08.3.1.4 Arabic medicine

Arabic medicine has two main sources. One is Greek which the Arabs incorporated into their rational system of knowledge during their most enlightened period from the tenth to the twelfth century. The other source is the medical tradition of the bedouins which gradually merged with the teachings of the prophet Muhammed, and is crystallized under the term "prophetic medicine."

The two bodies of medical knowledge have been in conflict with each other primarily because of the prestige of the prophet and the theocratic influence of the clergy. The Greek medical tradition was regarded as alien by the

rank and file of the *ulama* the theocratic scholars who, until this day, dominate much of the intellectual life in Islamic countries.

Seen from the perspective of the tenth century AD it is inconceivable that the great advances in medicine should emerge in the Christian West. After the decline of the Roman Empire and with it the Greco-Roman civilization, elite Greek medicine was rescued by the Arabs. During the first three or four centuries after the Hijra, Arabic became the common language of the Islamic world and the language of science. Greek medicine was taken over wholesale by the Arabs who, to the degree that scientific medicine survived, stuck to it until they were subjected to the impact of European science in the nineteenth century. The translation of the Greek medical sources was performed by a comparatively small group of scholars, primarily in the ninth century. Hundreds of Greek works were translated into Arabic and became part of the scientific legacy during the golden age of Arab dominion. The Hippocratic and Galenic systems were saved for world civilization by these early scholars.

Arab practitioners added to the body of knowledge developed by the Greeks the subjects of pharmacology and surgery, which flourished in Andalusia; and ophthalmology, which flourished in Egypt and Mesopotamia where eye diseases for climatic and ecological reasons were the scourge of the people.

This body of knowledge was, as pointed out above, systematized in a treatise on medicine by Ibn Sina, Avicenna, and partly through his famous canon of medicine (*al-qanun fit-t-tibb*). Greek medicine was restored in the West. It was part of the curriculum in Salerno for centuries. The Greeks and the Arabs also made contributions to psychiatry, which of course was an unknown term before the nineteenth century.

The high standard of medical practice in the Arab domain was to a large degree promoted by the medical center in Gund-Shapur. This pre-Islamic center not only promoted an exchange between Indian and Greek medicine, but set a standard for medical craftsmanship which made the physician a respected personality in cosmopolitan Arab culture, although the school was in its heyday run by Nestorian scholars and physicians. The academy was probably founded in the first half of the sixth century after the Academy of Athens was closed in 529.

It is known that when the founder of the Abbasid Caliphate, al-Mansur, came down with a stomach disease and his regular court physicians were helpless, the chief physician at Gund-Shapur was called to Baghdad. When he successfully cured the ruler, the prestige of Gund-Shapur was established and with that its

high standards and medical ethics with its roots in Hippocratic traditions. It is probably also due to the influence of this academy that Arabic medicine by the Hindu and Urdu speakers is known as *Unani*. Unani is the English spelling of the Arabic term *Yunani* which means Greek, that is, Ionian.

Of particular interest is Greek and Arab psychiatry. The Greek physician Rufus of Ephesus wrote a treatise on melancholia which was translated and supplemented by Ishaq ibn Imran, a court physician in Kairouan, at the beginning of the tenth century. Melancholy was treated with medicine and psychotherapy which included music.

Considerable insight in mental illness is testified by the treatments recommended for fixed ideas. The main point is that the ideas were taken seriously and not dismissed, as they sometimes are by modern physicians. The treatment was based on the authority of the physician. It seems that Rufus of Ephesus had reflected on the power of authority of the formation of cognitive states which independently of the Greek tradition, was introduced in European clinics by Franz Anton Mesmer (1734–1815) in his so-called magnetic treatment based on the power of suggestion. Although Mesmer's method was dismissed as fraudulent, he was the first to introduce a systematic application of the authority of the practitioner as a cure of hypochondriacs and hysterics. A monument of his achievement has been raised in the English lexicon in the form of the term "mesmeric."

Profound psychosomatic understanding appears between the lines in Arab medical texts. This is particularly the case with regard to the demeanor and lifestyle demanded of the physician. He had to be a respectable person of high ethical standards and the bedside manners were an essential part of the cure.

Some of the great practitioners are surrounded by a heroic lore which has survived until the present day. Of particular interest is the story of the great Razi who healed the emir of Rhagae (in ancient Persia). This ruler had a paralyzed leg and Razi understood that the ailment was a hysterical condition. Razi ordered drinks and a hot bath "to ripen the humors." But suddenly he drew his knife as if he was prepared to kill the emir, who sprang to his feet, cured of his hysterical ailment. Razi, of course, did not use the term hysteria, but the story testifies to a remarkable psychological insight (Bürgel, 1976, p. 51).

The great Arab scholar Ibn Khaldun (1332–1406) wrote a famous treatise on the decline of Arab civilization which had flourished from the tenth to the twelfth century.

After that the development of knowledge, including medicine, did not just come to a standstill, but fell into a deplorable decline. The reason is complicated and the explanations that are offered are controversial. Some maintain that the reasons are political, as did Ibn Khaldun; others assume that the reasons are found in the economic developments. What we do know, however, is that one of the effects of the decline was a resurgence of personalistic explanations in the realm of medicine. Bürgel (1976) gives the following explanation of this turn of events:

If we want to know which spiritual forces were most potent in paralyzing the scientific impetus of the golden age, we have to ask what the essentials of science are. I think that one essential is imminent to science, its very soul and and life-blood, and this is rational argumentation propelled by an insatiable curiosity. The other thing essential to science belongs to the history of economy as well as to the history of science, and it is the interest in, the demand for, and the consumption of scientific products. What forces were harmful to these essentials? As for the economic element, a strong and ever-increasing influence was exercised by Islamic mysticism, which turned people to the inner instead of the outer world so that they restricted their natural demands to a minimum. As to curiosity, I have noted the prevailing concept of science as a constant stock of knowledge. Rational thought had several renowned enemies, some of whom could trace their origins to antiquity. I refer to astrology, alchemy, magic and, finally, of Islamic origin, the so called prophetic medicine. These four were looked upon as sciences by the great majority, and even by most of the scholars. Nevertheless, they were hothouses of irrationalism, the rational disguise making them even more harmful. (p. 54)

We recognize in the resurgence of personalistic theories of causation the question of destiny. In monotheistic Islam the basic question with regard to illness was its religious meaning. Pious people regarded illness as the will of God and wondered whether rational cures could be regarded as an obstruction of their ordained destiny. Illness and suffering were regarded as an atonement for sin and it was common opinion that people suffering from, for example, blindness or epilepsy were assured a position in Paradise because of their patient suffering.

Suffering in itself came to be regarded as meritorious sublimely expressed in a cult of martyrs. "He who dies on a sickbed, dies the death of a martyr and is secure against the inquisition of the tomb." This saying is attributed to the Prophet according to *Hadith* the body of lore which is used to give prophetic

legitimacy to opinions and procedures not explicitly sanctioned by the Koran. According to the tradition, the Prophet claimed that "Healing resides in three things: a draught of honey, a cut by the cuppingglass (scarification), and the branding by fire (cauterization). But as for branding I forbid it in my people" (Bürgel, 1976, p. 57).

The belief coded in this quotation refer to honey as a medicine. It is still regarded as such at the margins of Islamic culture, for example, among the Sidama of Ethiopia. The cupping is still practiced in the Mediterranean where it has developed a special form through the application of glass. A cup is heated and applied to the surface of the skin. When it cools, a vacuum-effect sucks the skin into the cup. It may be regarded as a milder form of therapeutic infliction of wounds. Cauterization is still practiced as a folk remedy in the periphery of the Islamic orbit. A common practice is to drill a piece of wood, as in the old technique of fire-making. When the stick almost catches fire through the energy of drilling, it is applied to the skin. A number of burns are believed to cure minor sickness. Through prophetic medicine folk remedies were given medical legitimacy, among which was the bedouin idea that all diseases were caused by disorders of the stomach.

Magic healing practices became widespread, and indeed are still widespread in the Islamic orbit. Of particular prominence is the magical protection of talismans. Most visitors to Muslim countries have noticed that children have small bundles around their necks with scriptural talismans. Words from the Koran or the Prophet written on small pieces of cloth or paper are regarded as a necessary protection. A widespread practice in former times was to write holy texts on a washable material, rinse it and give the rinsings to the patient as a medicine. In the popular lore, the Prophet is reported to have performed ritual washing at the bedside and afterwards to have administered the water to the sick as a drug. The lore and some of the Hadith should of course be taken with a grain of salt. Charlatans have always tried to exploit the reputation of prophets and sages to their economic benefit.

The history of the decline of scientific medicine in the Islamic orbit after the twelfth century is only one example of the vulnerability of rational knowledge. Although it cannot be proved, it is probable that empirical science is dependent upon a division of labor through which economic and political interests are divorced from the management and application of knowledge. Seen from the point of view of history, the worst possible situation in the management of knowledge is theocratic

monopolies. Theocracies have often been passionate opponents of rational knowledge and have not hesitated to oppress and even execute voices of reason and knowledge. Criticism of prophetic medicine therefore was practically nonexistent. One of the few critics, like Ibn Khaldun, pointed out that it was not part of the Prophet's mission to teach medicine. Another was the Andalusian poet Ibn al-Khatib who repudiated the explanation of plagues given by prophetic medicine pointing out that the validity of the traditions should not be questioned if they were incompatible with the senses (Ullmann, 1970).

The Islamic world today is struggling to reconcile Western science with their traditional *Weltanschauung* and, as Bürgel points out,

The Islamic world has for over a century entered upon a new period of acculturation. From its very beginning, Islam has revealed a peculiar skill in accommodating and amalgamating foreign cultural influences without losing its own identity. It is just now proving that it has lost nothing of this marvelous faculty. But if there is no chance for the Galenic system as a whole to be revived in our day, one may think of reviving particular achievements—for example, certain well-tried therapies and old drugs—which are in fact now being scientifically tried out and reproduced on a large scale by Unani pharmaceutical companies and state pharmacies in Pakistan, India and Sri Lanka. (Bürgel, 1976, pp. 60–61)

#### 1.08.3.1.5 Chinese medicine

Chinese medicine has evolved as a system of knowledge almost completely independent of the other great civilizations—the Greco-Roman, the Arab, and the Indian. Whereas these three systems have, as we have seen, strongly influenced each other, contact with China before the age of discovery was negligible. Also, in the development of Chinese medicine we can recognize the shift from personalistic to naturalistic explanation which is the *sine qua non* (absolute necessity) of rational knowledge. The traditional Chinese medicine is best known through the classic work *Huang Ti Nei Ching Su Wen* which is claimed to be from the third century BC. However, recent historical research (Veith, 1972) has demonstrated that it most likely was compiled during the Han dynasty (202 BC–221 AD). This proves that naturalistic explanations emerged in China at about the same time as in Greece and India. It is notable that this happened during a period when economic and political developments had reached a degree of maturity calculated on the basis of comparative studies as with the

establishment of money, markets, and orderly government.

The basic concepts of Chinese medicine are today common knowledge in the West. The two principles of life, “yin” and “yang,” are operating in the human body as everywhere else in the Chinese cosmos. Health is dependent upon the right balance between these two principles. Yang represents heaven, sun, fire, heat, dryness, light, the male principle, the exterior, the right side, life, high, noble, good, beautiful, virtue, order, joy, and wealth—all the positive elements of being. Yin represents the opposite: earth, moon, water, cold, dampness, darkness, the female principle, the interior, the left side, death, low, ignoble, bad, ugly, vice, confusion, and poverty (Foster & Anderson, 1978). Excessive yang causes fever, yin produces chills.

The hot–cold dichotomy in traditional Chinese medicine is reminiscent of the Ayurvedic tradition, but probably developed independently in China. It was probably developed as far back as 180 BC. But the major work on the hot–cold dichotomy is from the fourteenth century. In Chia Ming's work *Essential knowledge for eating and drinking* (*Yin-shih-hsü-chih*). (AD 1368), he describes

43 kinds of fire and water, 50 kinds of grains, 87 kinds of vegetables, 63 kinds of fruits and nuts, 33 “flavorings” and condiments, 68 varieties of fish, 34 kinds of fowl, and 42 kinds of meat! The entry for each of these 460 entries tells to which of the five flavor categories it belongs, its “character” (specified degrees of hotness or coolness) and the other foods that should be eaten with it. The character of natural rainwater is cold, while that of water from a stalactite cavern which is warm; the flavor of both is sweet. Glutinous rice is said to be warm, and eaten in excess causes fever. Soybeans and fragrant leeks are warm, and eaten in excess causes fever. (Foster & Anderson, 1978, p. 64).

Acupuncture is regarded as a “cold” operation especially suited to diseases caused by an excess of yang.

Diseases are classified according to causation into external and internal diseases. Diseases caused by external forces are yang diseases, yin diseases by internal forces. In Chinese thinking yin and yang have been understood as a single entity combining in any manifestation in the cosmos both positive and negative elements.

Chinese cosmology recognizes five elements: water, fire, metal, wood, and earth. This idea of basic elements which was also part of Greek cosmology developed independently in China. Although this idea from our position of knowledge seems primitive and erroneous, it

cannot be denied that it put the human mind on the right track towards modern physics and chemistry. In the great hide and seek of knowledge, the Chinese were at the portal of rational, empirical science, but stopped short of entering.

Manfred Porkert has pointed out that Chinese thinking remained in the inductive mode:

“The scientist who uses the inductive and synthetic mode of cognizance will observe first, and then speculate on his observation; the scientist who supplies the causal and analytic mode will first speculate and act, and after that he will observe. My hypothesis is that, at the dawn of systematic speculation, philosophers of all civilizations first adopted the inductive and synthetic mode of cognition because of its directness and simplicity. Causal analytic thought apparently did not enter the scene before the 4th century BC. In China it only constituted a brief interlude in the works of the mohists and of Hsün-tzu; in the West, the opus of Aristotle marked the beginning of a tradition that was to continue into our days. (Porkert, 1976, p. 63)

Although its concepts seem foreign to the Western mind, it cannot be regarded as inferior to the Hippocratic tradition, the concept and ideas of which were not entirely different from the Chinese with regard to the theory of basic elements. A respectable body of knowledge was accumulated in the Chinese tradition and the efficiency of some of the cures, therapies, and drugs are no longer questioned.

The Chinese physicians never reached the prestige accorded to modern Western physicians or their Arab colleagues in the golden age. According to Confucian philosophy, which is above all concerned with social ethics and the relation of the individual to society, the top of the social hierarchy was the literatus. The tiller of the soil was given the second rank. At the bottom were the soldiers and the merchants. Physicians were considered to be technicians (*shu-shi*) and were assigned the middle ranks of society together with engineers, craftsmen, and artists.

Chinese medicine confirms Benedict's ideas on patterns of culture (1934). Although her ideas are no longer part of mainstream anthropology, Chinese culture's meticulous concern for detail and the patient execution of craftsmanship whether in cooking, art, or medicine demonstrates a consistent pattern which never fails to impress the observer. Their belief in their own tradition of knowledge, also in medicine, is demonstrated by the persistence of the hot-cold dichotomy in the medical traditions of modern Hong Kong. Acupuncture is about to become

part of Western medicine although its mode of operation in the human body is still unknown.

#### 1.08.3.1.6 American medicine

The situation in America is unique and different from the other civilizations we have been discussing. The reason for this is of course that the civilizations of America more or less disintegrated under the impact of European conquest. The knowledge systems of the Aztec, the Inca, and the Maya have not been adequately brought forward to our times because the social organization which sustained them broke down and no adequate writing system was developed to codify the knowledge in texts. Although Arab medicine went into a period of decline, medical centers like Gund-Shapur served as a guarantor against total decay and the traditions had been safely recorded in a medical canon. The lack of a basic canon is the main reason why pre-Columbian medical knowledge has only survived at the so-called folk level.

Although the terms folk medicine and folk level may offend some egalitarian sensibilities, it is a meaningful term from the point of view of the organization of knowledge. The vast bodies of knowledge accumulated by the complex civilizations need specialized institutions to be stored, retrieved, and above all applied. A pedagogic system of sorts is necessary to transfer knowledge from one generation to the next. When this system breaks down, people everywhere have struggled to save some of the knowledge through their own management. Also, in the civilizations which maintained their political integrity, rational medical knowledge survived, although outside the legitimate orbit defined by clerics as in some of the Islamic countries, varieties of folk medicine flourished. When people have little access to expert knowledge, they resort to curers and try the best they can to retrieve the necessary knowledge from the lore of their families and the local community and in the case of European immigrants to the USA, from books.

The folk medicine of America has been primarily studied by folklorists, but in recent years anthropologists have joined forces with the folklorists in the development of ethnomedicine.

In the USA a plurality of folk medicines exists. Each immigrant group has to some extent developed its own folk medicine. But it is customary to recognize three distinct types: Euro-, Black, and Spanish-American.

Euro-American folk medicine is unique because most of the immigrants were able to read and thus were not merely dependent upon

oral traditions. But in the early years of frontier life, particularly in the nineteenth century, doctors were limited and not always elite experts. However, Indian experts were highly respected as "powwow" doctors. Pickard and Buley claim that "in some western communities in the early years there were native American doctors who were held in quite as high repute as regular white doctors" (Pickard & Buley, 1945, p. 36).

A part of American culture in general is the do-it-yourself tradition which has created a special literary genre. Richard Carter's *Valuable vegetable medical prescriptions for the cure of all nervous and putrid disorders* (1815) was widely distributed.

The native American knowledge came to its right in books like S. H. Selman's *The Indian guide to health* (1836), and William Daily's *The Indian doctor's practice of medicine* (1848). An impressive number of home remedy books in the medical mainstream tradition were also printed, the best known is possibly John Gunn's *Domestic medicine or Poor man's friend, in the house of affliction, pain and sickness*, first published in 1830. By 1885 it had gone through 213 editions.

The high level of literacy among many of the immigrant settlers gave the folk medicine a professional touch which is unique and may be regarded as an intermediate type of knowledge from the point of view of social organization. Through literacy, the line of communication from specialist centers to the people was shortened and knowledge was not subjected to the same kind of distortions we know from oral traditions.

The best known of the "authentic" folk medicine of the Euro-American type is that of the Pennsylvania Dutch (Amish). As Gebhard (1976) points out, ideas still alive among the Amish, for example, in the saying "Feed a cold and starve a fever," date back to Celsus (50 AD) and the use of cobwebs to cure bleeding goes back to Galen.

The black folk medicine represents a purely oral tradition. The best known variety is the so-called "voodoo" or "conjure" which developed in the early nineteenth century around New Orleans. Black folk medicine has three main sources: Catholicism, seventeenth and eighteenth century European occultism, and traditional African medicine and belief.

Snow classifies the black explanation of disease as "natural" and "unnatural": "The first includes deficiency in protecting the body against bad weather, and divine punishment for sin. The second has to do with the individual's position as a member of society" (Snow, 1973, p. 272). As "unnatural" Snow refers specifically

to a belief in witchcraft, a theory of illness which Foster and Anderson (1978) characterize as personalistic. "Nothing else in the fieldwork elicited so much emotional response as the question about witchcraft. Whether answers were negative or affirmative, the vehemence was often startling" (Foster & Anderson, 1978, p. 274).

The belief in witchcraft is probably of European, not African, origin. African witch belief has never reached the degree of hysteria as in Europe and later in the USA, and witch craze is an authentic Euro-American phenomenon. The beliefs, therefore, have probably been taken over from the European settlers. The idea of protecting the body against bad weather has a ring of humoral pathology which suggests Greek influences. What is of authentic African origin is the herbal lore and practical cures and remedies including a most capable midwifery. African elements are certainly also part of voodoo rituals.

Spanish-American folk medicine represents a more coherent system than the others and is based on an equilibrium model of health based on a theory of balance between hot and cold *calidades*. Illness is the result of an excess of heat or cold, and the origin of these notions is fairly obvious. Spanish-American folk medicine is based on the Greek pattern. Since Spain, particularly in Moorish times, was the center of medical knowledge in Europe, it is not surprising that the Greek-Arab articulation of humoral pathology dominated the medical scene in the homeland of the *conquistadores*. It was brought to the New World with the European settlers and remained the major medical paradigm well into the eighteenth century. From the elite centers of medicine in Spanish-speaking America it found its way to the mestizo peasants and acculturated native American groups. To a large degree it replaced pre-conquest medicine. However, it has been suggested that, for example, the hot-cold dichotomy in Mexican folk medicine has its roots in Aztec beliefs (Lopez-Austin, 1974).

### 1.08.3.2 Small-scaled Societies

#### 1.08.3.2.1 Africa

In Murdock's (1980) comparative study we noticed the great variety of explanation of disease and misfortune. A typical example of spirit aggression is recorded from the Sidama people of southwestern Ethiopia and may serve as an example both of theory and practice in this "medical genre." It will also give a portrait of a prominent character in the medical practice in small-scaled societies, the shaman (Brogger, 1986).

Disease among the Sidama is commonly explained as the result of spirit-aggression. The Sidamas are firmly convinced that they are surrounded by an army of spirits.

Some of the spirits live in large trees, others near the river. They are invisible, but are capable of invading the bodies of men and talking through the mouth of the person they choose as their host. The spirits are capable of striking men and animals with disease and misfortune, but can also be of help if they are treated properly.

The spirits rarely strike only once. They prefer to establish a lasting relationship with a person. When such a relationship is established, the spirit demands regular attention through spirit sessions, *hayatas*, in which communication with the spirits is based on a state of dissociation or trance in the host. This state is reached through drumming and singing during the spirit session.

When a relationship with a spirit has been established, it cannot be dissolved and lasts until the person dies. When a man dies, it is very often claimed that he was killed by his spirits. To get some systematic knowledge of the spirit cult, a sample of 29 cases was selected from one local curer. Although the spirits are regarded as heavy burdens, most people were afraid of losing them. A spirit usually heralds its choice of host with a sickness. Of the 29 cases recorded, 20 started with some sort of sickness. The individual histories of acquisition have the same general pattern, but with some variations of details. The following case is fairly typical, although richer in details than most. This man was 65 years old at the time of the fieldwork, and had entertained the relationship with the spirits for more than 30 years.

I hated the sheitanna in the beginning, I was insulting them. But one day when I came from the market I asked my wife to make coffee for me. As I drank this coffee, I suddenly began to shake, and I did not know what was happening to me, and I asked my wife. She told me that I became very strange. At that time I was the student of Abba (an Italian priest). I explained to him what had happened to me. Abba told me that spirits were dangerous, and he gave me a kind of paper with the image of Jesus Christ and told me to keep it under my pillow and to pray. This cured me for some time. But when the Italians left Ethiopia, Abba left with them, and the spirits caught me again. I got pain all over my body and I asked people what I should do, and they told me to make a hayata and pay gabo, gifts, to the spirits. I gave him a sheep and honey, but although the spirit made me shake, it did not talk. Therefore I brought a kalicha to the hayata, and he made the spirit talk. I had two spirits, Dillo and Warro.

These were the masters, but other spirits would come at the same time. These spirits can kill and make a person sick, but they can also be of help. They gave me children, and I built a galma for them. In the early days I had hayatas very frequently, sometimes every two months. That protected me and my family from sickness. My father also had spirits. But his spirits were different. My spirits came from Dulanna, all spirits come from big people like Guto and Dulanna. (Brogger, 1986, p. 66)

Usually the spirit will after some time demand its own house, galma. The galma is a smaller replica of the sidama house, but constructed for the sole purpose of serving the hayata sessions. It is built close to the main hut as a kind of chapel for the households of the compound. Guto and Dulanna are the names of particularly powerful shamans who often introduce new spirits to the sidama "pantheon."

The attacks of the *sheitanna* are in many cases interpreted by a *kalicha*. But in a number of cases the presence of the spirits is so obvious that no consultation is regarded as necessary. When contact has been established by a kalicha and his spirit or spirits, professional assistance will rarely be necessary. Most of the people who served as hosts for spirits understood when the spirits demanded a tribute. This did not happen only through sickness—a spirit could sometimes let its demands be known through spells of instant possession. In a number of cases the spirit would approach a future host through a dream.

A 55-year-old and very active hayata organizer with his own *galma* got his first *sheitani* in the following way: "It first started through a dream. I saw it like a picture of a human being and it told me to make hayata. But I had no disease and I refused. It then reappeared in a dream." (Brogger, 1986, p. 67)

But he still hesitated. The same dream was repeated several times, and gradually his mental state changed during the day. He could no longer concentrate on his work and he sometimes had hallucinations. Sometimes he was talking to himself, and one day he pointed at an imaginary sacrifice and said: "These intestines of the sheep we must bring to the kalicha." Finally, he could no longer resist, and he made his first hayata and became completely normal. From then on he gave hayatas regularly.

Not every Sidama had a spirit; an enlightened guess is that about 10% of the adult population has a long-standing personal relationship with one or more spirits. But those who have no spirit of their own may participate in the hayatas of close relatives and give gifts on these occasions. Thus, most of the community of grown-ups are



in one way or another affiliated with one or more spirit. When an ardent spirit-worshipper dies, the spirit is often inherited by a close relative. In fact 10 of the above 29 cases inherited their spirits from their mother, father, or spouse.

The local curer is technically in anthropological terminology, a shaman. Although he is formally concerned with healing, his main service is to articulate social conflicts in terms of the local system of interpretation of disease and misfortune. As the chief administrator of the spirit cult and its main source of authority, he is crucial to our understanding of shamanism.

The political leaders among the Sidama are the elders. The stateliness of the elders contrasts sharply with the demeanor of the shaman. If stateliness is the hallmark of the elders, eccentricity is that of the shaman. They surround themselves with a set of symbols which give them a certain flamboyance, particularly their jewelry and hairstyle. They will often let their hair grow long and appear in robes reminiscent of Arab traders. They adorn themselves with conspicuous rings of twisted copper, bracelets, and necklaces. They show little care for dignity but rather inspire an aura of mystery bordering on the sinister.

The elders are respected, shamans are feared. On the basis of their functions in the local communities the elders could rightly be called priests. They bear the responsibility for communal sacrifices, they may promote fertility through their blessings, and they mediate the tribal communication with the Sky-God, Magano. They are the guarantors of the respectable and the moral order.

The shamans, on the other hand, negotiate the relationship with a number of capricious spirits which haunt the local communities in Sidama. We have seen how the spirits impose themselves on ordinary people, and how communication with them was established through the spirit session, the hayata. Most of the worshippers of spirits merely attend to the problems of their immediate families.

There is no formal process of recruitment of shamans. A shaman establishes himself by his own choice as a public practitioner in the art of spirit-management. In order to project the image of a shaman he has to change his way of dressing and general appearance. He will either build a special hut or arrange his house in a way that facilitates the accommodation of clients. Among ordinary men the spirits are regarded as jealous and demanding, and unnecessary contact with them should be avoided. But when someone is struck by misfortune and sickness, the presence of the spirits is actively solicited so they can be persuaded to withdraw their influence.

The cures of the shamans are efficient within their context of understanding, as in cases where the illness or disturbance belongs to the moral order and has its roots in disturbed social relationships.

The healing traditions of shamans have attracted the attention of modern Western medicine and psychology recently and they cannot be dismissed as charlatans, as demonstrated by Andrew Strathern (1995).

### 1.08.3.2.2 *New Guinea*

Among the Baktaman of New Guinea according to Barth (1975) sickness is thought to be caused by both spirits and sorcerers, but also by breaches of taboos. But, maintains Barth,

Baktaman also operate with a concept that seems close to a Western pre-scientific notion of "disease"—a condition, often communicated or initiated by a particular incident, causing discomfort of recognized kinds and even death. Thus, for example, one man who developed a cold chastised another for having been spitting and blowing mucus all over the place when he had been sick; this had been eaten by the pigs, stored in their fat, and communicated to the patient when he ate the pork. Pressed to explain the nature of what was communicated an informant fell back on the idiom of spirit; but it makes sense for him none the less to declare that a particular condition of being sick is caused not by sorcerers or spirit agents, but "just sickness." (p. 137)

Sickness then can be brought about by the following causes: spirit attacks, male sorcery, female sorcery, breach of taboo, menstrual pollution, and plain sickness.

It is not unreasonable to suspect that the category "plain sickness" has been added to the Baktaman taxonomy recently under the influence of Western medicine, small elements of which were brought to the attention of the tribe by anthropologists.

The Baktaman have developed a "technique" of disease prevention with an interesting and familiar ring: the amulet. Baktaman children, like children in the Mediterranean, wear small bundles around their necks, usually a selection from dog femurs, water-lizard mandibles, cassowary claws, and ginger wood. The spirits from the animals are supposed to guard against spirit attacks. Grown-ups also wear talismans, but their amulets are secret and hidden in minute bags suspended from the neck to hang over the breastbone near the seat of one's spirit consciousness.

Various cures are available, but none of them seem to have a rational basis. They have

developed medicines, but their mode of action is magical. For example, spice bark is chewed and blown over sick persons with a combined whistling/blowing gesture normally used in conversations signifying “get lost.” Infections are transferred to the taro-plant by injecting a small specimen of pus from the sore under the skin of the taro. Bleeding of the infected area is also practiced. If these cures fail, the ultimate remedy for sickness is pig sacrifice with an appeal to the ancestors with a mountain spirit as an intermediary. The sacrifice is administered by a shaman. It is not reported that the shaman induces trance in the curing process. Trance, however, is an essential prerequisite of a shaman as among the Sidama.

#### 1.08.4 THE GROWTH OF THE WESTERN SYSTEM OF UNDERSTANDING

##### 1.08.4.1 The Role of the Psychiatric Hospital

Until the eighteenth century, personalistic explanation of disease, and particularly mental disease, was common in Europe. In the clinical literature it is referred to as animistic explanations (Brogger, 1986). In cognitive terms there is no major difference between the belief in spirit aggression in eighteenth century Europe and the personalistic explanations we have encountered in the ethnographic literature. Among the Sidama the spirits are managed by the shaman, in the European tradition by the priest. Although the shaman and the priest both have the responsibility of relieving the distress caused by assumed spirits, the priest should not be classified with the shaman. The priest never resorts to trance, but holds an office based on some sort of expertise and legitimacy which is dependent upon specialized, bureaucratic institutions. The shaman has to sustain his position through his own charisma and role play. He or she is dominating in small-scaled societies. Priesthood is an institution of complex societies. In the European tradition possessions were treated by exorcism, and the church has codified rituals of exorcism.

The belief that mental distress is caused by evil spirits is surviving, to a degree, even in a modern industrialized society like the USA (Goffman, 1961). But today legitimate treatment is based on the scientific *Weltanschauung* with the psychiatric hospital as one of the core institutions. The psychiatric hospital, however, does not have an honorable pedigree. It evolved from institutions that were created to house the poor, the uprooted, the unemployed, and the insane—in other words the outcasts emerging in the wake of the disruption of the agrarian society at the dawn of industrialism in

the seventeenth century. The outcasts in Paris, London, or Philadelphia in the eighteenth century were confined respectively to La Salpêtrière, Bedlam, and Pennsylvania Hospital.

The insane were given worse treatment than the other inmates. Foucault (1965) give the following description of their situation:

when the waters of the Seine rose, those cells situated at the level of the sewers became not only more unhealthy, but worse still, a refuge for a swarm of huge rats, which during the night attacked the unfortunates confined there and bit them wherever they could reach them; madwomen have been found with feet, hands, and face torn by bites which are often dangerous and from which several have died. (pp. 70–71)

But by the end of the eighteenth century more humane treatment of the insane was gradually introduced. It is significant that these changes followed in the wake of modernization in the full sense of the word both within the realm of economics and production which, it may be argued, paved the way for the age of reason in Europe and the USA.

The first hospital to remove the chains from the patients was St. Boniface in Florence, Italy. But the most famous event is Philippe Pinel's removal of the chains in his capacity of the newly appointed director of La Bicêtre in Paris during the French Revolution in 1792.

This was not done without opposition. A famous exchange between Pinel and one of the radical leaders, Couthon, is interesting because it forecasts an understanding of certain psychiatric symptoms which were not generally understood before the middle of the twentieth century. After having been insulted by one of the patients, Couthon turned to Pinel and said: “Now citizen, are you mad yourself to seek to unchain such beasts?” Pinel replied calmly: “I am convinced that these madmen are so intractable only because they have been deprived of air and liberty (Rosenhan & Seligman, 1995, p. 40). By these words Pinel was ahead of his time. The iatrogenic aspect of certain psychiatric symptoms was not generally understood before the American anthropologist Goffman published his work *Asylums* (1961).

##### 1.08.4.2 The Cross-cultural Issue and Human Nature

Two systems of understanding can be recognized at our present stage of knowledge with regard to the cross-cultural issue. First, the universalist approach has as an underlying paradigm that one system of understanding may cover the human family as a whole. The

second system, the particularistic, is based on the assumption that cultures must be understood on their own terms and cannot be unified in one master theory. The Indian *Weltanschauung* may, for example, appear as strange and incomprehensible to us, and therefore in terms of Western folk belief regarded as "wrong." If we try to translate this *Weltanschauung* into the terms of our own and, for example, insist that the cows are holy simply because they are economically important, we act as universalists. A particularist will respect the Indian view and try to understand and even acquire it as a system of knowledge. It is not a question of an absolute dichotomy, and a taxonomic scheme may be a little premature.

During a symposium at the University of California, San Diego, in 1980, a group of leading scholars in the field of cultural theory met to discuss the state of their art. One of their objectives was to establish contact with the psychological sciences, for in the words of Shweder and LeVine (1984):

The symbol-and-meaning approach to culture seems to be an unusually well kept secret in the psychological sciences, most notable among developmental researchers working on such culture-saturated phenomena as personhood and self, morality and convention, social cognition and interpersonal relationships, self regulation and emotional response. It was the best bet of the planning group that this was the right time to bring recent developments in culture theory "out of the closet."

It is relevant at this stage to point out that American anthropology made great contributions when a group of scholars met and established some sort of consensus and joined forces under a common paradigm of understanding. The Columbia group of the 1930s is a case in point.

The group met in San Diego in 1980 and was following in the wake of an established tradition of the organization and management of knowledge in the USA, and may serve as an example of a cultural pattern in itself. Although no clear consensus came out of their efforts, they set the agenda for cultural studies for the next decade or so and some of their key participants, Richard A. Shweder, Robert A. LeVine, Roy G. D'Andrade, and others, with Clifford Geertz as an inspiring pioneer of cultural studies, have taken over the torch from the Columbia group.

During the next decade the field of cultural psychology was developed. Stigler, Shweder, and Herdt (1990) summarize its scope with the following statement:

Cultural psychology is the study of the way cultural traditions and social practices regulate, express, transform, and permute the human psyche, resulting less in psychic unity for humankind than in ethnic divergences in mind self, and emotion. Cultural psychology is the study of the ways subject and object, self and other, psyche and culture, person and context, figure and ground, practitioner and practice live together, require each other, and dynamically, dialectically, and jointly make each other up. (p. 1)

A comprehensive curriculum has yet to be designed.

#### 1.08.4.3 The Interface Between Psychology and Anthropology

One of the great pioneers in psychology, the German Wilhelm Wundt, was one of the first to use ethnographic material to inform psychological problems. He wrote in the genre which today is referred to as culture and personality. But although his influence in the growth of modern psychology is enormous, his writings on cultures never caught the imagination of his colleagues.

It was above all Sigmund Freud who became the pioneer in the exploration of the interface between psychology and anthropology. In a number of works he tried to explain how psychoanalysis could be used to explain the origins and functioning of social institutions. Freud assumed that his model of the structure of the mind was valid cross-culturally. He assumed that the phenomena of repression, by which painful experiences are kept out of the consciousness by unconscious forces, was universal. He regarded rituals and taboos he studied in the ethnographic literature as caused by processes similar to those he studied in his consulting-room in Vienna, that is, to ward off anxiety. His views have been summarized by Bock (1988, p. 32) in the following illuminating phrase: "Culture is to society as neuroses is to the individual."

Psychoanalytic anthropology initiated by Sigmund Freud caught the imagination of several generations of psychologists and anthropologists, particularly in the USA where it has served as a major source of inspiration until the present.

The pioneering work in psychoanalytic anthropology is *Totem and taboo* (Freud, 1950). In this ground-breaking work, Freud approaches these two classical anthropological problems. The term taboo is today part of the language of everyday life with only a slightly extended semantic sphere of reference compared to its strict anthropological meaning. In anthropology today it covers all types of

prohibitions from the incest taboo to Jewish and Indian dietary rules. In Freud's thesis, the Polynesian taboo served as a point of departure, and the term is linguistically Polynesian.

Freud approached his patients in Vienna not only as a doctor within the Western clinical tradition, but as a natural historian. Many of his neurotic patients had restricted their lives with self-imposed prohibitions almost indistinguishable from the authentic taboos from the South Seas. In his explanation, he offered a new and comprehensive understanding of social custom in psychological terms. His basic assumption is that just as neurotic behavior is the result of a compromise between unconscious erotic and hostile impulses, taboos are expression of ambivalent emotions.

The taboo surrounding Polynesian rulers are cumbersome and deprive the ruler of normal and gratifying social intercourse. In the realm of the conscious sphere of discourse, the taboo is an expression of the highest esteem. But in the hidden agenda of the unconscious mind it is a punishment for their exaltation, a revenge taken on them by their subjects.

The taboo on physical contact with the dead is common in preliterate societies. Freud maintains that "the taboo has grown up on the basis of an ambivalent emotional attitude...the contrast between conscious pain and unconscious satisfaction over the death that has occurred" (Freud, 1950, p. 61).

Although Freud's interpretation is controversial and not generally accepted today, it nevertheless is an expression of the psychoanalytic insight in human cognition which today is part of our cultural system of understanding generally. The key concept is "rationalization," a mental process through which deeper and hidden motives are presented to the world as well as to the conscious ego in an acceptable and morally legitimate fashion.

Freud articulated in his theory of the human soul one of the most important insights from nineteenth century humanism: the human capacity to sustain illusions. Intellectuals and philosophers have known for millennia that fraud is one of the great problems of human existence and have developed logic to aid their fellow men in the search for truth. Freud, however, went beyond the syllogisms and realm of pure cognition into the hidden hinterland at the border of consciousness and beyond.

This thinking was in the spirit of the nineteenth century which in philosophical terms was explored by Nietzsche, particularly in his work *Jenseits von Gut und Bose* (*Beyond good and evil* [1935]). The decline in the belief in the power of reason is heralded by Nietzsche in the following words: "The German soul has corridors and

hidden, connected channels, and it has caves, and secret hiding-places and dungeons." This first rumbling of early post-modernism inspired Freud to an attack on the hidden abysses in the human mind by reason, which is the main project of psychoanalysis.

Before Sigmund Freud, Karl Marx had presented his own views on the human capacity for illusions in his thesis on "false consciousness." He applied the term to the realm of political consciousness and maintained that the workers were living in a situation of false consciousness as long as they did not realize that they were oppressed. The false consciousness—with religion, the opium of the people, as the main culprit—was, however, the result of capitalism and therefore not an unavoidable situation.

Freud was of the opposite view and was convinced of the fact that rationalization was part of human cognition everywhere. Ibsen, who also elaborated on the great insight of the nineteenth century, gave the idea a literary articulation in *The wild duck* where he made it clear that human beings could not live happily without the lies of life, the cherished illusion people sustain in everyday life.

Freud's treatment of totemism also set the stage for an investigation of the relationship between parents and children in the human family and it was part of his theory of the developmental stages of early childhood. The mythological core in the exploration of totemism is that of King Oedipus who unknowingly killed his father and later married his mother. Upon discovering this, he blinded himself and went into exile.

Freud had discovered during the analysis of his patients the problems caused by the fact that children's erotic and hostile impulses are frequently directed toward family members. He was particularly interested in the relationship between mother and son and the sometimes explicit wish on the part of a small boy to eliminate the father to have the mother's undivided attention. But since small boys also love their fathers, the classic ambivalent situation emerges with great psychological costs, the psychological distress caused by the oedipus complex.

In presenting his insights on these sensitive issues Freud created his own myth inspired by the prevailing Darwinian idea of the primal horde, the assumed prefamily groups before the dawn of human civilization, dominated by a violent and jealous father who keeps all the females for himself:

One day the brothers who had been driven out came together, killed and devoured their father

and so made an end of the patriarchal horde...The violent primal father had been the feared and envied model of each one of the company of brothers: and in the act of devouring him they accomplished their identification with him...The totem meal would thus be a repetition and a commemoration of this criminal deed, which was the beginning of social organization, of moral restrictions and of religion...Society was now based on complicity in the common crime; religion was based on the sense of guilt and remorse attaching to it, while morality was based partly on the exigencies of this society (fraternal society) and partly on the penance demanded by the sense of guilt (exogamy and attempts at atonement) (Freud, 1950, p. 141–146)

Although a pioneer in the application of ethnographic knowledge in the understanding of the human soul, Sigmund Freud like James Frazer relied solely on written sources and never did any fieldwork himself. Those were the days before professional anthropological fieldwork brought the social life of distant cultures within the reach of the intellectual community.

#### 1.08.4.4 The Impact of Anthropological Fieldwork

The anthropologist who set the standard for anthropological fieldwork was Bronislaw Malinowski. His works from the South Seas could partly confirm and partly correct some of Freud's assumptions. With regard to repression of sexuality Malinowski could confirm Freud's opinion regarding its psychological costs (Malinowski, 1927, 1932). With regard to the universality of the oedipus complex, however, Malinowski pointed out that among the matrilineal Trobrianders in the South Seas, it was the mother's brother rather than the father who was the source of discipline in the life of a small boy, and therefore the primary object of childish aggression.

Comparative research through modern anthropological fieldwork not only demanded a revision of Freud's theories. Also, ideas of so-called primitive mentality, promoted above all by Levy-Bruhl (1923), which maintained that individuals in preliterate societies think in a manner not only quantitatively, but also qualitatively different from individuals from complex societies, had to be revised. Cognitive anthropologists who have studied the system of knowledge in preliterate societies with the regard to the mastery of their environment, have found rational systems which demonstrate that no generalized primitivity of thought can be observed (Price-Williams, 1980).

Of particular interest in this connection are investigations of how people in complex

societies make judgements under uncertainty. As Kahneman, Slovic, and Tversky (1982) have demonstrated, people under certain kinds of stress regress into "primitive mentality" which then should be regarded as a mode of thought depending upon circumstances and not on culture.

Cognitive studies of non-Euro-American cultures, however, may inform our understanding of modes of thought in general as demonstrated by Bateson (1958) in his study of dichotomous thinking and the reversal of opposites in the Naven ritual of New Guinea. Gardner, in his book on structuralism (1981), suggests that cognitive anthropologists like Levi-Strauss may broaden and deepen cognitive psychology in the same way as have linguistics and developmental psychology.

#### 1.08.4.5 Culture and Personality

The influence of anthropological fieldwork was greatly enhanced by the works of Mead who started publishing her studies from the South Seas in 1928. Her works influenced not only her own field of anthropology, but also psychology and above all public opinion. Her books were widely read (Mead, 1935, 1949), and she became a celebrity and a most respected anthropologist, although some of her conclusions were met with some reserve. After her death a virulent criticism of her work and methods has been raised by the Australian anthropologist Freeman (1983) who claimed that she had been misled by her informants and her claim that Samoan adolescence was relatively free of conflict was wrong. After the professional discussion on her work had run its course, the general agreement is that Freeman overstated his case, although some of his criticism had to be accepted.

When Freeman published his work, however, the culture and personality school of thought was already history. But Mead had left a lasting impact both on American anthropology and public opinion.

Her work grew out of the Columbia group under the leadership of Franz Boas. One of Boas' main concerns was the rampant racism both in Europe and in the USA in the first decades of the twentieth century. His main assumption was that it is the culture not the genes which determine human behavior and institutions.

The school of thought which emerged from the group at Columbia University was focused on the configurations of themes found in different cultures and their mode of thought was influenced by Gestalt psychology. The

Gestalt psychologists had pointed out that our perception is self-organizing and that patterns emerge on the basis of certain principles like similarity and proximity. The insight of the Gestalt psychologists that wholes have properties not found in the constituent parts of a pattern were applied to culture and reversed the trend towards atomism in American anthropology.

Distribution of traits, elements of customs, material culture, or kinship were, at the beginning of the twentieth century, used to inform rather speculative evolutionary theories and theories of cultural diffusion. The trait investigations almost disrupted culture systems, turning them into a hodgepodge of traits.

Benedict's *Patterns of culture* (1934) is the most elegant articulation of the configuration approach of the Columbia group which crystallized into the culture and personality episode in American anthropology. This school of thought was strongly influenced by psychoanalysis which is obvious in the following words of Benedict: "Tradition is as neurotic as any patient, its overgrown fear of deviation from its fortuitous standards conforms to all the usual definitions of the psychopathic" (p. 252).

The Columbia group was also inspired by ideas of tolerance, the roots of which can be traced to John Stuart Mill's ideas of freedom. Through her books from her various field-works, Mead (1928, 1935, 1949) not only convinced the anthropological community, but people in general, that human beings in different cultures are different, not because of any difference in their genetic make-up, but because the human personality is molded by culture into different configurations of basic human themes. In *Sex and temperament in three primitive societies* (1935), Mead demonstrated that ideas of masculinity and femininity are determined by culture and not by nature, and thus made a considerable effort to rid modern society of the yoke of traditional ideas of social roles. Her influence was not merely scientific, but also ideological. She published her major works in a period when parts of Europe were in the grips of a bigot and sinister racism with roots partly in misinformed and partly misread anthropology and biology.

Through Mead, the didactic role of anthropology came into focus. But this role is not entirely unproblematic. When the center of the anthropological stage is given to great humanists like Mead, the cause of reason has little to fear. But with the potential ideological impact of anthropology in mind, it is essential that the highest standard of method is observed when it comes to questions of values. The culture and personality school suffered a set back in the

wake of its strategic use first in World War II and later in the Cold War in the study of national character. The portraits painted by the scholars of the national character of an assortment of other nations were clearly influenced by political concerns and the geopolitical mission of the USA. But even if the problems of culture and personality are not in the focus at the present time, there is an enduring interest in the relationship between the individual person and his culture both in psychology and anthropology.

#### 1.08.4.6 The Paradigm of Ethnic Identity

The problems formerly treated under the headline of culture and personality are today being addressed under the headline of ethnicity. An ethnic group is normally considered to be a subgroup within a larger community or nation. It is of particular relevance in the USA where many different cultures meet within the framework of a judicially defined citizenship. The idea of the big melting pot gave way, particularly during the 1960s, to a resurgence of interest in ethnic identity. Formerly subdued and sometimes harassed groups, who used to be ashamed of their culture, changed their strategy and demanded respect for their traditions and style of life. This was a worldwide phenomenon indicating a shift of paradigm in cultural understanding. The Euro-American culture, which held a hegemonic place in the cosmopolitan scale of values as described by the Italian philosopher Gramsci was challenged by dominated ethnic groups of people all over the world: The native American, the Scandinavian Lapps or Saami, the Maoris of New Zealand joined forces in an ethnic uprising demanding respect for their unique cultures.

This great movement of self-respect focuses on identity and is related to nationalism as it developed in Europe in the nineteenth century with the emergence of new national languages and new nations. A hegemonic culture like the German was challenged by a number of ethnic groups within the framework of the Austrian-Hungarian Empire: the Czech, the Slovaks, the Hungarians, and a mosaic of people in the Balkans struggled to construct their own national identity based primarily on shared beliefs, shared language, and mythological origin. As Anderson (1983) has demonstrated, the feeling of identity and togetherness on the basis of national ideas is based on an imagined community dependent upon modern media of communication.

Many of the ethnic groups in the USA derive from the various European nations who

brought their national identities with them across the ocean. For good reason, Barth's work on ethnicity (1969) became the most quoted anthropological text in the 1970s and 1980s.

The psychological effect of identity problems has been explored by Erik Homburger Erikson and very sensitively described in *Childhood and society* (1950). Although Erikson does not present his description and analysis of the Sioux Indians of the Great Plains and other groups under the label of ethnicity, he demonstrates how the Sioux identity, as it had developed through history, was based on their tradition of being mounted warriors and bison hunters. He shows in detail how their identity in Sioux pedagogics is developed in intimate articulation with the stages of development, which he regards as pan-human, to establish an ethnic self-confidence elegantly adapted to their ecological situation. But through military defeat, economic dependency, and other features of reservation life, the self-confidence of the Sioux has been broken. Some of their psychological reactions are reminiscent of the old-prisoner-syndrome of concentration camps with the characteristic Freudian identification with the aggressor.

The field of ethnicity is a particularly promising common ground for a collaboration between anthropologists and clinical psychologists. The relevance of ethnic dimensions in psychotherapy has been explored by Abel and Metraux (1974). The blacks from Brazil, Africa, or the Caribbean represent different cultural configurations and should not be confused with the blacks in the USA.

The ethnic differences are not in principle different from the socioeconomic differences which have come to the attention of scholars of the patients of Freud and Adler. As Ansbacher (1959) points out, three-fourths of Freud's patients were rich, while most of Adler's patients were poor. For good reason Adler coined the term "inferiority complex" and regarded the will to power as the dominant theme of life, whereas Freud maintained that the basic theme was "eros." The clinical picture of their clients was different and can only be understood if the relationship between culture and personality is taken into account.

When cultural differences are of a greater order than among not too distantly related ethnic groups, investigators have recorded *culture bound disorders*. Whether these are specific to ethnocultural settings or variants of disorders found under different schemes of classification in the West is not clear. Kiev (1972) has tried to show the parallels in the Western diagnostic system and classify the

Central and South American *susto*, soul loss, and the Chinese *koro*, the fear of the penis or the labia withdrawing into the body, as anxiety states. The Southeast Asian *amok*, the symptoms of which are eruptive violence followed by amnesia and exhaustion is classified as a dissociative state. He classifies these culture bound ailments as a subgroup of Western neurotic disorders. Yap (1969) on the other hand classifies culture bound disorders as reactive psychoses.

The ethnocentric bias of Western psychiatry is a challenge to anthropology as demonstrated by Marsella and White (1982) which hopefully can be remedied through an open-minded exchange of ideas and rigorous cross-cultural research.

#### 1.08.4.7 The Cross-cultural Correlational Approach

A promising venture into cross-cultural research is associated with another group of scholars at Yale University: John Dollard, George Peter Murdock, and John Whiting. Jointly they developed the cross-cultural correlational approach at Yale Institute of Human Relations. We have already taken advantage of Murdock's encyclopedic work on theories of illness. One of the most prominent studies in the cross-cultural correlation approach is that of Whiting and Child (1953). They criticized the studies of national character and the configurations school because of its lack of scientific method and embarked upon an ambitious program of correlation studies. In his summary of Whiting and Child, Bock (1988) selects the following statement of their method:

In the correlation testing method the supposed antecedent condition is looked for as it occurs or fails to occur in the natural course of events in a number of cases. Instances are collected of its presence and its absence, or of its presence in various degrees. The supposed consequent condition is also looked for in each of these cases and determined to be present or absent in a given degree. It is then possible to determine whether there is a consistent relation between the two, and thus whether the hypothesis is confirmed or negated. Statistical techniques may be applied and the evidence of connection may be based on objective procedures which are repeatable. (p. 108)

The hypotheses to be tested are derived from psychoanalytic theory. Two kinds of customs were to be tested: childtraining practices and beliefs concerning the causes of illness. Whiting and Child identified five so-called systems of behavior based on the Freudian theories of

child development: oral, anal, sexual, dependence, and aggression. The children are assumed to develop socialization anxiety in each of these systems depending on the degree of frustration or indulgence they experience. High oral indulgence is noted when a child is nursed whenever it pleases. Severe weaning customs will cause "oral socialization anxiety," and the hypothesis assumes that high oral anxiety will produce theories of illness based on oral behavior: you are sick because of something you said or ate. This hypothesis is mentioned because it produced the most convincing correlation in the whole study.

Whiting and Child (1953) also explored the problem of male initiations which in many societies are extravagantly painful with scarification, circumcision, whipping, and hazing. They assumed that the cruelty of the initiators (i.e., the fathers), had something to do with the sleeping arrangements in the homes and the postpartum sex taboos. They made the assumption that in societies where mother and infant share the same quarters for an extended period of time and at the same time the postpartum taboo prevented the father from having sex with the mother for more than one year, cruel initiation ceremonies were to be expected. Correlation analysis confirmed the hypothesis.

We recognize the Freudian touch of the hypothesis in the element of rationalization in the staging of the ritual where the fathers under false, but unconscious excuses, seize the occasion to give their aggression and even sadism a free rein in the good name of pedagogics.

This Freudian insight is one of the best allies in the protracted struggle to reduce the suffering and abuse in human life because it rips the mask off moralists who exploit occasions to indulge their cruelty in the name of saintliness. The problem with psychoanalytic insight, however, is that it is hard to prove by acceptable and rigorous scientific methods. It is to the great merit of Whiting and Child that they brought their Freudian hypothesis to an irrefutable test.

Although Whiting and Child have been criticized for, among other things, overlooking the dramatization of male solidarity in puberty rituals, the correlation method still holds promise although it no longer occupies center stage as in the 1950s. One of the reasons is the great cost of statistical investigation of such a scale. To this can be added the new look on culture which tends to regard the approach of Whiting and Child, focusing on traits, as old-fashioned.

Culture is a symbolic system which is similar to a language and calls for its own methodology as pointed out by LeVine. In his words of a speech community:

Their capacity for mutual understanding is accompanied by a remarkable consensus about rules of pronunciation and grammar such that linguists can discover the rules prevailing in the whole community not from an extensive survey, but from a small number of informants. This redundancy of rules in a speech community is theoretically significant in illustrating how language functions as social communication, it is methodologically significant in permitting a mode of systematic inquiry distinct from the sample survey. (LeVine, 1984, p. 68)

## 1.08.5 SOCIAL SYSTEMS AND THE INDIVIDUAL

### 1.08.5.1 The Dominant Kinship Dyad

The individual tends to be somewhat distant in the analysis of social systems. A set of hypotheses with regard to the dominant kinship dyad was presented by Hsu (1965). In Hsu's work the individual does not take center stage, but his approach is in tune with the analysis of the social process which was to change the course of anthropology in the late 1950s and 1960s. According to Hsu's hypothesis, human societies everywhere give priority to one kinship dyad from the core dyad of procreation and the intrinsic qualities of this relationship are assumed to influence several cultural parameters. From the core dyad of mother and father other essential dyads can be traced: father-son, mother-daughter, brother-brother, and so on.

Each dyad had intrinsic qualities. The mother-father or husband-wife dyad have the following intrinsic qualities: discontinuity, equality, sexuality. The father-son dyad has the quality of continuity because a son becomes a father himself and through the links between father and son a lineage can be traced. Authority is intrinsic because of the differences in age and command of resources. Even in egalitarian societies, where the fathers try to be buddies with their sons, authority cannot be denied, only under-communicated. In other words, the oedipal situation cannot be avoided. There is hardly an element of sexuality in the relationship. The husband-wife dyad on the other hand is discontinuous, one marriage does not beget another, there are no lineages of marriages. The relationship is contractual even if it lasts a lifetime, but it always comes to an end. Since an intimate relationship cannot be sustained if the authority dimension dominates, the relationship is intrinsically egalitarian. Its very *raison d'être* is sexual.

According to Hsu's hypothesis, cultures always give priority to one of the possible dyads, and the intrinsic qualities of the dyad



which becomes dominant will be reflected in the cultural system. Societies where the father-son dyad is dominant will respect traditions and authority and sexual matters will not be culturally dominant. If on the other hand the husband-wife dyad dominates, respect for traditions will be less and authority will be under-communicated if not resented. Sexual matters will be culturally dominant.

The effects of the father-son dyad have been more or less thoroughly explored by Hsu (1971) and the evidence is in favor of the hypothesis. In Arab and traditional Chinese culture, the father-son dyad is unquestionably dominant and the respect for tradition and authority is obvious. Likewise sexuality is not a prominent theme in the public sphere of literature, media, and discourse. In the USA on the other hand, the husband-wife dyad is clearly dominant. Although the respect for the constitution is unquestionable, the image of North Americans as progressives and egalitarian is not unfounded. The question of sexuality is a dominant theme both in the private and the public sphere.

Hsu's hypothesis is not uncontroversial. This, however, is the fate of culture theories generally. So far other dominant dyads, like, for example, mother-daughter, have not been explored.

#### 1.08.5.2 The Iatrogenic Approach

In the Durkheimian tradition of anthropology and sociology, the systems have dominated at the cost of the individual in the English speaking world in the first part of the twentieth century. The person has more or less been regarded as a puppet in the strings of the social system. In the 1950s, however, the works of Max Weber reached the centers of anthropological discourse and the person as an actor entered the stage.

At the London School of Economics, Leach radically changed the systemic approach which until then had dominated British social anthropology. In his analysis of political systems (Leach, 1954), he demonstrated how human beings as decision-makers interacted with the social system. That changed the image of small-scaled societies as timeless depositories of unchangeable traditions. This image had been created from lack of information. Professional field descriptions before 1950 were more or less snapshots freezing moments in time.

At the University of Chicago, Erving Goffman demonstrated how the anthropological method of participant observation could be applied to cultures beyond the small-scaled societies. In *The presentation of self in everyday*

*life* (1959), Goffman analysed the strategies of personal behavior with the theater as a metaphor. He demonstrated how human beings played their roles, not in the make-believe way of the stage, but with the same objective: to impress the audience. His thick description of "impression management" codified in language processes which formerly were not part of conscious discourse. He described how social reality is created through role-play, how a select part of a person's role repertory is sustained in a situation through strategic interaction. Life after all is composed of situations, most of them trivial. Goffman made the situation the main object of his study and demonstrated how social reality is created on the spot and thus may be regarded as a kind of illusion. After Goffman it is no longer possible to regard culture as a thing, it is a process sustained by strategic interaction. The insights of Goffman connect with psychoanalysis on the inner stage of consciousness. Human beings also venture to impress themselves, the ego is an actor who creates the person from the raw material of basic drives.

In his study of psychiatric hospitals we recall the observation of Pinel during the French Revolution when he pointed out that the behavior of "lunatics" had something to do with the way they were treated (Foucault, 1965). Goffman demonstrated that many of the symptoms recorded by psychiatrists were samples of strategic interaction. One of the strategies is situational withdrawal. A perfectly comprehensive reaction to an impossible situation is codified by the psychiatrists as regression. Goffman opened up the Pandora's box of iatrogenic symptoms, symptoms created by the medical profession. His insight is valid beyond the psychiatric hospital, it applies to total institutions everywhere and he raised a problem which remains to be explored in depth: how psychological disorders may be imposed on the individual by society and by the medical and psychological professions. If these professions decide that a condition is pathological, it becomes a social reality, and a psychiatric disorder is magically established. This is sometimes an overlooked part of the construction of social reality. Anthropology may through the insights of how social systems and institutions mold the individual, and the person as a category, and vice versa, cut some of the strings which bind the individual to social systems.

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# 1.09

## Life-span Developmental Theories

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### 1.09.1 INTRODUCTION

In her book *Culture and commitment*, Margaret Mead (1970) proposed that modern life has profoundly changed our experience of the life course. In relatively simple societies, Mead suggests, the life structure for individuals was clear and predictable. Children could look at the lives of their grandparents, and take those lives as models for their own future. In such a society, "change is so slow and imperceptible that grandparents, holding newborn grandchildren in their arms, cannot conceive of any other future for the children than their own past lives. The past of the adults is the future of each new generation: their lives provide the ground plan" (pp. 13–14), a plan often believed to be supported by divine and mythic processes (Labouvie-Vief, 1994).

In contrast, suggests Mead, contemporary society involves profound differences in how the life course is experienced. An example of such a society is the USA during and after the beginning of the Industrial Revolution, when streams of immigrants sought new opportunities on this continent. Parents no longer were the experts in how to live, and children were as likely to learn from their peers as their parents. Thus, the past no longer was a guide to the future, but rather the future became shaped by the present. Learning and developing are no longer so one-sided, but elders are likely to learn from the young, just as the young learn from the old.

Along with Mead, many authors (e.g., Hareven, 1978) have suggested that social changes such as the Industrial Revolution and the information revolution have altered the way we experience life. No longer do we live in communities or countries that are relatively isolated, and homogeneous, but we are becoming citizens of a global community constituted by individuals from diverse cultural, ethnic, and racial backgrounds. Further, technological changes have produced increases in the life-span as a result of the elimination of early mortality. This "greying" of the population has dramatically increased the number of vital individuals of advanced age.

All of these unprecedented historical changes have changed the way we look at development through life. This is true in several basic ways. First, in the past, emphasis was on socializing

individuals into stable adult roles defined by cultures that were relatively homogeneous. Thus many theories described adulthood in terms of stability, while childhood was described in terms of change and transformation (e.g., Baltes, 1987; Baltes, Lindenberger, & Staudinger, 1998; Labouvie-Vief & Chandler, 1978). However, contemporary life highlights change and transformation along the adult portion of the life-span as well. As a result, there has been a wide-ranging re-evaluation of what constitutes "maturity," as well as the pathways by which maturity is reached. In fact, this new emphasis on continued growth in adulthood could be referred to as the "birth of adulthood," borrowing from Philippe Aries' (1962) famous statement that earlier historical periods brought a similar "birth of childhood" as a distinct developmental period.

A second consequence of historical changes has been that many cultures no longer are based on groups of individuals homogeneous in racial and ethnic features, but rather represent a "melting pot" of groups characterized by diversity (see Cavanaugh, 1997). This focus on diversity also has affected the way we view adulthood. While in the past, adulthood often was defined by a single outcome (such as a strong ego or abstract reasoning), now we are realizing that outcomes can vary widely depending upon the context in which individuals grow up. Thus, not only has development been extended into adulthood, but the pathways of development also have taken on more diversity. This "multilinearity" (Baltes, 1987; Fisher & Biddell, 1992; Labouvie-Vief & Chandler, 1978) is another feature important in recent writings about the life-span.

Finally, all of these historical changes also have had profound implications for the ways in which we look at the process of aging. Since aging often is defined relative to some theoretical apex of optimal functioning, redefining that apex has direct implications for views of aging. Often defined as a regressive deviation from some youthful standard of optimal functioning (e.g., Labouvie-Vief, 1985), aging is now understood as a period that has its own developmental challenges and opportunities, and that needs to be understood within the context of the total life course (see Erikson, 1982; Labouvie-Vief, 1980; Staudinger, Marsiske, & Baltes, 1995).

In this chapter, we highlight these recent re-evaluations in theoretical outlook. First, we will briefly highlight some of the general theoretical changes that have shaped current views of development in general, and of life-span development and aging in particular. Second, we will turn to a summary of how these changes are expressed in the evolution about thinking about intellectual functioning, while in the following section we will discuss similar changes in the domain of self and personality. We will discuss several theoretical movements that represent blends of these areas that have traditionally been isolated.

### 1.09.2 ISSUES IN LIFE-SPAN DEVELOPMENT

Although the main concern in this chapter is with psychological theories of life-span changes, it is useful to place these theories within a broader context of intellectual-historical changes. Concepts of human nature and the meaning of mature adaptive competence are embedded into broad historical patterns that cut across many disciplines, from philosophy through literature to art and religion. All of these domains have seen pervasive changes in how the ideal human is defined, for example, what blend of reason or faith, emotion and intuition, of independence or interdependence, of “masculinity” or “femininity,” and of similar polarities should characterize that ideal individual that crowns our theories of development, and what standards, as a result, should guide how to measure the resulting characteristics. In this section, we will outline some of these changes. In doing so, we will highlight their pervasive and general nature and merely anticipate the themes they set for future psychological questions.

#### 1.09.2.1 The Postmodern Turn

If one were to name a single characterization that Western intellectual tradition has held up as applying to the kind of ideal individual we envision when we think of advanced levels of development, probably it would be that of rational thought and conduct. Yet, while the basic definition of the nature of rationality has remained more or less constant for many centuries, the last hundred years or so have witnessed a wide-ranging transformation of the concepts of “reason” and “rationality.” Many disciplines (from philosophy, mathematics, and physics to art, literature, and religion) have participated in criticizing and deconstructing the traditional concept of reason. Following Plato (see Labouvie-Vief, 1994), the essential

features of reason were understood as its independence of the senses, the emotions, and other organismic processes. Reason was thought to transcend variations in context and to refer to abstract and conscious decision-making capacities located inside the individual mind or brain. It described our mental natures rather than our participation in a bodily, material universe, and it referred to an active “grasping” rather than a receptivity. In short, what was emphasized was reason’s objective nature as opposed to its subjectivity.

The rationalist model has stood as an ideal of maturity throughout the ages, and has served as the basis for the most important early theories of development, including those of Freud and Piaget (Labouvie-Vief, 1994). However, as the twentieth century began to witness broad global transformations in the nature of health, information, and community—the kind of changes, indeed, that Mead refers to—dissatisfaction with the old models of mind and development has arisen in many disciplines. As this faith in the power of logos is being criticized, many contemporary views of the mind have begun to return to the dimensions of mind and self that were abandoned by the classical view. Emotion and intuition, time and process, variability and the role of context, all of these elements that previously were devalued have come to the forefront in discussions. This contemporary confrontation with the other side of rationality is often referred to as “postmodern” attitude (Gergen, 1991; Lyotard, 1987). The postmodern attitude asks that we transform the dogma of the inseparability of reason from other, traditionally less valued functions of thought. Thus, a new view is emerging that holds that reason has inherent elements that are located in the domain traditionally relegated to the subjective: of the discourses unique to a culture, of conventions that have ritualized what we imagine of the rhetorical and practical, of the mythic and symbolic, and of the organic and emotional (see Labouvie-Vief, 1994).

#### 1.09.2.2 Development in the Postmodern Era

As far as psychological conceptions of individuals and their development are concerned, the postmodern revolution has had several effects on conceptions of the life course.

##### 1.09.2.2.1 The “end point” of development

A first profound change has been in the nature of what we call mature, ideal functioning. In traditional theories, such as those of Freud (e.g., 1925/1963) and Piaget (e.g., Inhelder & Piaget, 1975), the end point of

development usually coincides with early adolescence and the achievement of sexual and physical maturity. Along with this early apex, theories stressed such attributes as self-control and the ability to adopt cultural roles. However, in more recent theories, the notions of biological and psychologic maturity or generativity have been specifically decoupled; hence concepts of maturity, following Mead's (1970) suggestion, extend beyond the faithful, stability-guaranteeing adherence to societal norms and emphasize continued capacity for critique, openness, and transformation of individuals and society. As Jung (1933) stated, the emphasis becomes less on socialization and more on individuation. In general, we value the individual's attempts at meaning making, while valuing less the propensity to adopt the meanings that are given by conventional social roles and settings.

A good example of this new emphasis is given by Baumeister's (1986) discussions on the nature of the self. In medieval times, there was little emphasis on inner self and its struggles. For example, there was almost no autobiographical writing. Interest in portraying life stories rather was in exemplary and ideal life, as in the stories surrounding saints. Similarly, in painting, the focus was not on the portraits of ordinary individuals. Rather, the emphasis was on biblical and religious topics and images, which were depicted in a very formal and idealized way, such as icons. Nor did the individual author or artist matter: for example, early medieval icons or manuscripts were not signed; the role of artists was not to impress their own individuality on the work, but rather to provide a faithful copy of an original. In the sixteenth century, in contrast, a focus on the inner self emerged. The notion became popular that the "self" is a hidden, inner space that is different from one's outer appearance, and writers and artists' now attempted to characterize this new inner self.

#### 1.09.2.2.2 *The role of context*

One of the consequences of the new model of persons was the vindication of the role of context (see Labouvie-Vief & Chandler, 1978). Thus, rather than thinking of individuals as mere replications of the same universal law, individuality and the contextual features that give shape to it became significant as essential features of developmental theories. A new theoretical emphasis on context was influenced by trends early in the twentieth century, when there was a flurry of philosophical and scientific writings that dealt a profound blow to the notion that what is right and true could forever be defined in an objective, universal, timeless

way (see Labouvie-Vief, 1994, for discussion). Instead, many thinkers began to propose that truth (at least as far as it concerns relatively complex states of affairs) is not a static and universal thing, but that it is best understood as a dynamic process, something that evolves, that is being constructed, and that is being continually redefined. Truth has, therefore, an explicit historical and developmental perspective, and is moved out of the realm of axioms and ideals into the very context of human practice (see Labouvie-Vief, 1994).

These contextualist assumptions have modified recent theoretical attitudes regarding psychological development and aging, as well as the fulcrum that is to balance the two, namely maturity. Early theories tended to adopt either of two dualistic views: organicism (see Reese & Overton, 1970), with its emphasis on transcultural and transhistorical processes, or mechanism with views that focus on environmental contingencies that shape behavioral repertoires. In contrast, many current views combine assumptions of these two models. They assume that, on the one hand, sequential and fairly pervasive processes of growth and decline are a part of life-span changes; yet on the other hand, these changes are modified by historical or cultural contexts, as well as by specific individual experiences. Indeed, a widely held view in the late 1990s is that basic developmental pathways may be shared by many groups of individuals; yet at the same time, the specific up- and downturns or even the specific directions such pathways may take for any one individual, are based on the experiences they encounter along the journey of their development. Thus from a core pattern, one may envision different pathways that partially divide in fan-shaped fashion (see Baltes, 1987; Bronfenbrenner, 1979).

#### 1.09.2.2.3 *Individualism vs. relatedness*

With the rediscovery of context also came the realization that the mind is not just an intrapersonal entity of separate selves. Although awareness of self and mind as relatively separate is essential for functioning in a complex society (see Elias, 1982; Labouvie-Vief, 1994), it is also true that all individual minds or selves participate in the interrelationships that constitute a collective. Thus, classical models of mind and self have idealized the individual in terms of such characteristics and values as individuality, autonomy, independence, achievement motivation, and identity; in contrast, such values as relatedness, compassion, and dependence were seen as less important and indeed as less mature (see Bakan, 1966; Gilligan, 1982; Guisinger & Blatt, 1994).

In contrast, many theories have begun to account for the profound ways in which humans are interrelated (e.g., Bowlby, 1969, 1988; Erikson, 1982). Indeed, such interrelatedness must constitute a basic, biologically based propensity in a species that is neotenuous and has a long period of extrauterine development. Failure to be in relation to others, in turn, is associated not only with a host of psychological disorders, but also biological disorders such as depressed immunocompetence (Bakan, 1966; Weiss, 1987). As a consequence, a number of more recent models of development have emphasized the need for a balance between autonomy and dependence of relatedness as attributes of mature adults (e.g., Erikson, 1982; Gilligan, 1982; Guisinger & Blatt, 1994; Kohut, 1977).

#### **1.09.2.2.4 Coherence of the individual**

The roles of context and relation also have supported how we look at the single individual, not just the individual in relationship to other individuals. An assumption often made in the past—in fact explicit in Plato's theory of mind—was that characteristics emanate from some inner structures such as an ego or some other central processor that gives coherence and unity to self and experience. However, many recent theories also emphasize that different, and sometimes vastly contradictory, experiences and attributes may be activated by different contexts or relationships. Thus individuals are no longer seen as having a single coherent mind, self, or personality, but are seen to combine in themselves contradictions and conflicts in ways that define the unique individuality of a person. Thus concepts of the mind or self are becoming more "modular," with specific modules activated in different contexts and relationships (e.g., Ashmore & Ogilvie, 1992).

#### **1.09.2.2.5 Modes of thought**

Another aspect of the mind that has been deconstructed is the notion that the mind can be described by a single mode of thinking. Traditional theories of development have placed primary emphasis on rational thinking and conscious decision-making. In turn, such functions of thought as are expressed in artistic, literary, or religious activities were considered to form a less valued aspect. Indeed, they were considered to be less mature or more childlike (see the discussions on Piaget and Freud below).

Labouvie-Vief (1994) recently has addressed this issue. In her view, rationality primarily has been associated with progressive trends in

development, while emotions, intuition, and symbolic thinking were identified with regressive trends. In actuality, however, rational views of development are usually undergirded by a consistent language of images and symbols. For example, we typically think of rational functions as masculine while those related to the emotions are stereotyped as feminine. Similarly, we tend to associate the rational with light, airy heights, and golden hues, while associating the nonrational with darkness, earthy and watery depths, and silvery or even black shades. Such symbolism is pervasive in art, literature, and religion, but it also permeates the theoretical discourse about development. Thus an emerging way to think about the mind is to propose the parallel development of two functions. Accordingly, many theorists (e.g., Bruner, 1986; Epstein, 1990; Labouvie-Vief, 1994) now suggest that more narrative, organismic, and experiential modes of thinking form modes of thought that can develop in parallel to rational thought; these modes, then, can show progressive features and occur even at very advanced levels of complexity.

#### **1.09.2.2.6 Growth vs. decline**

A further shift that has occurred in our views of human nature is a deconstruction of the notion of perfection that was basic to past views of rationality. As Gergen (1991; see also Sampson, 1977) suggested, the traditional view was that rationality assured progress and success, that it ultimately had as its goal perfection. This belief in the perfectibility of human nature also has guided many past theories of development. For example, in Piaget's theory, development reaches its apex as individuals understand logical laws; thereby, Piaget believed, ideas of necessity and perfection could come to guide behavioral regulation.

This "psychology of more" has posed a conceptual problem for researchers in the field of adulthood: if youth is construed as perfection, it is difficult to reconcile the notion of adulthood growth with some of the obvious declines that are a part of adult life. Yet, as suggested by Labouvie-Vief (1980, 1981, 1982a, 1982b; see also Baltes et al., 1998), losses are not unique to later life. Indeed, the whole process of development is interwoven with losses, and often gains in development are related to losses in a direct gain-loss relationship. Thus often what is called decline or regressions can result from the operation of developmental processes that are usually related to progressive development, or growth.

Several principles demonstrate this trade-off between growth and decline. For example, as



development proceeds, old structures often become displaced or function with less efficiency. Thus it is well demonstrated in the child development literature (for review, see Labouvie-Vief & Schell, 1982) that as individuals move on to more complex levels that are based on more abstract rules and principles, lower-order performance actually becomes less efficient. Another principle is that of selective depletion, a principle widely important in evolutionary processes. Much as evolution is based on an oversupply of organisms out of which only the “fittest” are selected, so a similar mechanism operates in developing systems. For example, the progressive maturation of the brain during the first stages of life actually involves profound losses as well. Originally, neurons are supplied in abundance, but as workable networks are established, many neurons are “weeded out” through a process of selective death. Finally, development typically involves increasing degrees of specialization, or the trading off of potential for realized structure. Thus, smooth functioning at the system or collective level can be traded off for specialization at the individual level.

In Labouvie-Vief's work, these trade-off processes are proactive and progressive. In addition, Baltes (1987) has added to these progressive trade-off processes reactive ones that may reflect processes of aging proper. Thus, as in the process of growing older, psychological and social resources decline, individuals actually may evolve highly adaptive and resilient compensatory strategies. In addition, individuals who experience a loss of capacity may compensate by selecting one area and focusing an increased amount of attention and energy on it. Such selective optimization at times is associated with extraordinary and highly evolved skills.

#### **1.09.2.2.7 Stability vs. plasticity**

The above discussion further highlights that even in later stages of development, a considerable capacity exists for positive change. Earlier stability models, such as Freud's psychoanalytic theory, assumed that individuals were fairly fixed in later life, and indeed that interventions such as psychotherapy were not to be recommended for adults older than about 40 years of age. In contrast, following the lead of Bloom (1964), more recent positions criticized this stability position. Instead, there now is a tendency to explore the potentials for change and plasticity even in late life.

In sum, the twentieth century has been witness to a dramatic revision in notions of development and concepts of what constitutes

advanced development. Where original concepts often emphasized a tightly regulated developmental course that primarily reflected biological regulation, that was culturally and historically universal, and that had as its goal a coherent and conscious individual, in the late 1990s views are highlighting characteristics that appear more appropriate to an older and worldwide community.

### **1.09.3 COGNITIVE DEVELOPMENT**

It is proper to start a discussion of theories of life-span development with a review of cognitive development. As already stated, early theoretical views of development were very much influenced by a prevalent rationalism, and academic psychology definitely saw as its primary task to expand on and refine early notions of the rational person, which stood as a prototype of successful, adaptive development. In fact, as Riegel (1976) noted, until the middle of the century, most life-span discussions centered around issues of cognitive development which virtually dominated the literature.

Following historical trends, we begin this section with psychometric research on intelligence, which served as a sample case in which many questions about life-span development were raised for the first time. Thus, psychometric research on intellectual functions formed the arena in which questions about life course development were raised and methodologically elaborated. This section is followed by discussing the more recent influence of cognitive-developmental tradition, which has added to these earlier discussions new ones about the structure and function of intelligence and cognition—concepts which had been developed in the context of predicting educational outcomes in the young (see Baltes & Labouvie, 1973; Labouvie-Vief, 1985)—at later stages of the life-span.

#### **1.09.3.1 Psychometric Intelligence**

As Gardner and Clark (1992) have described it, the “psychometric approach to intellectual development seeks to define and quantify dimensions of intelligence, primarily through the collection of individual differences data and through the construction of reliable and valid measurement scales” (p. 16). Thus, researchers who adopt the psychometric approach address questions such as: How can intellectual development be quantified? How can such quantifications be used to predict achievements later in life? How can the intelligence of individuals be meaningfully compared? What factors make up

intelligence, and do the factors change with age? (Siegler & Richards, 1982).

Early research on the life course of intelligence was strongly guided by the stability assumptions of the existing theoretical frameworks, which considered intelligence usually as a basic biological endowment with a strong genetic base. In fact, Riegel (1976) noted that early research on intelligence neglected contextual differences, and cultural background was not even believed to affect levels of intelligence. In contrast, debates in intelligence research in the late 1990s focus primarily on issues related to the contributions of genetic/hereditary and sociocultural/contextual influences to individuals' intellectual performance, on the different developmental trajectories of intellectual abilities across the life-span, and on secular trends in intellectual performance (Flynn, 1984, 1987; Schaie, 1994, 1996). In particular, research on the development of intellectual abilities in adulthood and old age has shown different patterns of aging for different mental abilities and has documented much more intra-individual plasticity, and interindividual and intercohort differences than was previously expected (Baltes, 1987; Labouvie-Vief, 1985; Schaie, 1996). Thus, in the following we will review the major findings with regard to the development of intellectual abilities across the life-span, placing particular emphasis on intelligence development during the adult years.

Perhaps the most interesting research in terms of psychometric intelligence since the late 1950s has been conducted with adults of different ages, plotting the developmental trajectories of different mental abilities across the adult life-span and into old age. This research has greatly enriched our knowledge about intellectual development across the life-span and has mostly revised the decrement model of intellectual aging that persisted in the 1940s, 1950s, and 1960s (Labouvie-Vief, 1985). Although early research suggested dramatic "declines" in intelligence beginning as early as 20 or 30 years of age, more recent findings with regard to intellectual functioning demonstrate that these age differences are confounded with cultural-historical change, and that genuine normative declines set in much later, probably in the sixth life decade. In addition, researchers now emphasize the potential of gains and continued growth along with losses (Baltes, 1987; Labouvie-Vief, 1977) as well as the capacity for plasticity during all phases of adulthood (Baltes & Baltes, 1990; Dixon & Bäckman, 1995). In particular, three developments have greatly influenced the research on intellectual functioning in adulthood.

#### 1.09.3.1.1 Differential decline

First, the adoption of Horn and Cattell's (1966) theory of *fluid* ( $G_f$ ) and *crystallized* ( $G_c$ ) intelligence as an organizing theoretical framework alerted researchers to the possibility of ability-specific age trajectories and the multidirectionality of intellectual development during the adult years (Baltes, 1987). Horn and Cattell's (1966) proposition that abilities which primarily reflect a person's knowledge inculcated through the process of acculturation (i.e., crystallized intelligence,  $G_c$ ) continue to increase into the sixties and seventies, whereas abstract reasoning abilities (i.e., fluid intelligence,  $G_f$ ) start to decline in middle adulthood, is now supported by a large body of research (Horn & Hofer, 1992). Horn and Hofer's (1992) explanation that the observed declines in  $G_f$  seem to be primarily related to the loss of ability to maintain close attention and to divide attention is consistent with findings obtained from studies conducted within the information-processing paradigm. Numerous experimental studies have shown that changes in abstract reasoning abilities show strong associations with changes in working memory and reduced speed of processing (Salthouse, 1991). As several researchers have suggested, these age-related changes in working memory and speed of processing may reflect neurological changes in the aging person and may explain that even under most optimal training conditions, older adults do not outperform younger adults on complex cognitive tasks such as a mnemonic skill (Baltes & Kliegl, 1992; Kliegl, Smith, & Baltes, 1989).

#### 1.09.3.1.2 The role of context

The second development that has contributed to a revised view on intellectual development during adulthood is related to findings from a number of longitudinal studies of aging (see Schaie, 1983). Such studies tend to suggest much less dramatic declines than cross-sectional research had indicated. In particular, the work of Schaie (1996), working with the Seattle Longitudinal Study (SLS), has shown that these patterns point to very systematic and theoretically important differences between cross-sectional and longitudinal research.

The SLS is unique because it was designed as a cohort-sequential study (Schaie, 1965, 1986; see Figure 1). This research design consists of a coordinated series of longitudinal and cross-sectional studies and permits the joint study of individual development and the effects of cultural-historical change. Thus, this design overcomes the shortcomings of the cross-

sectional and longitudinal methods traditionally used in developmental psychology and allows generalizations about the nature and dynamics of age-related change, including the variability created by sociocultural conditions. Moreover, the SLS has followed its aging participants over a 35-year period, thus providing the most comprehensive data set on intellectual development in adulthood (see Schaie, 1994, 1996).

What are the major findings from the SLS? Although there is a plethora of findings from the SLS (for a comprehensive review, see Schaie, 1996), only the major findings will be highlighted here. First, Schaie and his colleagues found that there is no uniform pattern of age-related changes in adulthood across all intellectual abilities. Cross-sectional analyses, for example, show that some primary abilities (i.e., spatial orientation, inductive reasoning) peak in young adulthood, whereas others peak in midlife (i.e., verbal meaning and number; see Figure 2). In contrast, longitudinal analyses showed at least modest gains for all abilities from young adulthood to early middle age (see Figure 3).

Second, longitudinal analyses also showed that, except for word fluency, average age decrements in psychometric abilities cannot be reliably documented prior to age 60. However, reliable average decrement was indeed found for all abilities by age 67 (Schaie, 1994). Third,

results from the SLS have also documented substantial generational (cohort) differences in psychometric abilities, that is, different generations show different patterns of intellectual aging. For example, more recent cohorts have been shown to perform at a higher level on the abilities of verbal meaning, inductive reasoning, and spatial orientation, whereas older cohorts outperform younger cohorts on number and word fluency.

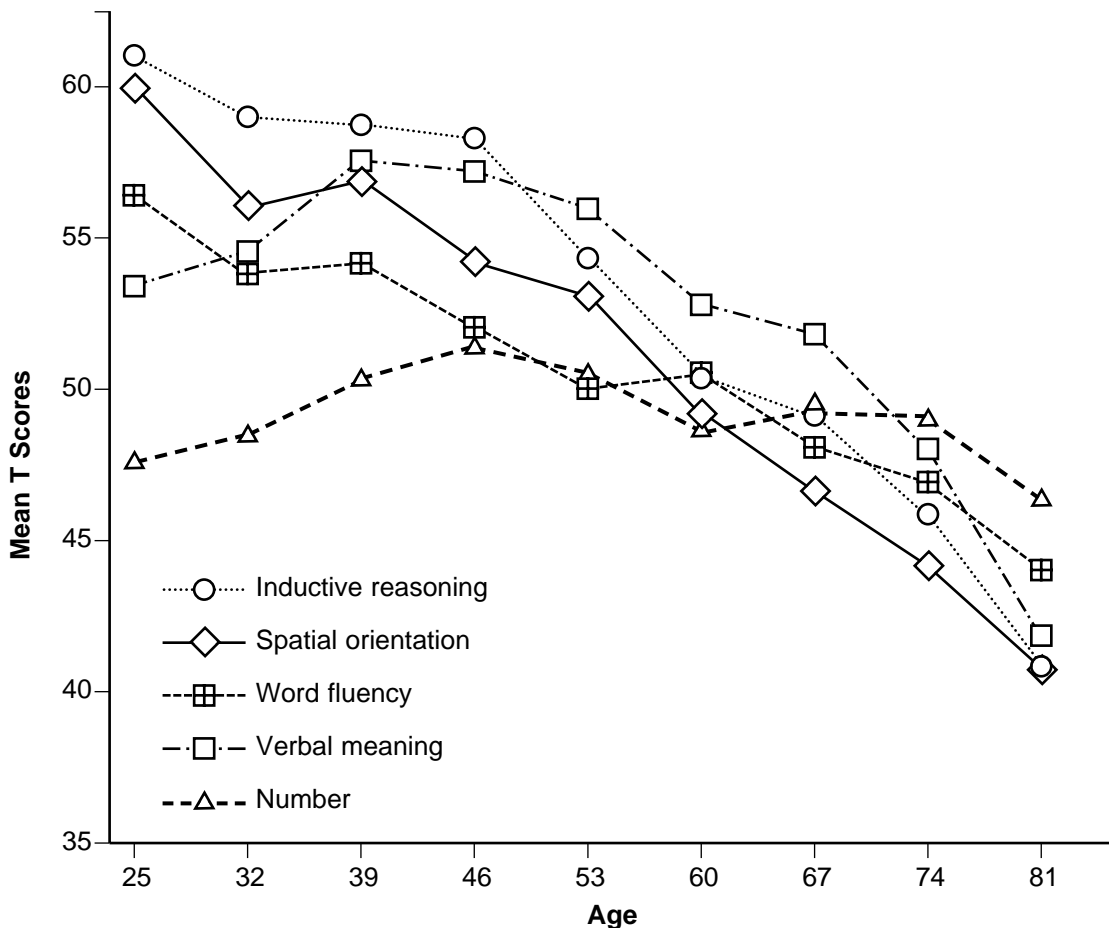
Similar patterns of generational differences in intellectual functioning have also been found in other Western countries and have been termed the "Flynn effect" (Flynn, 1987). The Flynn effect, in essence, refers to the well-documented rise in intelligence test performance in subsequent generations. This steady increase seems to reflect the combined effect of a variety of environmental influences, such as increased complexity of life, improvements in education, health care, and nutrition and other factors more difficult to pinpoint (Flynn, 1987).

The SLS also provides a great amount of information with regard to contextual antecedents of individual differences in age-related change, thus emphasizing the sociocultural/contextual dependence of patterns of intellectual aging. Schaie and his colleagues, for example, showed that the absence of cardiovascular and other chronic diseases (Hertzog, Schaie, & Gribbin, 1978), the presence of favorable environmental circumstances, the

Study Waves					
1956	1963	1970	1977	1984	1991
S <sub>1</sub> T <sub>1</sub>	S <sub>1</sub> T <sub>2</sub>	S <sub>1</sub> T <sub>3</sub>	S <sub>1</sub> T <sub>4</sub>	S <sub>1</sub> T <sub>5</sub>	S <sub>1</sub> T <sub>6</sub>
(N = 500)	(N = 303)	(N = 162)	(N = 130)	(N = 92)	(N = 71)
	S <sub>2</sub> T <sub>2</sub>	S <sub>2</sub> T <sub>3</sub>	S <sub>2</sub> T <sub>4</sub>	S <sub>2</sub> T <sub>5</sub>	S <sub>2</sub> T <sub>6</sub>
	(N = 997)	(N = 420)	(N = 337)	(N = 204)	(N = 161)
		S <sub>3</sub> T <sub>3</sub>	S <sub>3</sub> T <sub>4</sub>	S <sub>3</sub> T <sub>5</sub>	S <sub>3</sub> T <sub>6</sub>
		(N = 705)	(N = 340)	(N = 225)	(N = 175)
			S <sub>4</sub> T <sub>4</sub>	S <sub>4</sub> T <sub>5</sub>	S <sub>4</sub> T <sub>6</sub>
			(N = 612)	(N = 294)	(N = 201)
				S <sub>5</sub> T <sub>5</sub>	S <sub>5</sub> T <sub>6</sub>
				(N = 628)	(N = 428)
					S <sub>6</sub> T <sub>6</sub>
					(N = 690)

S = Sample; T = Time of measurement

**Figure 1** Design of the Seattle Longitudinal Study. "The course of adult intellectual development," by K. W. Schaie, 1994, *American Psychologist*, 49, p. 305. Copyright 1994 by the American Psychological Association. Reprinted with permission.



**Figure 2** Cross-sectional mean T score for single markers of the primary mental abilities (1991 data). “The course of adult intellectual development,” by K. W. Schaie, 1994, *American Psychologist*, 49, p. 306. Copyright 1994 by the American Psychological Association. Reprinted with permission.

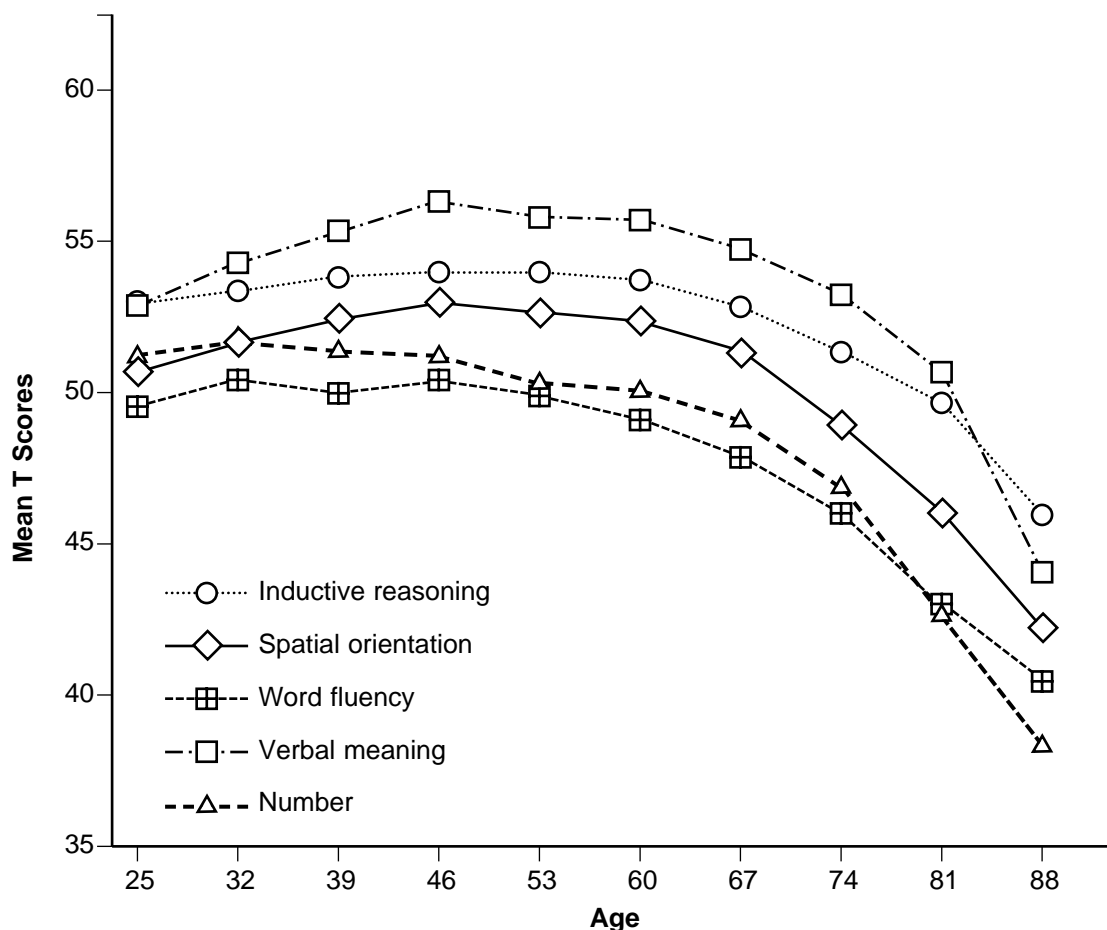
involvement in intellectually stimulating activities, the maintenance of a high level of perceptual processing speed, a flexible personality style at midlife, as well as satisfaction with one's life's accomplishments (Schaie, 1984) all are important contributors to successful intellectual aging. The role of complex living arrangements, including a person's work environment, for the maintenance of high levels of intellectual functioning has also been documented by Kohn and Schooler (1973; Schooler, 1987) and has been documented in animal models for a long time (for a review, see Labouvie-Vief, 1985).

#### 1.09.3.1.3 Plasticity of intelligence

Finally, Schaie and Willis (1986) provided the most compelling evidence for the intraindividual plasticity (i.e., intraindividual modifiability) of intellectual functioning when they showed that reliable decline in the fluid abilities of spatial orientation and inductive reasoning was reversible through specific training efforts.

This finding, by the way, is consistent with the increasing research documenting the plasticity of the aging human brain (for a review, see Labouvie-Vief, 1985; Scheibel, 1996).

This latter finding can also be seen as part of the third major development that contributed to a changed view on adult intellectual development. In the early 1970s, Baltes and Labouvie (1973) suggested that researchers should systematically examine the modifiability of older adults' intellectual abilities. During the first phase of cognitive intervention research with older adults, these suggestions resulted in a number of studies showing that older adults' performance on intelligence tests could be improved through simple practice (Hofland, Willis, & Baltes, 1981) as well as through special training programs (Baltes, Dittmann-Kohli, & Kliegl, 1986; Baltes & Willis, 1982; Hayslip, 1989). Later, Baltes and his colleagues (Baltes & Kliegl, 1992; Kliegl, Smith, & Baltes, 1989), however, also showed that older adults' range of plasticity is more limited than younger adults' range of plasticity. Using the “testing-the-limits



**Figure 3** Longitudinal estimates of mean T scores for single markers of primary mental abilities (from 7-year within-subject data). "The course of adult intellectual development," by K. W. Schaie, 1994, *American Psychologist*, 49, p. 306. Copyright 1994 by the American Psychological Association. Reprinted with permission.

paradigm," Baltes and Kliegl (1992), for example, trained older and younger adults in the mnemonic strategy of the method of loci and showed that in no instance were the trained older adults able to outperform the trained younger adults. Instead, training actually increased the separation between younger and older adults, providing evidence that older adults' reserve capacity seems to be more limited compared to younger adults' reserve capacity, an argument that had previously been made by Botwinick (1977).

An interesting contribution of cognitive intervention research in adulthood is that it also highlights the interface between cognitive functioning, personality, and affect. In particular, several researchers have shown that improvements in older adults' intellectual performance can even be achieved, through interventions that focus on personality-related processes such as self-efficacy beliefs (Labouvie-Vief & Gonda, 1976; Lachman, Weaver,

Bandura, Elliott, & Lewkowicz, 1992) or anxiety reduction (Hayslip, 1989; Hayslip, Maloy, & Kohl, 1995) rather than on direct cognitive strategies training. These studies have provided substantial evidence for a rather strong connection between personality and cognitive functioning in adulthood, an issue to be further discussed in a later section.

### 1.09.3.2 Everyday Intelligence

In recent years, researchers' interest in adults' intellectual performance has also shifted to some new areas of inquiry. In her 1985 review of the literature on adult intelligence and cognition, Labouvie-Vief (1985), for example, argued for a "re-examination of the concept of intelligence as an adaptive capacity" (p. 501) and presented findings with regard to the adaptive function of cognition in adulthood. This notion was echoed by Salthouse (1990)

when he stated that “one of the greatest challenges in the field of psychology and aging is to account for the discrepancy between the inferred cognitive status of older adults based on their performance in psychometric testing situations and cognitive laboratories and that derived from observations of their successful functioning in everyday situations” (p. 310).

Thus, in recent years older adults’ performance with regard to practical or everyday problems has become a major focus of cognitive aging research (Poon, Rubin, & Wilson, 1989; Puckett & Reese, 1993). Several researchers, for example, have examined older adults’ intellectual performance with regard to tasks that are designed to simulate situations of everyday life (for review see Diehl, Willis, & Schaie, 1995). Overall, this research has shown that older adults do not solve everyday problems as effectively as middle-aged individuals but that they usually outperform young adults (Denney & Palmer, 1981; Denney & Pearce, 1989). That older adults’ practical problem solving ability is not completely independent of their functioning on psychometric tests was shown by Diehl et al. (1995). These authors found that older adults’ performance on a set of observed tasks of daily living showed substantial relations to the abilities of perceptual speed, memory, fluid intelligence, and crystallized intelligence. Indeed, fluid intelligence was found to be the strongest predictor of older adults’ performance on everyday problem-solving tasks (see also, Willis & Schaie, 1986). Overall, these findings provide support in favor of a hierarchical perspective of everyday cognition. The main assumptions of this perspective have been outlined by Willis and Schaie (1993) and can be summarized in three postulates. First, problems encountered in everyday life require the activation and application of multiple mental abilities and processes for their solution. Second, different types of everyday problems require the application of different mental abilities. Third, competence with regard to basic cognitive abilities and processes is a necessary but not sufficient condition for the successful solution of everyday problems; domain-specific knowledge is likely to be required as well for successful everyday problem solving.

In summary, theory and research on the development of psychometric intelligence has always been at the forefront of life-span developmental psychology (see Baltes, 1987) and has greatly influenced researchers’ thinking in other domains. For example, concepts such as multidimensionality, multidirectionality, plasticity, or sociocultural influences on development are among those that are now increas-

ingly incorporated in research on personality development and the self. Moreover, perspectives on positive development in adulthood and aging were first explored in the context of intellectual and cognitive development and are now translated into other areas of human development.

### 1.09.3.3 Cognitive-developmental Theories

As compared to the psychometric approach, cognitive-developmental approaches are relative latecomers to the field of life-span changes in cognitive functioning. Yet their origins come out of the same concerns about validity that gave rise to much contemporary research within the psychometric tradition, namely issues relating to the validity of tests when applied outside of the settings for which they were originally developed. Thus, similar to Piaget’s original criticism that quantitative differences in intellectual achievements reflect core differences in the organization of thinking, researchers in the field of adulthood wondered if thought structures in adulthood and later life might not reflect coherent reorganizations of thinking (see Labouvie-Vief, 1980, 1982a, 1982b; Riegel, 1973).

#### 1.09.3.3.1 Piaget’s legacy

When Piaget’s cognitive developmental view arrived on the US scene, it created no less than a revolution in the field of developmental psychology. His proposal that intelligence and cognition evolved in the same way that other biological structures do, in terms of a series of qualitatively different and sequentially organized stages, proved an extremely powerful umbrella for a host of developmental changes from infancy to early adulthood. In the late 1990s, it is widely accepted that the evolution of structures akin to those he proposed underlies a large variety of cognitive competencies, from those that are rather formal and concern themselves with scientific thinking, to those that are more focused on everyday kinds of issues such as the understanding and regulation of emotions and representations of the self (Labouvie-Vief, DeVoe, & Bulka, 1989).

Nevertheless, the theory recently has been widely criticized. In keeping with the issues discussed in the first section of this chapter, Piaget’s theory evolved in an intellectual context that emphasized the priority of rational functioning, which formed the endpoint of Piaget’s theory. Similarly, the theory proposed a single, universal pathway to this endpoint. It further emphasized coherence of functioning at

each stage of development, such that stages were assumed to provide broad integrative frames for all of the individual's thought and behavior.

This objectivism in Piaget's theory emerges as somewhat of a paradox, since we have noted that the theory was based on the basic assumption—shared in many ways with Freud—that mental life could be explained as a form of biological life. Thus, his work also marks a watershed in a broad trend to re-embed the mind in the organismic and biological world. In the abstract, that assumption is evident in many aspects of the theory: in its empirical implementation, however, Piaget's theory is successively slanted towards a form of mentalism that becomes dissociated from its organic base.

Riegel (1973) has commented on this slant and argued that in Piaget's theory, development is represented as a successive abstraction of thought from a rich organismic base. While in the theory we are confronted with children who are emoting, playing, symbolizing, and struggling with issues of authority, Piaget's adolescent is described almost exclusively in terms of idealized structures of mathematical and scientific thinking. These structures begin, according to Piaget, in cognition's close ties to sensorimotor processes, but gradually they become more autonomous and less dependent on immediate sensory experience. Thus sensorimotor intelligence moves into the symbolic processes of preoperational intelligence, and then begins to concern itself with more abstract relations and operations.

In "concrete operations," these still are closely tied to the concrete realities of the perceived world, but in "formal operations" they become the ability to operate on purely abstract processes, relations, and thoughts. Thus, in Piaget's theory, meaning systems that originate in the organismic, the sensorimotor, the figurative, the dynamic, and the personal gradually are displaced by ones that are abstract, conceptual, stable, conventional, and impersonal. But in general, the theory does not offer an integration of the two, except in the most abstract discussions of the dynamics of development.

These general criticisms have led, since the late 1970s, to two important expansions of a Piagetian view. One pathway has been to develop neo-Piagetian theories that expand Piaget's views to other domains and contexts (see Case, 1991; Fischer, 1980). In general, these theories hold to the notion that there is an ordered sequence of levels of complexity. Yet they make a clearer differentiation between this logical sequence of levels of complexity, and

how these levels actually are translated into thought and behavior. Thus issues such as context and domains of familiarity all become significant variables to be added to a theoretical account of development. Further, another significant trend has been that the levels are generalized beyond the domain of scientific thinking to domains that are more strongly related to emotions, self, values, and relationships (Case, 1991; Damon & Hart, 1982; Fischer & Ayoub, 1994).

Another core approach has primarily focused on adults, and on re-evaluating notions of maturity that may be better applicable to adulthood. As argued by Labouvie-Vief (1994), the role integrating formal and organismic aspects of thinking may become especially important in adulthood, where individuals become more active participants in the construction of knowledge. To an extent, the dynamics of early development may require that the individual put aside private and inner forms of meaning, since the process of socialization requires an outward turn. However, in later development that outward turn must be balanced with an inward orientation: a higher degree of introspection, understanding of intrapersonal dynamics, and historical and psychological analysis.

#### *1.09.3.3.2 Mature reasoning structures*

Riegel's (1973) notion of the emergence of dialectical operations in adulthood has inspired several authors to describe such dialectical, multilevel modes of thinking. Riegel's work has remained rather abstract, but many empirical approaches took their impetus from Perry's (1970) influential study on how college men rediscover the relationship of reasoning to subjective and interpersonal variables. A similar study has since been done with women (Clinchy & Zimmerman, 1982; see also Belenky, Clinchy, Goldberger, & Tarule, 1986). Both of these studies argued that formal thinking makes the individual uniquely vulnerable to a dualistic emphasis on product rather than process, stasis rather than change. This rigidity was loosened as the youth moved into a position of contextual relativism. Now the individual rejected the notion of any one certain truth and asserted instead that truth is inherently relative to a context. Eventually, this fairly radical relativism is integrated in a final position of "commitment in relativism": in the midst of multiple logical choices, the individual now accepts the need for an integration that is ultimately subjective.

The notion that a stage of contextual relativism follows one of the certainty of

formal operations has influenced most subsequent work, but most authors have sided with Kohlberg in searching for a final stage that goes beyond a merely subjective synthesis and strives for some higher order integration. Just how to describe this most mature level of cognitive functioning has varied, however, from author to author. One approach is the work of Commons and collaborators (e.g., Commons, Armon, Richards, & Schrader, 1989; Commons, Richards, & Kuhn, 1982). These authors suggested that, although formal thinkers are able to abstract order relations in a systematic manner, the entire system is not regarded as a single entity that can be analyzed in terms of characteristics and relationships and thus systematically compared to other systems. In contrast, more advanced thinkers are able to describe features of different systems in terms of an underlying common language which yields transformations across systems. This work is echoed by that of other authors who also note that the hallmark of mature adult thinking is the ability to move from intrasystem thinking to the ability to construct co-ordinations and transformative relationships between abstract systems (e.g. Labouvie-Vief, 1982a; Fisher, Hand, & Russell, 1984; Sinnott, 1989).

Other authors have specifically suggested that adulthood brings an enhanced ability to think dialectically (e.g., Basseches, 1984; Kramer, 1989; Labouvie-Vief, 1982a, 1982b, 1992; Reich, 1990; Sinnott, 1989); thus, adults are better able to analyze systems and concepts in terms of history, context, subjective processes, and change and movement. Kramer (1989), for example, proposes that thinking from adolescence to mature adulthood develops from a dualistic and absolutistic, through a relativistic, to a dialectical level. For example, in a comparison of young, middle-aged and elderly adults, Kramer and Woodruff (1986) found older adults to display the highest level of relativistic and dialectical thinking. There also was some evidence that Piagetian formal thinking was prerequisite but not sufficient for dialectical thinking, but that relativistic thinking actually was necessary but not sufficient for formal operations.

Much of the work above has concentrated on relatively abstract features of the coordination between systems. Just what are the kinds of systems and system features that mature individuals are able to analyze? Several studies have focused on how individuals can coordinate such abstract concepts as truth and certainty on the one hand, and subjectivity and interpretation on the other. For example, Kitchener and King (1981; see also Kitchener & Brenner, 1990;

Kitchener, King, Wood, & Davison, 1989) have offered a detailed account of how individuals come to coordinate such categories as objective knowledge and subjective belief. These authors also carry further Perry's contention that the mark of mature rationality is a return of a new, if more educated, form of subjectivism. Rather, they suggest that from this more abstract form of subjectivism, the individual again begins to search for a set of "objective" criteria. However, these criteria are more procedural and dynamic: even though one may not, in principle, know what is good evidence, one has a general procedure for distinguishing solid evidence from less trustworthy evidence by examining the process through which knowledge has been gained.

This work has many implications for modern discussions about the nature of mature reason. Beyond these implications, it may also help highlight the cognitive roots of some major psychological differences between adolescents and adults. Labouvie-Vief (1982a), for example, has argued that the adolescent's understanding of truth as certain and computable may be a significant factor in her or his defense structure. Since that structure is based on a dualism between objective and subjective processes, it does not involve a mechanism by which such defensive cognitive distortions can be analyzed and, therefore, corrected. In turn, the movement to a model of knowledge that is more historically situated may bring a more integrated structure that, by analyzing its own subjectivity, provides a more powerful device for self-regulation and correction (Blanchard-Fields, 1986; Pascual-Leone, 1984; Wood, 1983).

#### *1.09.3.3.3 Cognitive development and the self*

The research discussed above suggests that as individuals move into mature adulthood, they are better able to relate the process of thinking back to a set of subjective processes (intentions, values, historical conditions, etc). Thus not surprisingly, these lines of thinking also have been extended to individuals' thinking about self and emotions. A major influence on this work has been Kohlberg's (1984) theory of moral development. Kohlberg (e.g., 1984) has addressed himself most explicitly to morality, but his theory has a much broader sweep and in fact involves an implicit theory of evolving self-structures around which values and emotions are organized. These implications have been worked out by a group of subsequent researchers both in the childhood (e.g., Damon & Hart, 1982; Selman, 1980) and adulthood (see below) areas. For example, Kegan (1982) has presented



a theory of self-development from adolescence to mature adulthood. According to Kegan, the youthful and/or conventional self remains fused with an interpersonal and then institutional matrix, and the ability to experience distinctness and an individuated self remains limited. That capacity to maintain a more autonomous sense of selfhood emerges at the final stage, when self and other can be understood as entities that transcend interpersonal and institutional meanings. Thus, both a more authentic sense of selfhood and a deeper capacity for intimacy can result.

Armon (1984) has applied Kohlberg's notions to conceptions of what constitutes a good or ethical life and work. In her study, preconventional thought is focused around merely individual interests and desires, while conventional thinking becomes organized around such goals as socially beneficial work, positive interpersonal experiences, financial security, and social utility. For the postconventional individual, the potential conflict between social commitment and self-fulfillment is heightened and the individual aims at balancing responsibility to self with that to society.

Fowler (1981) has also applied Kohlbergian (as well as Eriksonian) notions to another domain, that of faith and religious values. Fowler argues that the preconventional individual has a magical and concrete conception of God. At the conventional level, the orientation is based on acceptance of cultural values with only little critical evaluation: religion now is oriented towards maintaining dogma and interpersonal validation. In contrast, the postconventional individual becomes both critical of conventional religion, and evolves a perspective in which conventional religious frameworks form separate though equivalent paths towards more universal spiritual goals.

Another theory proposing a broad reorganization of the self in relation to the social collective is Loevinger's (1976; Loevinger & Wessler, 1970) model of ego development. Loevinger proposed that the impulsiveness of the child gives way to a conventional mode in which one's own subjective inner life is suppressed and subordinated to social norms. The mature individual, however, evolves a more flexible language in which the conflicts between impulse and norm, self and society, or inner and outer, are first acknowledged and then integrated within more self-chosen standards. Cognitive complexity thus replaces a youthful language of self-regulation preoccupied with physical, mental, or emotional control, good-bad dichotomies, and little or no tolerance of intrapersonal or interpersonal conflict. The developmental significance of Loevinger's con-

ception and measure has been demonstrated in age-related trends in both cross-sectional and longitudinal samples (e.g., Cook-Greuter, in press; Hauser, 1976; Redmore & Loevinger, 1979). Ego development is also a good index of such dimensions of developmental complexity as moral development and empathy (see Loevinger, 1976b) and intellectual development (see below).

Labouvie-Vief and collaborators (Labouvie-Vief, Chiodo, Goguen, Diehl, & Orwoll, 1995; Labouvie-Vief, Diehl, & Coyle, 1996) developed a framework within which to examine such changes. In this framework, statements about self and others (in particular, parents) are classified into five developmental levels that are ordered in terms of increasing complexity (see Table 1). In this research, younger or less mature individuals framed self and others in terms of a conventional perspective: self and others were described in terms of an organized, codified, and abstract set of role expectations. At a more advanced level, the institutional values become susceptible to doubt and criticism: for example, such values can be "carried too far." Instead, a dynamic perspective evolves in which descriptions of self and others convey in vivid language the unique and evolving experience of individuals within the context of their particular life histories. Lives now are understood in the context of multiple frames, cultural, social, and psychological, for example. There is keen insight into the psychological dynamics that are at the root of human diversity, yet an understanding that such diversity appears to be regulated by a common human heritage.

Just as the self becomes viewed more from the perspective of historical patterns and general emotional transformation, so do the parents. Younger individuals and those over the age of 60 primarily describe their parents in the interpersonal context of their roles as providers of emotional and financial support to the self, or, to a lesser extent, in the institutional context of their societal position. Few youthful individuals represent their parents as more autonomous individuals in their own right. In contrast, around midlife there is a peak of responses that are appreciative of the unique individuality of parents: Participants describe their parents not just as carriers of parental and other social roles, but show an awareness of the conditions that shaped the parents and made them become the persons they are or were.

These results are consistent with views which suggest that a restructuring of representations of one's parents are part and parcel of the reorganizations in self often associated with middle adulthood (Jung, 1933; Kohut, 1977;

**Table 1** Levels of self- and other-differentiation.

<i>Level</i>	<i>Description</i>	<i>Example<sup>a</sup></i>
0 Concrete/ presystemic	The language used is simple and concrete. Characteristics and physical traits are seen as global. Events are detailed in simple seriation. Action-oriented behaviors describe activities. No references to goals or psychological processes occur.	I am nice. My dad is very nice because he buys me cool stuff. She is the nicest mom in the world. I'm tall.
1 Interpersonal/ protosystemic	Simple evaluations are made that reflect the values of the immediate social group. Traits described are nondifferentiated. Individuals are described in terms of relationships (simple descriptors) and social networks. An emphasis is placed on features of the self or others that make for in-group acceptance.	I like to fool around in class and make my friends laugh. My mom is young that is why she understands when I get into trouble. My father is very protective of me, and he cares about me.
2 Institutional/ intrasystemic	Interpersonal descriptors indicate a clearer sense of the individual within the social group. Traits at this level indicate a more self-directed and goal-directed individual whose evaluations are guided by achievement-oriented and conventional goals, values, and roles. Achievement of these goals and values is a frequent theme.	I am an empathic and committed friend. I work hard to support my children and really love them. A caring person, she will give her last dime to her children or anyone else she thinks is in need or something. He was a strict disciplinarian, bit of a drinking problem but never missed a days work.
3 Contextual/ intrasystemic	Descriptions are critical of convention, involve an awareness of how traits change, and give a sense of individuals with their own value system. Institutional goals are re-examined and put into historical or psychological perspective. Descriptions involve references to processes and contrasts over time.	I am a singer, an actress and a writer and want to use these talents more creatively than I do now. Accumulates material things and educational & professional credentials to prop herself up. At 83 my father is slowing down quite a bit and is not the same as he was most of his life.
4 Dynamic/ intersubjective	Roles and traits are described at a complex psychological level and reflect awareness of underlying, often unconscious, motivation and reciprocal interaction. Activities and goals are seen as subject to continual revision as one gains knowledge of oneself and others. Reference is to multiple dimensions of life history, and an emphasis on process, becoming, and emergence.	I work for profit now rather than for satisfaction, partly because of my (guilty) need to continue to support my family. A fine balance of strength & vulnerability in may aspects, however a marked inability to discern male competence and motive.

<sup>a</sup> Errors in grammar and spelling reflect actual responses.

Labouvie-Vief, 1994; Levinson, Darrow, Klein, Levinson, & McKee, 1978). Overall, however, it is also notable that relatively few individuals display the higher levels of parent representations. This finding that higher levels of representation are quite rare in the population and that they tend to be concentrated in the middle age period is also replicated by many other studies (see Labouvie-Vief, Chiodo et al., 1995).

#### 1.09.3.3.4 Emotional development

Advances in cognitive complexity, such as the ones discussed, also have implications for the understanding and regulation of emotions (Labouvie-Vief, DeVoe, & Bulka, 1989). Following suggestions by Piaget (1980), a number of researchers have shown that as individuals re-evaluate and reinterpret aspects of reality, their understanding of the nature and causes of

emotions changes as well. For example, from childhood to adolescence, several changes in emotional understanding and regulation are apparent (see Fisher & Ayoub, 1994; Harter & Monsour, 1992; Labouvie-Vief, *in press*; Labouvie-Vief, Hakim-Larson, DeVoe, & Schoeberlein, 1989). First, the emotional vocabulary becomes more differentiated, referring increasingly to complex blends of emotions and to emotions of contrasting valence. Second, the standards by which emotions are regulated become more abstract as the need for direct dyadic supervision is replaced by more internal and normative controls. Third, emotions also become understood as more internal processes and are described in terms of thoughts, wishes, and rules rather than direct physical actions and consequences.

Important as these skills are in the process of early socialization, they are particularly significant in the context of development at early life stages when individuals need to define their emotional lives in accord with external criteria: they begin to master a set of rules that permit them to regulate behavior in accordance with cultural dictates. In contrast to this outward movement, adulthood may bring a compensatory movement inward. A focus on inner dynamics, on private experience, and on rich organismic experience and emotive content now comes to the fore, a process Gutmann (1987) refers to as the "greening" of the mature individual.

This notion was supported in research by Labouvie-Vief and colleagues (Labouvie-Vief, DeVoe, & Bulka, 1989), showing that from adolescence to middle adulthood, emotional understanding and regulation show systematic differences. The language of emotions of younger individuals primarily emphasized their inner and mental nature. It was static; terms were used as descriptors and emphasized how one *should* feel; external rules and standards of conduct rather than the felt experience characterized individuals' expression of emotions. In contrast, mature adults—those around middle adulthood—gave evidence of a significantly reorganized emotional language. Feelings were described in terms of a vivid felt process, dealing not with static states, but with process and transformation. At the same time, individuals began to differentiate an inner realm of emotional experience from an outer realm of convention. The conflict between these realms was acknowledged, and the individual was concerned with accepting impulses and thoughts that previously seemed too overwhelming to accept.

This research suggests that in individuals who are older/more mature, emotions are repre-

sented in less dualistic and polarized ways. This conclusion is also supported by research of Blanchard-Fields (e.g., 1986; Blanchard-Fields, 1996) who showed that older adults' thinking about emotional conflicts is better integrated. For example, older individuals are better able to differentiate their (emotionally influenced) interpretations from a body of data they evaluate; they also are less likely to view others' behavior in static terms, but more likely to explain it in terms of contextual factors.

Evidence such as this may suggest that older adults may become experts at dealing with emotionally relevant information, a conclusion that is congruent with research examining how individuals of different ages interpret narratives such as stories that have a highly emotional and symbolic content (Adams, 1986; Adams, Labouvie-Vief, Hobart, & Dorosz, 1990; Jepson & Labouvie-Vief, 1992). That research suggests that adolescents and college students in reading text primarily focus on literal features, as they attend to the structure of actions and events depicted in the text. However, for mature and older adults the primary interest is not in this literal action-event structure, but rather in what it reveals about underlying emotional and motivational patterns of the human condition. To that extent, the mature adults' interest in text becomes more abstract and symbolic: A narrative does not refer to the concrete here and now of protagonists and their actions, but rather is taken as indicative of human actions in general (see also Jepson & Labouvie-Vief, 1992).

It is important to note that this focus on emotions is not always the result of genuine integration. For example, later adulthood may bring a more general bias for emotion-based responses, as noted by Blanchard-Fields (1994) and Carstensen (1992; Carstensen & Turk-Charles, 1994). In the research by Labouvie-Vief, and in Adams' work too, it was noted that the psychologizing approach appeared to occur with high frequency in older individuals. However, it did so in styles that differed in complexity and elaboration. Some adults would give summaries that involved complex back-and-forth references between the text base and some psychological process they thought the text symbolized; in fact, in Adams' (1986) study, such responses peaked in middle-aged adults. Other responses were rather global and undifferentiated, with affectively laden responses such as "The trouble is that nobody believes in God anymore," and it was these responses that were most frequent in the older group.

The observation that emotion-based language sometimes is rather global and undifferentiated suggests that even though older adults may use a more emotion-based language, this

emotional language may not necessarily imply the ability to reason about emotions in complex and coordinated ways. Thus other mechanisms than emotional understanding may play a role. For example, Brandstädter (e.g., Brandstädter & Greve, 1994) and Carstensen (1996) both suggest that awareness of time and mortality are a major factor by which an increasing acceptance of emotions is ushered in, even though this awareness may be stimulated by events, such as illness, that are not related to age. However, although such realization may effect a switch in the preferred processing system as implied by Carstensen's work, such a simple switch must be distinguished from the kinds of processes of cognitive-affective integration we are concerned with. Further, it is also possible that certain cognitive restrictions, particularly in the domain of fluid intelligence, may limit some older individuals' ability to modulate complex affects (see Labouvie-Vief, Chiodo et al., 1995, Labouvie-Vief & Diehl, 1996; Labouvie-Vief, Diehl et al, 1995). Thus conceivably, an increase in emotion-based language might reflect *less* complex emotion-regulation strategies.

These findings on emotion language may be related to research on coping and defense processes. A number of studies (for review see Aldwin, 1994; Diehl, Coyle, & Labouvie-Vief, 1996) on coping/defense processes across the life-span have demonstrated that older individuals, when compared to younger age groups, show a pattern of more positive coping and less defensive coping. This pattern continues at least into the sixth decade. For example, in the research by Diehl and collaborators, older individuals scored lower

One current tendency in the literature is to propose that such findings indicate a flexible coping strategy by which older individuals adjust to the realities of later life by the adoption of "secondary" or "emotion-focused" coping strategies (see Blanchard-Fields, 1996; Brandstädter & Greve, 1994; Carstensen, 1996; Schultz, 1996). However, it is not clear at this time whether coping strategies actually may represent a mixture of strategies. For example, data of the Diehl et al. (1996) study suggest that secondary strategies of meaning transformation (e.g., through the rescaling of goals and aspirations) may not invariably imply genuine transformation of affect, but also can indicate its denial or repression. Thus it is possible that some individuals maintain positive adaptation and self-organization under conditions of relatively high-level functioning; however, others may rely on coping strategies that involve denial, repression, and gating out of disturbing affect. Even though such more fragmenting

techniques may serve to maintain emotional equilibrium in the face of declining resources, they may do so at the cost of an overall picture of more norm dependence and rigidity.

### 1.09.3.3.5 Wisdom

All of the above proposals indicate that rational processes on the one hand, and processes related to self and emotions on the other, are profoundly interconnected. Even though this is true of all of the life-span, it appears to be only at relatively advanced levels that individuals are able to represent and understand these relationships and integrate them within single, nonconflicting systems. This ability to bridge the tensions between the universal and the contextual, the theoretical and the pragmatic, and the rational and emotional is often referred to as wisdom (Baltes & Staudinger, 1993; Clayton & Birren, 1980; Labouvie-Vief, 1990; Staudinger & Baltes, 1994, 1996).

While all of the work discussed above related to processes of wisdom (see Sternberg, 1990), the most detailed research project thus far has attempted to provide a specific operational definition of "wisdom." According to Baltes and his colleagues (Baltes, Smith, & Staudinger, 1992; Baltes & Staudinger, 1993), wisdom, defined as expert knowledge with regard to important but uncertain matters of life, can be characterized by several components as outlined in Table 2. Individuals receive ratings on each of these components, and an overall "wisdom score" is derived. As is true of the studies reported in this section, Baltes and his collaborators found that although age in itself was not a sufficient condition for the development of wisdom, older adults were more likely to be nominated as wise, and those nominated in fact produced the highest wisdom scores. Baltes et al. (1992) also found that personality measures, in addition to standard tests of crystallized and fluid intelligence, predicted wisdom scores, underscoring the interface between personality and cognition (see also Staudinger & Baltes, 1996).

## 1.09.4 PERSONALITY DEVELOPMENT

As Riegel (1977) noted in his work on the history of life-span development, the area of personality and social relations was a relative newcomer to the field. Early theorists such as Freud had proposed that personality was set rather early, and that it was unlikely to change (except in the direction of regression) in the second half of life. In contrast, the post-World

**Table 2** Use of the wisdom-related criteria to evaluate discourse about life matters.

<i>Criterion</i>	<i>Instantiations</i>
Factual knowledge	Who, when, where? Examples of possible different situations Multiple options (forms of love and marriage)
Procedural knowledge	Strategies of information search, decision-making, and advice giving Timing of advice Monitoring of emotional reactions Cost–benefit analysis: scenarios Means–ends analysis
Life-span contextualism	Age-graded contexts (e.g., issues of adolescence) Culturally graded contexts (e.g., change in norms) Idiosyncratic contexts across time and life domains (e.g., terminal illness) Interrelations, tensions, priorities of life domains
Relativism	Religious and personal preferences Current vs. future values Historical period Cultural relativism
Uncertainty	No perfect solution Optimization of gains vs. loss Future not fully predictable Backup solutions
<hr/>	
	Examples of responses (abbreviated)
Low score	A 15-year-old girl wants to get married? No, no way. Marrying at age 15 would be utterly wrong. One has to tell the girl that marriage is not possible. [After further probing] It would be irresponsible to support such an idea. No, this is just a crazy idea.
High score	Well, on the surface, this seems like an easy problem. On average, marriage for 15-year-old girls is not a good thing. I guess many girls might think about it when they fall in love for the first time. And, then, there are situations where the average case does not fit. Perhaps in this instance, special life circumstances are involved, such that the girl has a terminal illness. Or this girl may not be from this country. Perhaps she lives in another country and historical period. Before I offer a final evaluation I would need more information.

"The search for a psychology of wisdom," by P. B. Baltes and V. M. Staudinger, 1993, *Current Directions in Psychological Science*, 2, p. 78. Copyright 1993 by the American Psychological Society. Reprinted with permission.

War II era has seen a dramatic rise in inquiries into change and stability in personality and self in adulthood and later life.

The question about change and stability in personality will be answered according to the investigators' theoretical position (Block, 1995a, 1995b; Costa & McCrae, 1980; Helson, 1993; McCrae & Costa, 1990) as to how personality has been defined and measured, and on how stability and change have been assessed. To date, two major theoretical approaches have dominated the field of personality psychology and have influenced research on personality development. Developmental level approaches have been strongly influenced by the psychodynamic tradition. This tradition historically was the first to elaborate on personality organization, and has proposed a

set of rich and detailed hypotheses about developmental stages of life, emphasizing qualitative changes in personality adaptation at different life periods. The trait-theoretical approach, in contrast, has been much more methodologically oriented, and emphasized the basic continuity and stability of personality across the life-span. However, both approaches in recent years have begun to draw on each other, the psychodynamic tradition providing important theoretical questions, and the psychometric tradition offering a strong set of methods to examine some of these questions. Both of these approaches, moreover, have begun to embrace contextual propositions that more and more explore the ways in which self and personality respond to the unique settings in which individuals develop and age.

#### 1.09.4.1 Developmental Level Approaches

Level approaches to adult personality are strongly influenced by psychoanalytic thinking, along with its more recent expansions into object relations and self theory (see Greenberg & Mitchell, 1983). Psychoanalysis itself reflected a significant movement in redefinitions of mind and self, pointing out that emotions and relationships with others are not derived processes, but the very ground on which cognition is built. Psychoanalysis thus followed a broad trend to take the study of the mind out of the transcendent and to make it part of the natural world.

##### 1.09.4.1.1 Freud's legacy

Freud's theory pioneered the proposition that rational processes are built on a layer of desires and needs or instincts. Yet paradoxically, even though he helped link back the mind with natural processes, Freud—along with many other theoreticians, such as Piaget (see previous section)—retained an objectivist bias, since as in traditional philosophy, the superiority of rational secondary processes over the “irrational” functioning of the primary processes was maintained. Thus development in large part becomes a matter of the victory of conscious, ego-oriented thought, while thought related to the emotion- and symbol-based secondary process was considered of less value.

This dualism is reflected in the terms Freud chose to refer to the psychic structures that were primarily related to each of these thought processes: the id and ego, respectively. It is interesting here to reflect on the historical origin of these terms. As Bettelheim (1983) pointed out, when Freud's work was translated from the original German into English, the meaning of these terms were significantly changed, as well. Freud's translators chose such abstract, Latin, scientific sounding words as “id” and “ego,” in contrast to Freud's choice of such simple, common language words “I” (the German *Ich*) for ego, and “it” (The German *es*) for “id.” The word “I,” of course, indicates an immediate, direct personal identification with part of ourselves. And it is interesting that for Freud, that direct place of self-identification is the structure that mediates decision making, planning, and conscious thought. Thus, the self is, in effect, equated with logos functioning. This is where we are assumed to feel ourselves most “at home,” where our sense of reality is most secure. The word “it” in contrast connotes a distanced, rejecting, and critical attitude toward that other part of ourselves.

This devaluation of nonrational processes makes Freud's theory unsuited to deal with many issues important across the life-span. To begin with, the theory is individualistic, since it puts values of independence, good ego boundaries, and so forth at the center (see Gilligan, 1982; Labouvie-Vief, 1994). The focus is on the separate individual, even from the start: desires and instincts are processes first located in the individual and then cathected onto another (Greenberg & Mitchell, 1983). That other is not treated as a separate self, but merely as a projection screen for one's own wishes. This lack of genuine relatedness is particularly important in the area of gender relations and gender development, since masculinity is favored sexually as a primary, more valuable state. By metaphorically extending notions of the superiority of the phallic principle into the mental domain, ego, consciousness, moral restraint are lined up with masculinity and maturity, while the feminine is considered a less developed state. As a result, and following millennia of historical tradition (Labouvie-Vief, 1994), in Freud's theory women are not really represented as being different from the self, as having an independent existence, their own desires, their own subjectivity. Rather, they are merely represented as others, beings inferior to men, thus making them objects to the male action, mind and gaze (see Benjamin, 1988; Labouvie-Vief, 1994).

Another important limit of Freud's theory is that it does not deal with forms of thinking more closely related to the primary process domain in a progressive and positive fashion. For example, religious processes, group processes, and love all are treated as a regressive return to infantile wishes for merging. Such wishes are to be suspended from the rule of ego-oriented thought. Thus, Freud's theory does not distinguish between mature and immature forms of these feelings.

Finally, Freud does not account for cultural construction of this hierarchical model. He assumes it is inherent in universal processes of gender dimorphism and ego formation rather than a cultural model. Ego and superego structures thus are viewed as stable structures that do not require further change. Indeed, further change tends to be pathologized as “regressive”; and Freud believed that psychotherapy cannot be successful in the second half of life.

##### 1.09.4.1.2 Beyond the ego

Some of these limitations of Freud's theory were addressed by two individuals who themselves were intimately familiar with Freud's

ideas. The first, Carl Gustav Jung, was one of Freud's collaborators and his hoped-for successor. When he began work with Freud, Jung had already done important work on the nature of the unconscious. However, as he continued to work with Freud, his position gradually changed from that of his mentor, and eventually he broke from Freud in a bitter confrontation that has become famous.

Jung was critical of several basic tenets in Freud's theory. He believed that Freud had too negative a view of the nature of the unconscious, and that he put too much emphasis on the importance of ego control. Instead, Jung believed that the basic organizing principle, though it included the ego, was not primarily conscious but rather unconscious. Jung termed this general organizing principle the self, anticipating many modern efforts to delineate the nature of the self.

Jung also believed that Freud's view of the unconscious was too much based on the individual's personal experience: the unconscious in Freud's theory consisted mostly of contents that have become repressed in the individual's personal experience. Jung argued that this interpretation did not allow positive accounts of an individual's symbolic life as it is expressed in religion, art, dreams, and so forth. He believed that the unconscious also contained many experiences that are part of our heritage as a species, and that express important emotional experiences related to love and hate, sexuality and the generational flow. These experiences are expressed in the form of story telling and myth, ritual and visual symbol (see Labouvie-Vief, 1994).

Jung also argued that one result of Freud's idealization of the rational was an overvaluation of the masculine and a devaluation of the feminine. Unlike Freud's theory, which held that healthy development requires that men suppress the feminine and women the masculine, Jung believed that successful development requires that the person integrate the two. Thus he anticipated much recent research on the nature of androgyny.

As a result of these conceptual shifts, Jung and some of his students suggested that Freud's view of development was more pertinent for the first part of life, when in the interest of cultural adaptation and socialization the individual must suppress aspects of the self, including contrasexual tendencies. All of these movements (together with the cognitive limitations discussed in the previous section) encouraged the formation of fairly rigid dualisms between mind and body, thinking and emotion, conscious and unconscious, outer and inner, good and evil, masculine and feminine, and so forth.

In contrast to the socializing emphasis of early life, Jung (1933) suggested that around the middle of life, development shifts to individuation. Individuals now have an opportunity to accept unconscious motives, to integrate contrasexual aspects and tendencies, and in general to integrate the dualisms formed in early development. In this way, they begin to transcend a narrow self-identification with ego and superego and become guided by a more genuine dialectic relationship between ego processes and the deeper patterns of human experience expressed by the self. Jung felt that these patterns indicate deep longings and desires that are often unconscious. Yet they are expressed in stories and symbols worldwide, and they express universal aspects of the human experience. These midlife tasks, Jung believed, often were resolved in a crisis. Jung in fact coined the term "midlife crisis," and he suggested that such a crisis was a necessary and positive aspect of later life development. He thought that as a result of that crisis, individuals were able to experience an upsurge of creativity and well-being.

#### *1.09.4.1.3 Ego and culture*

Like Jung, Erik Erikson (1982) felt that Freud's model of development was framed too narrowly, resulting in an overemphasis on autonomy, a failure to account for the diverse role of culture, and a regressive view of the role of religion and spirituality in the lives of individuals. One of the important steps Erikson took was to expand Freud's psychosexual view of development to a psychosocial one. He did so by first rooting the development of autonomy in the basic fact of human relatedness. Second, he generalized modes of relating beyond adolescence to mature adulthood and later life, where individuals were better able to give up narrow self-concerns about their own survival and participate in the caring for and management of culture.

One significant feature of Erikson's theory is that it implicitly proposes a generalization of the Freudian notion of "identification." For Freud, processes of identification took place in the "phallic" period as a result of the internalization of parental attributes and rules. In contrast, Erikson proposes that the self defines itself through successively widening circles of relations. Thus, at mature adult levels, the self becomes invested in culture and even humankind and the human condition, in general.

Another significant feature of Erikson's theory is that he assumed that each stage of human development was defined by a genuine

dialectical tension (he called this tension "crisis") between the opposing tendencies of autonomy and relatedness. Thus, the function of the first important relation, child–mother, is to build a secure base of trust, a foundation from which later autonomy could be built. However, each stage is defined in terms of the degree to which these oppositions can be integrated, and failures to achieve complete integration are the norm rather than exceptions. Thus, most individuals are able to develop a core self-sector that permits the experience of trust and hope, yet also may retain more problematic self-sectors in which basic mistrust and withdrawal prevail. At successive later levels, these core qualities evolve into broader, more generalized ones.

One of the core crises of adulthood, that of generativity vs. stagnation, revolves around the task of surrendering the youthful need for self-idealization (Gutmann, 1994). As individuals grow older they usually are confronted with the limits of their creativity, their physical beauty, or their sexual attractiveness. Finding these sources of admiration and of pleasure dwindling, individuals may respond with an increased need for admiration and self-affirmation. Yet they also are able to step back from this need for admiration and, in experiencing its painful hold on the self, let go of it and transmute it into a broader understanding of the human situation. And, out of this experience of loss, the individual can recover a sense of admiring and idealizing others. Altruistic forms of passing on one's knowledge or financial resources, such as in forms of mentoring or philanthropy, are a good example of such admiration of others. Ideally, such generative support of others is no longer subordinated to one's own need to find advancement of admiration (see also McAdams, 1994).

While according to Erikson the experience of generativity is necessary to ward off a pervasive sense of stagnation in middle adulthood, true generativity may not come easily to most adults. Ultimately, the breadth and depth of generative concerns also are related to the ability to face and integrate negative experience (Erikson, 1982). As Jung (1933; see also Whitmont, 1969) in particular has pointed out, in growing up individuals usually learn to split off negative aspects of reality and idealize its positive side. Yet mature adulthood is the time when these neglected aspects need to be integrated to allow a more balanced view of reality. To face ward off negative experience, yet not be defeated by it, is a difficult challenge that not all individuals face successfully. Accepting destructive impulses in oneself and others—confronting hate, envy, evil and

corruption—can be devastating; yet moving to maturity requires that we be able to do so without exploitation or masochism.

Many individuals retreat from that crisis. Why? Erikson thinks that ultimately, this is rooted in our need to defend ourselves, our inability to trust. Jaques (1965) also suggests that the midlife re-examination is not necessarily resolved successfully by all. Instead, adults may retreat from the crisis:

the compulsive attempts, in many men and women reaching middle age, to remain young, the hypochondriacal concern over health and appearance, the emergence of sexual promiscuity in order to prove youth and potency, the hollowness and lack of genuine enjoyment of life ... are familiar patterns. These are attempts at a race against time. And in addition to the impoverishment of emotional life contained in the foregoing activities, real character deterioration is always possible ... Increase in arrogance, and ruthlessness concealing pangs of envy—or self-effacing humbleness and weakness concealing fantasies of omnipotence—are symptomatic of such change ... These defensive fantasies are equally as persecuting, however, as the chaotic and hopeless internal situation they are meant to mitigate. (Jaques, 1965, p. 511)

Erikson also suggested that after the crisis of generativity vs. stagnation, the individual faces a last crisis, that of integrity vs. despair. At the close of life, individuals need to accept their own life cycle and the people in it as something that had to be and that permitted of no substitutions. Alternatively, individuals may not be able to reach such acceptance. Rather than accepting life as their own responsibility, they may feel that time is too short, that alternate roads are blocked; they may harbor feelings of disgust, misanthropy, and a chronic displeasure with particular institutions and particular people.

#### *1.09.4.1.4 The duality of the self*

Although both Jung and Erikson pushed the boundaries of Freud's theory, many recent criticisms suggest that one of their core concepts—that of the basic duality of the self—was not sufficiently developed, and both maintained a bias towards autonomy, individuality, and masculinity. That concept of duality should be seen as the full conjoining of two fundamental developmental lines which Bakan (1966) called agency versus communion and others have called an orientation to separateness versus connection (e.g., Gilligan, 1982; Miller, 1976), power and achievement versus intimacy and love (McAdams, 1985), or individuation and attachment (Franz & White,



1985). These orientations influence experiential forms of motivation, adaptation, and strategies of coping and defense. In mature development, these lines are integrated in configurations that include a consolidated self which combines autonomy and productivity with healthy relatedness.

One example of such a theory is Kohut's theory of the self (Kohut, 1977). Development, according to Kohut involves the dialectic of two tendencies, one related to our need to be admired by others, the other the need to admire others. The first tendency is related to narcissism/grandiosity, which in their immature forms reflect a defensively inflated sense of self in excessive need of admiration. Yet in its more positive forms, it gives rise to mature creativity. On the other hand, the ability to renounce one's narcissism gives rise to the ability to mirror and idealize others, a core ingredient of one's ability to love. These tendencies may occur in balanced relationship; but in less ideal cases, one may be inflated at the expense of the other. Kohut also thinks that midlife can be crisis-like, as individuals realize that early adaptations are no longer useful.

Many scholars have noted, however, that each tendency is maladaptive when present in exaggerated or unmitigated forms (e.g., Bakan, 1966). Thus when normal development is disrupted, distorted modes of adaptation occur along one developmental line or another. For example, Benjamin (1988) suggests that one immature form of these two lines occurs in the interlocking of roles of domination and submission, where the dominator combines self-inflation with the contempt and maltreatment of the other, while the victim or subordinate surrenders healthy narcissistic needs and develops excessive needs for mirroring. Specific psychopathologies associated with each form have been outlined by Blatt and Shichman (1983). The self-defining, "introjective" axis involves a complex of affects and behaviors associated with excessive concerns about identity, self-control, and self-worth. The introjective character uses counteractive defense mechanisms to manage aggression directed toward the self and others, and to fend off experiences of loss of individuation. The interpersonal or "anaclitic" axis involves a constellation of issues concerned with disrupted relatedness and intimacy. Avoidant defenses (denial, repression) are used against feelings of loneliness and alienation. Anaclitic pathologies involve issues of dependency, unity, and belonging.

Theoretical frameworks such as these have become extremely important in theories of development. One theoretical framework that

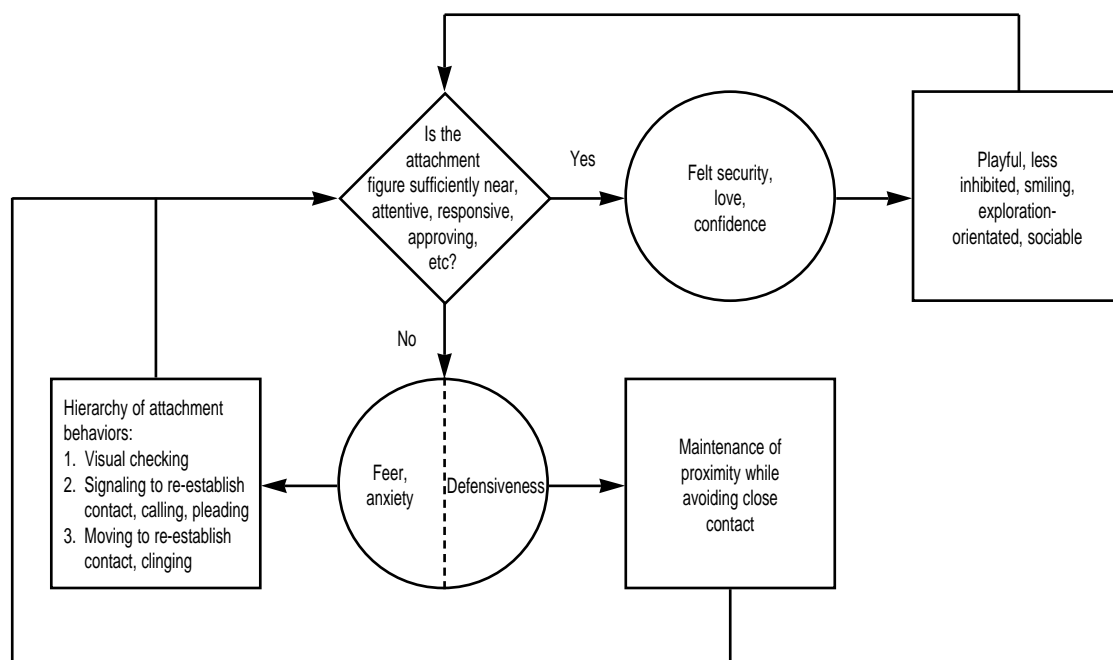
has been highly successful in working with this polarity is attachment theory as developed by Bowlby. Attachment theory also is an outgrowth of psychoanalytic theory, but was reworked by Bowlby into an ethological theory including principles of control theory. Attachment theory is specifically built on a conjoining of needs for autonomy and attachment, as shown in Figure 4 (Hazan & Shaver, 1994). Ideally, individuals are able to shift from exploration and the development of autonomy to security- and closeness-seeking behaviors. However, if early relationships do not encourage flexible shifting between both tendencies, individuals are likely to develop different forms of less adaptive attachment styles. Further, these styles are assumed to have a stable influence throughout the life course.

#### **1.09.4.2 The Trait Approach**

On the surface, the trait or individual differences approach to personality and personality development appears not ideally suited for the study of change and transformation across the life-span (Allport, 1937; Cattell, 1979; Costa & McCrae, 1994; Eysenck, 1982; McCrae & Costa, 1990). Trait theorists believe that an individual's personality can be defined as a limited number of basic behavioral tendencies that the individual has and consistently displays. These basic tendencies may be inherited or acquired and may or may not be malleable over the course of development. Behavioral tendencies and temperament traits that are usually examined by trait theorists include extraversion/introversion, emotional lability (i.e., neuroticism), shyness, conscientiousness, rigidity/flexibility, hostility, and others. Trait theorists assume that over the course of the life-span, these basic tendencies interact with external influences to produce characteristic adaptations which show a high degree of stability (Costa & McCrae, 1994; McCrae & Costa, 1990). An important consequence of this assumption of stability/continuity is that the structure of individuals' personality can be assessed using self-report questionnaires or objective ratings from others.

##### **1.09.4.2.1 Traits as dimensions of stability**

Historically, the trait-theoretical approach has centered around three major questions: How many traits are there?; How many traits are necessary to describe individuals' personality in a comprehensive manner?; and How stable are personality traits across the human life-span? In order to provide an answer to the



**Figure 4** The Attachment Behavioral System. "Attachment as an organizational framework for research on close relationships." by C. Hazan, & P. R. Shaver, 1994, *Psychological Inquiry*, 5, p. 3. Copyright 1994 by Lawrence Erlbaum Associates. Reprinted with permission.

first question, Allport and Odbert (1936) argued that all trait descriptions should basically be contained in common language, a proposition that was echoed by Cattell (1943a, 1943b), and resulted in the lexical strategy to personality assessment (Allport & Odbert, 1936). Other researchers (Cattell, 1947; Goldberg, 1981; Norman, 1963; Tupes & Christal, 1961/1992) built on Allport and Odbert's (1936) initial work and derived lists of trait names (for a detailed description, see Block, 1995a) which they analyzed in a variety of ways. In particular, the use of the mathematical technique of factor analysis resulted in various taxonomies of personality factors, ranging from 5 to 16 factors, that were considered sufficient for the assessment and description of personality. These endeavors based on the lexical approach were also paralleled by endeavors to construct questionnaires for the assessment of personality structures. Specifically, Costa and McCrae's (1992a) development of, and research with, the NEO and the NEO-PI-R has increasingly created the impression that five general personality factors (i.e., Neuroticism—N, Extraversion—E, Openness to Experience—O, Agreeableness—A, and Conscientiousness—C) describe individuals' personalities in a comprehensive fashion. Moreover, Costa and McCrae (1988, 1994) have provided consider-

able evidence showing that these five personality factors display a great deal of stability across the adult life-span.

Recent critics of the "Big Five" model of personality (see Block, 1995a, 1995b), however, have pointed out several limitations of this approach. First, Block has meticulously documented inconsistencies and subjectivity in the use of the lexical approach by different researchers (Block, 1995a). Second, Block has also questioned the inconsistent and often very subjective use of factor analysis, specific rotation methods, and the labeling of the derived factors to describe particular factor structures (Block, 1995a). Taken together, Block (1995b) has drawn two major conclusions based on his review of the relevant literature. First, he concluded that from the beginning the trait perspective has been inherently biased in its measurement approach toward the stability of traits, thus neglecting contextual influences and age-related factors that may contribute to variability in personality development. Second, he also concluded that advocates of the "Big Five" factor model present conclusions in support of the five-factor approach (FFA) far stronger than seems to be warranted (Block, 1995b). In contrast to the proponents of the FFA, Block (1995a, 1995b) has argued in favor of a perspective in personality research that incorporates both the

sociocultural context in which personality development occurs and the notion of age-period specific variability across individuals' life course (see also Caspi, 1987). Thus, Block (1995a, 1995b) has argued in favor of a perspective that focuses on change as well as on stability in personality development. Block's position and concerns regarding the FFA have been shared by other personality theorists (Emmons, 1995; McAdams, 1992). Winter (1996), for example, has come to the conclusion that although "the five-factor theory is a promising development, it is hardly the final word even in trait psychology" (p. 471).

#### **1.09.4.2.2 Context as moderator of stability**

The contextualist orientation to personality and personality development rests on several basic assumptions. First, contextualists usually focus on subtypes of persons or personality syndromes and compare the developmental patterns of the identified subtypes over time, because they assume that personality development is *a priori* characterized by interindividual differences in the intra-individual continuity of personality characteristics (Block, 1981; Caspi, 1987). Second, contextualists place great emphasis on studying personality "the long way" (Block, 1993) in order to examine the effects of historical events (e.g., Great Depression, World War II, Vietnam War) and social changes (e.g., women's liberation movement, changes in child-rearing practices) as well as the effects of individual life events (e.g., parenthood, change in occupation, divorce) on individuals' personality development (Caspi, Bem, & Elder, 1989). Third, contextualists study personality from a transactional perspective (Magnusson & Törstedt, 1993). That is, they examine how the motivations, goals and resources of individuals interact with the surrounding social world; and when and why the organization of motivations and resources changes as a consequence of changes in the social world (Helson, 1993). In summary, the contextual approach to personality development situates individuals in their social world and examines how the reciprocal interactions between person and social context affect the emergence, maintenance, and transformation of distinct personality characteristics. Thus, although contextualists are, to some extent, interested in questions of stability and change of personality across the life course, their primary focus concerns the issues of differential change patterns for different personality types. The assumption that both stability and change are part of personality development across the life-span is central to this perspective.

### **1.09.4.3 Empirical Research on Self and Personality**

#### **1.09.4.3.1 Stability and change**

The different perspectives in personality research differ greatly in their assumptions about stability and change. Researchers who subscribe to a trait perspective or any other kind of stability-oriented model (i.e., the attachment framework; Bowlby, 1988) assume a great amount of stability, whereas researchers who subscribe to a contextual perspective are more inclined to expect changes in personality. Given this controversial situation, a careful review of the respective literatures seems to be warranted.

What do studies in the individual difference tradition tell us about continuity or change in personality development? As Conley (1984) has pointed out, some of the most impressive evidence with regard to the longitudinal consistency of personality comes from studies of individual differences in children's emotional health. For example, ratings of children's emotional health have shown moderate consistencies over long time periods (Cox, 1970; Symonds, 1961; Vaillant, 1974) and have shown good predictive validity with regard to mental health outcomes (Johns, Mednick, & Shulsinger, 1982). Moreover, the temperamental qualities of three year olds have shown considerable predictive validity with regard to personality traits in young adulthood (Caspi & Silva, 1995; Kagan & Moss, 1962; Thomas & Chess, 1986).

These findings are complemented by research on personality development in adulthood. Using data from the Kelly Longitudinal Study, Conley (1984) showed for men and women moderate mean correlations for neuroticism indicators and for indicators of social introversion-extraversion across a 45-year period (see also Leon, Gillum, Gillum, & Gouze, 1979). In a subsequent study, Conley (1985) found that individual differences on the traits neuroticism, social extraversion, and impulse control were rather stable over a period of 20 years even when measured via different methods. Other longitudinal studies that have documented a great deal of stability in personality functioning are the Duke University Studies of Normal Aging (Siegler, George, & Okun, 1979), the Bonn Longitudinal Study of Aging (Schmitz-Scherzer & Thomae, 1983), and the Seattle Longitudinal Study (Schaie, 1996; Schaie & Willis, 1991). Although different measures of personality functioning were used in these studies, all three research programs showed remarkable stability (i.e., very limited change over time) in participants' self-reported personality descriptions.

The most vocal advocates of personality stability across the adult life-span have been Costa and McCrae (1980, 1988; McCrae & Costa, 1990) who have argued that after the age of 30 individuals' personality is "set like plaster" (Costa & McCrae, 1994). Based on data from the Boston Normative Aging Study (NAS) and the Baltimore Longitudinal Study of Aging (BLSA), Costa and McCrae (1988, 1994) have provided a considerable evidence in support of their position. For example, they have reported moderate to high 30-year stability coefficients (Costa & McCrae, 1994). Moreover, since the early 1980s, Costa and McCrae (1992b, 1994) have systematically elaborated the five-factor model of personality (John, 1990) and have devised a self-report questionnaire, the NEO-PI-R (Costa & McCrae, 1992a), that assesses the "Big Five." In terms of mean level differences, Costa and McCrae (1992a) have shown in cross-sectional studies that the "Big Five" show a fairly consistent pattern of age differences across the adult life-span: small declines are found for N, E, and O, whereas A and C show small increases. Although cross-sectional studies cannot rule out that the observed age differences reflect generational rather than maturational processes, findings from a six-year longitudinal study of self-reports and spouse ratings on the NEO-PI (Costa & McCrae, 1988) showed a longitudinal decline in self-reported N, but also a longitudinal increase in spouse-rated N. No significant time-related changes were found for either E or O. The most consistent longitudinal change found in adults over age 30 has been a decline in activity level (Costa & McCrae, 1988; Douglas & Arenberg, 1978; Field & Millsap, 1991).

Although this review of the literature shows that adult personality can be characterized by a fair amount of stability, high retest correlations by no means preclude the potential for change. For example, it is important to note that, in general, the test-retest correlations account for only 35–50% of the variance, leaving half of the variance unaccounted for. Moreover, this unaccounted variability is probably only in part due to lack of reliability of the measures, but is most likely related to true shifts in the ranking of people on the personality characteristic in question. Thus, overall it seems to be most appropriate to think about personality as an entity with relative stability depending on the life circumstances and the life experiences of each individual. We will return to this issue below.

#### 1.09.4.3.2 *Midlife crisis*

As suggested above, whether or not stability remains high depends on the life period under consideration. One of the life periods that in

particular has been associated with the potential for change and transformation is midlife. The notion that there are major reorganizations around midlife has been most strongly argued by theoreticians, most notably Jung (1932), Jaques (1965), and Levinson and collaborators (Levinson et al., 1978). All of these individuals have suggested that a reorganization of personality is a normative phenomenon, at least in an ideal sense. Thus, from a Jungian perspective, midlife is said to bring the opportunity to re-examine our constructions of good and bad, birth and death, dependence and independence, or masculinity and femininity; Erikson suggests a broadening of our ability to invest self in culture; and Jaques argues for a change in creative style from detailed and polarized to integrative and tragic. Further, Vaillant (1993) has suggested that around midlife there is a maturing of strategies of emotion regulation and defense (see also Labouvie-Vief, Hakim-Larson, & Hobart, 1987; Diehl, Coyle, & Labouvie-Vief, 1996).

Research related to the notion of the notion of a midlife crisis has not been extremely supportive of the notion, however. For example, there is little evidence that upheavals and dramatic change are related to a unique period of adulthood. Although some individuals do experience a crisis, those individuals may suffer from general problems of psychopathology (e.g., Rosenberg & Farrell, 1976). Thus, some researchers in the field have tended to conclude that the notion of a midlife reorganization has been overstated.

However, there is some fairly good evidence that many individuals experience some reorganization of self and values across the adult life-span. Why, then, should it be difficult to find clear evidence? There are two major reasons. One is the issue raised by Block (1995a; see also Labouvie-Vief, DeVoe, & Bulka, 1989) discussed earlier, that is the general bias in many current assessments towards stability. For example, our review of cognitive theories of adulthood suggests that the meaning individuals at different life-stages impart on test items may change quite profoundly with age or cognitive complexity. However, to the extent that tests rely on yes–no answers or simple scaling of agreement/disagreement with items, no room is left for different constructions of meaning.

This issue was raised by Labouvie-Vief et al. (1989) in the context of examining coping strategies across the life-span. While some researchers (e.g., Lazarus, 1991) have argued against generalized age differences in coping strategies, others (e.g., Diehl et al., 1996) have reported consistent patterns of strategies of

coping and defense. One reason for this ambiguity is that current codings of coping strategies confound strategies of different levels of complexity and cognition-affect integration within a single category. In earlier research on self-regulation strategies, we (Labouvie-Vief et al., 1987, 1989; see also Block, 1995a) already noted that the same strategy can have very different meanings depending upon the developmental level at which it appears. For example, turning to others may imply that individuals attempt to dissipate anxiety through aligning with others "in the same boat"—a less mature strategy—or that they seek out others in an attempt to consider alternative choices and raise their level of objectivity a more mature strategy.

Another major issue to be considered is that the major dynamic driving changes associated with midlife may not be so much period-dependent, but rather may follow general cognitive changes. We have already reviewed a considerable body of neo-Piagetian research that suggests that individuals around the middle of adulthood show the most complex understanding of self, emotions, motivations, in general, attributes, that fall under the rubric of "personality." At the same time, this literature suggests that more significantly than age, independent measures of cognitive complexity are the strongest predictors of higher levels of complexity. Thus, phenomena usually associated with a midlife crisis may be the result of general gains in cognitive complexity from early to middle adulthood.

Connections between adults' personality organization and their cognitive complexity have increasingly been emphasized by Labouvie-Vief and her colleagues (Labouvie-Vief, 1994; Labouvie-Vief, DeVoe, & Bulka, 1989). Labouvie-Vief et al. (1989), for example, found in a study with adults, that age, verbal ability, and level of ego development were systematically related to a more reflexive understanding of emotions and to the use of more flexible coping strategies. Similarly, in a study on adults' self-representations, Labouvie-Vief, Chiodo, et al. (1995a) showed that in older adults a combination of intellectual and personality variables were the significant predictors of participants' complexity of self-representations, whereas younger adults' complexity of self-representations was predicted by cognitive variables only. Taken together, these studies are the result of a more positive perspective on the aging self and represent first attempts to address the interfaces between intelligence and personality in later life.

While, in general, these data suggest that so-called midlife changes are the result of rather gradual transformations, somewhat more dra-

matic crises may, however, be experienced by individuals whose early adaptations turn out to be problematic, and who as a consequence may encounter a relatively sudden and dramatic need to regroup their adaptive efforts and to reorganize their life structures. Stewart (1996) recently has proposed such a contextual view. Thus she argues that for most of the women studied, midlife may be a time of re-examination of their lives. If life patterns are found wanting, these women may engage in a midlife adjustment, correcting their patterns by such changes as moving from family to work orientation. This group of women subsequently shows a positive turn in their patterns of adjustment. However, not all women who are dissatisfied with their lives apply such an adjustment, and failure to do so is related to negative consequences for their subsequent life course.

#### **1.09.5 CONTEXT IN PERSONALITY DEVELOPMENT**

As described earlier, contextualists are more inclined than trait theorists to assume changes in personality development and to examine stability and change in response to changes in context (see Bloom, 1964; Helson, 1993). Bloom (1964), for example, reviewed a number of studies of personality stability and also examined the stability of other variables, including physical characteristics and intellectual abilities. Based on this review, he postulated that the change gradient of most traits, when plotted against age, is a positively sloped function with decreasing acceleration. Specifically, he rejected the idea that personality is fully developed by early adulthood.

That stability and change are characteristic of personality development during adulthood has been shown by several studies. For example, using data which covered a time span of 50 years, Haan, Millsap, and Hartka (1986) found that consistency indexes were largest for adjacent age periods but were considerably smaller when examined over the entire time span. In addition, consistency coefficients varied by age period, suggesting that personality stability may be considerably reduced during periods of the life course that are characterized by extensive transitions and role changes (e.g., the transition to parenthood in young adulthood). A similar pattern of findings has been reported by Skolnick (1986) with regard to attachment relationships. In brief, Skolnick's study provided a modest degree of support for attachment theory, showing some significant correlations between early relationship experiences with the mother and later social behavior.

The most striking finding, however, was the variability of individual relationship careers across the life-span, indicating the "developmental openness" of early relationship experiences to influences in later parts of the life course.

That both stability and change characterize personality development even in advanced old age has been shown by Field and Millsap (1991) for the Berkeley Older Generation Study. Although Field and Millsap (1991) found moderate rank order stability for the traits Satisfaction, Extraversion, Agreeableness, and Intellect, they also showed significant mean level changes in some of these traits over a 14-year period as study participants grew into old age. For example, more than one-third of the participants increased significantly over time in Agreeableness, and both men and women and old-old and oldest-old participants declined significantly in Extraversion. Field and Millsap (1991) concluded that these findings do not support the common stereotype that personality "rigidifies" in old age.

Taken together, these studies contribute in two major ways to the literature on personality development. First, in combination with the long-term findings reported by Conley (1984, 1985), these results document that the consistency coefficients tend to decline as the testing interval increases, resulting in only a modest degree of stability in personality components when individuals are examined over long periods of time. Second, these studies also suggest that periods that are characterized by life course transitions and changes in important social roles (e.g., work and family) are accompanied by considerably less personality stability than is found elsewhere in the life-span (see also Kogan, 1990). Thus, these studies provide evidence for the hypothesis that interindividual stability in personality varies at different portions of the life-span (Moss & Susman, 1980).

Bloom's (1964) argument that patterns of stability and change may reflect the influence of contextual factors has been addressed in an exemplary fashion in the context of several longitudinal studies of women's personality development during adulthood. Most notably, Helson and her colleagues (Helson, Mitchell, & Moane, 1984; Helson & Moane, 1987; Helson, Stewart, & Ostrove, 1995) have adopted a process approach and have examined the interplay between social context and personality development for several samples of adult women. Helson, Mitchell, and Moane (1984) used data from the Mills Longitudinal Study (Helson, 1967) to investigate the ways in which adherence or nonadherence to social clock

projects in adulthood was related to women's changes in personality. Three social clock projects were identified in this study: the feminine social clock (FSC) for women who had adopted traditional feminine roles; the masculine occupational clock (MOC) for women who had chosen a career; and no social clock (NSC) project for those women who were neither committed to an FSC nor an MOC.

Using this typology, Helson et al. (1984) showed that women who adhered to the FSC scored significantly higher on personality variables indicative of conformity and norm orientation compared to MOC and NSC. During early adulthood, these women's personality development was dominated by the adaptation to the roles of wife and mother, which was frequently accompanied by a withdrawal from social life, the suppression of impulse and spontaneity, a more negative self-image, and decreased feelings of competence. Twenty percent of the women who adhered to the FSC relinquished this life structure and divorced between the age of 28 and 35. In contrast, women who had chosen the MOC at age 28 were less respectful of norms and more rebellious toward what they experienced as constrictive pressures. Although these women did not score lower on femininity or on well-being, they were more independent and self-assertive than their FSC counterparts (Helson et al., 1984). Long-term follow-up of the MOC women showed that those who continued to stay on the MOC into middle adulthood showed greater confidence, initiative, forcefulness, and intellectual independence than women who did not continue the MOC. Finally, women who were not engaged in any social clock project (NSC) also showed a distinct personality structure. In particular, they showed lower well-being and self-acceptance, were less independent and more norm-oriented. Virtually all NSC women were dissatisfied with their jobs, and the single women reported being lonely. By their early forties, some NSC women were better adjusted than in early adulthood and were working or had established a family. In summary, by taking an individual differences approach to patterns of adherence to the social clock, this study showed for a sample of adult women that distinct patterns of personality characteristics were related to different social clock projects and to different life outcomes across the first 20 years of adulthood.

In another study, Helson, Stewart, and Ostrove (1995) examined how different ego-identity patterns were related to women's personality characteristics and life outcomes in three different longitudinal samples. In particular, Helson et al. (1995) distinguished

four ego-identity groups depending on whether individuals had an integrated or unintegrated identity and whether they had actively searched for an identity or had accepted a foreclosed identity. Across the three samples, results were consistent in showing that unintegrated accepters showed less initiative than other women, that unintegrated searchers had less impulse control, and that integrated accepters scored higher on support of norms and traditional values. These relations showed high consistency across time. Moreover, in the three cohorts, identity status showed different relations to life outcomes such as marital status, family status, and work. Overall, Helson and her colleagues (see also Helson & Moane, 1987; Helson & Wink, 1992) have shown that women's personality structures changed not only in systematic and normative ways in early and middle adulthood (Helson, 1993; Helson & Stewart, 1994), but that the observed changes were often related to specific changes in social roles and transitions in social contexts, thus creating distinctly different life paths for individuals with different personalities (see also York & John, 1992).

A great deal of work based on the contextual approach has also been conducted by Block and his research group (Block, 1971, 1993). In 1968, Block and Block (1980) initiated a longitudinal study of personality development in which they enrolled a heterogeneous sample of 128 children from two nursery schools in Berkeley, CA, which has been followed into early adulthood. Block and Robins (1993), for example, examined the longitudinal consistency and change in self-concept and self-esteem from early adolescence to early adulthood. They found that the self-esteem of girls showed a fair amount of continuity from ages 14 through 23, whereas the self-esteem of boys showed marked restructuring from age 14 to age 18, and reasonable consistency from ages 18 to 23. For both sexes, self-esteem was not related to intelligence or to social class. Young women and young men who scored high on self-esteem were independently characterized by observers as resilient, assertive rather than submissive, undiscouraged by adversity, without fluctuating moods, decisive, having a sense of personal meaning, responsive to humor, and unpreoccupied by ruminative fantasy (Block & Robins, 1993). However, there were also a variety of sex differences suggesting that self-esteem is embedded into very different characterological contexts for the two sexes. Young women who scored high on self-esteem seemed happy, warmly extraverted, and deeply concerned about interpersonal relationships, whereas young men with high self-esteem seemed self-focused and defensively critical,

uneasy, and reluctant to make connections with others.

In another study, Block, Gjerde, and Block (1991) examined the personality antecedents of depressive tendencies in 18-year-olds (see also Gjerde, 1995). They found that as early as age seven, boys who subsequently reported feelings of depression were aggressive, self-aggrandizing, and undercontrolled, whereas girls with later depressive tendencies were shy and reserved, oversocialized, intropunitive, and overcontrolling. Similar gender differences were observed in early adolescence. At age 14, boys with later depressive tendencies were more likely to use marijuana and harder drugs, whereas girls with later depressive tendencies showed no tendency to use marijuana but did show a marked tendency to experiment with hard drugs. These girls also had a low self-esteem. Interestingly, in girls, preschool IQ correlated positively with depressive tendencies, whereas in boys preschool IQ was negatively related to depressive symptoms.

Finally, Block (1993) has reported data on the rank order stability of ego resiliency and ego control over a 20-year period. For the boys and young men, the correlations for ego resiliency were consistently positive throughout the years, suggesting that individual differences in ego resiliency are identifiable from an early age and continue over the next 20 years. The ego resiliency correlations for the girls, however, showed considerable discontinuity from childhood to adolescence and early adulthood. That means that for girls, being resilient during the childhood years had no implication for being ego resilient in adolescence or beyond. With regard to ego control, the cross-occasion correlations were consistently positive for both sexes, suggesting that from an early age on individual differences in the level of ego control are recognizable and continue to distinguish individuals for at least the next 20 years and, from the evidence of other studies (see Block, 1971), even beyond. Interestingly, however, the longitudinal relations between ego resiliency and ego control differed markedly again for girls and boys. Block (1993) explains the sex-specific pattern of relationships with the differential socialization of the sexes which makes girls grow up in a more structured and directive world than boys. In summary, Block and his colleagues (Block, 1993) have provided a great deal of evidence for the longitudinal plasticity and consistency of personality from childhood to early adulthood. In particular, they have shown that personality development proceeds differently for men and women, that consistency and plasticity are age-period specific and time-limited, and that contextual factors, such as

parental child-rearing styles (Roberts, Block, & Block, 1984) and social class membership, influence personality development (see also Caspi, Bem, & Elder, 1989; Caspi, Elder, & Bem, 1987, 1988).

Support for a contextual explanation of personality development across the adult life-span has also been provided by Jones and Meredith (1996). In this study, latent curve analysis was used to examine six major aspects of personality for a sample of 211 individuals across either a 30- or 40-year time span. Jones and Meredith (1996) found that a systematic cross-time pattern existed for five of the six personality characteristics: four of the five curves indicated personality change, and one curve indicated no change. However, even with a unifying basic shape to individuals' cross-time patterns, Jones and Meredith (1996) found individual differences in the degree and direction of change, with some people showing increases, others showing decreases, and yet others showing little change in personality across the time period examined. Moreover, changes in the personality characteristics were in most instances time-limited and age-period specific, suggesting that individuals responded to life-stage specific challenges related to changes in social roles or age-graded social transitions (see Caspi, 1987).

In summary, studies which have examined personality development from a contextual perspective complement findings from trait-oriented studies. Overall, these studies show that both consistency and change characterize personality development across the life-span (Helson, 1993; Helson & Stewart, 1994). Moreover, contextually oriented studies have shown that men's and women's personality development often proceeds differently and is related to different problems of maladaptation and different manifestations of mental health problems (see Nolen-Hoeksema & Girgus, 1994).

#### 1.09.5.1 Gender, Coping, and Personality

Empirical evidence that males and females undergo somewhat different development was provided by Cramer (1991). Throughout the school age period, males progressively externalized conflict, relying on protection and/or direct aggression as defenses, while females increasingly used defenses that internalized conflict, directing aggression inward. Similarly, Labouvie-Vief reports that women's predominant coping strategies are based on self-doubt and turning against the self, while those of men are more likely to use strategies of externalizing and dissociating (Diehl, Coyle, & Labouvie-Vief, 1996; Labouvie-Vief, Hakim-Larson, &

Hobart, 1987). Perhaps as a consequence of these patterns, males and females evidence different patterns of vulnerability/resilience for psychological disorders with age. Special interest has been directed to gender differences in aggression, which increase from childhood to adulthood, while female prevalence of unipolar depression persists into adulthood and is generally twice that of males.

With respect to depression, women of diverse social roles and ethnic backgrounds appear to display similar risk factors associated with perceptions of personal control, power structures, and gender stereotyping; even in a relatively advantaged, professional group, women continued to struggle with vulnerability to depression associated with achievement and affiliation needs in often hostile environments (McGrath, Keita, Strickland, & Russo, 1990). Nolen-Hoeksema and Girgus (1994) examined the literature on sex differences in unipolar depression and found the most consistent explanation regarding the depressive experience related to people's responses, or coping strategy, to depressive episodes. While men tended to favor active responses to negative moods, such as thinking of other things, ignoring their problems, or participating in physical exercise, women became more ruminative and introspective, which matches a more passive and emotionally focused feminine style.

Profound differences also exist in how men and women subjectively experience their successes in the academic and intellectual domains. For example, many studies have demonstrated gender deviations in the relationship of actual intellectual performance and one's self-concept about those performances. Men and boys are more likely to adopt a self-enhancing strategy in evaluative situations (Dweck, Davidson, Nelson & Enna, 1978; Martin & Nivens, 1987; Roberts, 1991; Roberts & Nolen-Hoeksema, 1989). That is, men tend initially to overestimate their performance and remain unaffected by negative evaluations, while women underestimate their performance and are more reactive to others' feedback, both positive and negative. Such splits are supported by the feedback boys and girls receive from parents and teachers (American Association of University Women [AAUW], 1992; Yee & Eccles, 1988).

Contemporary research suggests that such patterns continue into the late 1990s. Despite few objective differences in intellectual status between males and females, women on average continue to attain lower levels of achievement than men (e.g., Kaufman & Richardson, 1982; Tomlinson-Keasey & Blurton, 1992). Although in early childhood, girls equal or surpass boys on most measures of school achievement, by



early adolescence, they show a steady decline in self-esteem as they go through school. By high school, girls are found to retreat from achievement-related challenges, particularly in math and science (AAUW, 1992; Kerr, 1985; Terman & Oden, 1959; Yee & Eccles, 1988). Especially in such gender-stereotyped areas as math, this results in girls' disclaiming a sense of agency, even though they may outperform boys (Byrne, Shavelson, & Marsh, 1992).

The work of Gilligan (Gilligan, 1982, 1990) and others also shows that girls who, at the dawning of adolescence were self-assured and outspoken, become increasingly tentative and silent about what they know. In part, the girls' silence mirrors the silence of adult women who act as their models. "At the edge of adolescence, eleven- and twelve-year old girls observe where and when women speak and when they are silent" (Gilligan, 1990, p. 25). In part, they come to doubt that what girls and women know and experience has value in the adult world, because it too often "brings a message of exclusion—stay out; because it brings a message of subordination—stay under; because it brings a message of objectification—become an object of another's worship or desire, see yourself as you have been seen over centuries through a male gaze . . . keep quiet and notice the absence of women and say nothing" (Gilligan, 1990, p. 26).

As noted earlier, however, these different orientations may be particularly characteristic of the early half of the life-span, while the second half brings a relaxation of the demands for "sex-appropriate" behaviors (Jung, 1932; Labouvie-Vief, 1994). As reviewed by Labouvie-Vief (1994), some individuals around the middle of adulthood do appear to experience such integrative changes. Accordingly, some women around midlife claim domains they have avoided thus far, moving from a stance of inferiority to one of openness, assertiveness, and power. In turn, some men integrate their "feminine" aspects, accepting their vulnerability and nurturance. However, many studies have not carefully ruled out alternative interpretations. For example, Gutmann's discussions suggest that some men, rather than integrating notions of feminine power, become threatened and enfeebled by a sense of overpowering, "masculine" women. Further, such postulated midlife changes have not yet been differentiated from cultural/cohort changes that may imply a move towards less gender role polarization.

### 1.09.6 CONCLUSIONS

In this chapter, we have reviewed some recent trends in the domain of life-span development.

We have suggested that with the broad worldwide reorganization in population dynamics, age distributions, and information systems has come a new way to look at development across the life-span. Not only has the concept of development been pushed from early adulthood into later adult stages, but the kinds of characteristics supposed to indicate "mature" functioning also have been broadened. In general, there is a strong emphasis on complexity and introspection, on the integration of emotion and cognition, and on the ability to locate oneself in a complex network of contextual, historical, and interpersonal regularities. In addition, in contrast to traditional views, recent developmental approaches also have systematically highlighted the role of context in shaping individualized patterns of development.

This shift is visible both in the area of cognitive and of personality development. In both of these domains, research has increasingly departed from old stability assumptions and pointed out the wide range to which dimensions of cognition and personality change over individual and historical time. We have also placed emphasis on research that suggests that despite this diversity of developmental patterns, there may be several relatively uniform change patterns that characterize different stages of adulthood. Thus, research on transitions from early to late adulthood indicates that there may be a host of reorganizations that lend substance to older notions of a "midlife crisis," with increases in complexity of thinking about change and psychological process in self and others.

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# 1.10

## Social Psychological Foundations of Clinical Psychology

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### 1.10.1 INTRODUCTION: THE HISTORY OF THE SOCIAL-CLINICAL PSYCHOLOGY INTERFACE

One achieves mental health to the extent that one becomes aware of one's interpersonal relations. (Harry Stack Sullivan)

In this chapter, we will describe areas of basic research in social psychology that provide a foundation for applications in clinical psychology. First, it is important to define social psychology. We prefer the definition provided by pioneering social psychologist Gordon Allport who defined social psychology as “an attempt to understand and explain how the thought, feeling, and behavior of individuals are influenced by the actual, imagined, or implied presence of others” (Allport, 1985, p. 3). The breadth of this definition is readily apparent when one tries to imagine human situations not involving one or more of these elements. Even in the realm of biological phenomena, social processes have been found to interact to influence behavior. From the early part of the twentieth century, this breadth of inquiry that represents social psychology has been appealing to clinical psychologists interested in understanding the bases of abnormal behavior. Social and clinical psychology have a long history of interaction that we will trace very briefly here.

In 1921, Morton Prince and Floyd Allport changed the title of the *Journal of Abnormal Psychology* that Prince had begun in 1906 to the *Journal of Abnormal and Social Psychology* (*JASP*). They began this forum to reflect the key role of interpersonal processes in psychopathology and psychotherapy. These scholars had observed the pivotal role of interpersonal relations in various aspects of psychopathology observed during World War I and also believed that the study of social behavior benefited from research on abnormal mental processes.

While *JASP* was split into the *Journal of Abnormal Psychology* and the *Journal of Personality and Social Psychology* in 1965, explicit work at the interface of clinical and social psychology has been vigorous since the late 1920s. Many scholars deepened our understanding of the role of social processes in clinical psychology during this period. Various neo-Freudians including Harry Stack Sullivan and Karen Horney in the 1920s and 1930s emphasized social processes in phenomena which psychoanalysts previously had interpreted without much reference to interpersonal relations. In the 1930s and 1940s, Kurt Lewin (see Patnoe, 1988), known as the founder of experimental social psychology, promoted his ideas about an

action psychology that involved the application of theory to the solution of interpersonal and group system problems. Frank (1965) wrote about persuasion and healing. A host of workers authored or edited books that analyzed and stimulated integrations of social and clinical psychology, including: Goldstein, Heller, and Sechrest (1966), Brehm (1976), Weary and Mirels (1982), Strong and Claiborn (1982), and Leary and Miller (1986).

This high degree of interdisciplinary activity between social and clinical psychology is most vividly displayed in the 1990s via the research published in many scholarly forums. Furthermore, a journal, the *Journal of Social and Clinical Psychology*, explicitly representing this interface, was created in the 1980s (Harvey, 1983).

In the following sections, we will review broad aspects of social psychology's contributions to the foundations of clinical psychology. The following topics will be considered: social perception and attribution, including discussion of prejudice; persuasion and social influence; interpersonal attraction and close relationships; and altruism and helping behavior. We should stress that our review will be selective. There now is a vast landscape of scholarly interaction between social and clinical psychology, with work in each field influencing work in the other. For a handbook-length treatment of this landscape, the reader is referred to Snyder and Forsyth (1991).

### 1.10.2 SOCIAL PERCEPTION AND ATTRIBUTION

#### 1.10.2.1 An Essential Base

The topics of social perception and attribution occupy a central role in research and practice in clinical psychology. As will be shown in a later discussion of Rosenhan's famous study “On being sane in insane places” (Rosenhan, 1973), social perception, or how we perceive other people, is central to the understanding of labeling behavior as abnormal. Rosenhan essentially showed that even educated professionals, too quickly and without cautious scrutiny of the evidence, label others and then direct behavior toward them that corresponds with the label. In so doing, he revealed a major feature of social perception, namely that it often is biased in terms of its rational, careful consideration of available information. Beyond Rosenhan's demonstration, it has been recognized since the mid-twentieth century that social perception and attribution, which refers to inferences about causality and responsibility, are pivotal in determining how people interact

with one another. These processes, hence, are critical not only in the unfolding of a person's social interactions in all areas of life, but also in therapist–client interactions during therapy.

In social psychology, social perception often is treated as the general area that subsumes a number of subareas, including attribution, social cognition, impression formation, person perception, and self-perception (Weary, Stanley, & Harvey, 1989). But since the late 1950s, each of these subtopics has spawned a large, specialized literature. It has been a long time since scholars such as Heider (1958) and Kelley (1967) attempted to integrate the diverse ideas and findings. Nevertheless, their earlier analyses still have great reach regarding the development of theory and research pertaining to people's perception of others.

The concept of self is a general topic that traditionally was considered separately from social perception (James, 1890). However, self-perception, which refers to the inferences we make about ourselves, was conceived by Bem (1972) as involving a process in which people learn about themselves and their attitudes and dispositions by engaging in an attribution process not unlike what they do when they make inferences about others. In addition, Jones (1964) built on Goffman's (1959) ideas about self-presentation and developed a set of penetrating analyses of how people tactically present themselves to others in order to try to fulfill various goals of social interaction. For example, people may engage in ingratiation, self-promotion, self-martyring, and so on with the goal of social or material gain.

Early work on social perception demonstrated the remarkable degree to which people's own self-biases and motivations affect their perceptions of others. A classic example of this tendency toward bias in perception was provided by Hastorf and Cantril (1954). They showed students at Dartmouth and Princeton Universities film from a football game played in 1951 between those two teams. The game was rough, with lots of penalties especially against Dartmouth, and Princeton's star All-American selection Dick Kazmaier was injured in the rough play. Princeton won but commentary in the local papers continued for some time about the disgusting display of poor sportsmanship.

Hastorf and Cantril's procedure involved showing students at both schools the same portions of the game film and asking them to indicate on a questionnaire the rough play infractions they saw. Hastorf and Cantril found that Princeton students showed a very strong tendency to judge Dartmouth players as most responsible for the rough play. On the other hand, Dartmouth students de-emphasized their

players' rough play and judged the teams as generally equally guilty of infractions. Thus, there is historical evidence for the idea that people can observe the same stimuli and come to different judgments based on their motivations or self-biasing positions. Such evidence is important both to therapists who must make judgments about the dispositions of patients/clients and also to therapists and clinical scholars interested in analyzing the distortions in people's perceptions of others in their social environment.

### 1.10.2.2 Basic Ideas and Theories

In describing how social perception and attribution help us understand psychopathology, labeling of mental illness, and therapeutic interactions, the early foundation analyses will be described. Heider (1958) provided the most general statement of social perception and attribution in describing his naive or common sense psychology. This psychology was concerned with how the "person on the street" systematizes information to make inferences about the social world. It articulated general schemas that people seem to learn and have in mind in inferring how personal forces such as traits and skills interact with environmental forces such as luck in determining behavior. It included analysis of how people infer: the internal–external dichotomy in much social perception, with causal inferences focused either on people's traits and states of mind (internal attribution) or on environmental forces (external attribution); the quest to find dispositional properties behind others' actions because such properties help the perceiver feel more confident about inferences than do external attributions; the causes of success and failure of their own or others' behavior in achievement contexts.

#### 1.10.2.2.1 Heider

Heider (1976) believed that people perceive causal relations as part of their basic perception of the environment. He discussed how when the back door of his home slammed shut, he automatically began to develop inferences, starting first with the attribution that his wife had just come home from grocery shopping. In Heider's analysis, people are seen as trying to develop organized, meaningful perspectives about the numerous events that they observe practically every moment of every day, for it is only "by referring transient and variable behavior and events to relatively unchanging underlying conditions" (1958, p. 79) that

individuals can predict and control their environment. Hence, Heider's answer to why he believed people are almost always, if only implicitly, engaged in attributional activity is to enhance their understanding, prediction, and control over their personal and social environments.

Heider's 1958 book *The psychology of interpersonal relations* provided germs of insights that were developed in later syntheses of attribution concepts by Jones and Davis (1965), Kelley (1967), Jones et al. (1972), and Bem (1972). We will trace these basic ideas as they relate to the foundations of clinical science in the 1990s.

#### 1.10.2.2.2 Jones and Davis

Jones and Davis (1965) developed correspondent inference theory to trace how people search for dispositional causes behind others' actions. According to this model, people use the outcome and nature of actions (including whether the actions were seen as freely taken) to infer intention and disposition on the part of the actor. For example, when members of a jury are making a decision about the guilt of a driver involved in a traffic accident, they will examine the circumstances surrounding the accident. If the defendant-driver was witnessed to be speeding, they will consider why he or she was speeding. Freedom of action might be assessed in the context of the driver's perceived medical emergency. If such an emergency were perceived by the driver and deemed to be reasonable by the jury, this may be seen as reducing the driver's freedom to drive more responsibly; hence, the driver may be judged as less culpable. Did the driver have a history of speeding or receiving driving tickets (such evidence pertains to the driver's dispositions)? If the driver did have such a history, it may lead to a stronger judgment that he or she is culpable. Thus, in accord with this type of deductive reasoning, the jury may be construed as making a correspondent inference. A correspondent inference would be that the driver's actions were consistent with his or her dispositions.

Factors that Jones and Davis (1965) identified as affecting correspondent inferences included the extremity of an outcome and the social desirability of an action (with extreme outcomes and low social desirability both implicating correspondence regarding an actor's intentions or traits). In the traffic accident example above, if someone were killed because of the driver's speeding (extreme outcome), even the medical emergency might not ameliorate a strong judgment against the driver.

#### 1.10.2.2.3 Actor-observer hypothesis and "fundamental attribution error"

Jones and colleagues' work on attributional processes led to many influential ideas and findings. Jones and Nisbett (1972) proposed the actor-observer (A-O) hypothesis, which predicts that actors will attribute their behavior to situational forces; whereas an observer of the same action will attribute the behavior to dispositions held by the actor. Jones and Nisbett's reasoning for this proposed A-O difference was that observers focus on actors as the figure part of their figure-ground perception of the social world. Also, observers may not have experience observing a particular actor previously. Actors, however, do have experience self-observing how they react differently according to different situations. In addition, actors perceive the situation as figural vs. the situation.

While factors such as empathy (Regan & Totten, 1975) have been found to reduce the divergence between actors and observers, this hypothesis has had a long and prosperous life in the attribution literature. In fact, qualifying conditions for the actor-observer effect continue to be studied in the 1990s (e.g., Robins, Spranca, & Mendelsohn, 1996). In general, Jones and colleagues' work has had a rippling effect through clinical psychological research dealing with the inference of traits by individuals and therapists (Harvey & Galvin, 1984). This latter work cautions therapists about the possible mistake of inferring a person's behavior in terms of the person's dispositions, without careful examination of the situational context of the behavior.

Jones and colleagues' work also spurred development of the so-called "fundamental attribution error," which is a vital concept for clinical psychology and now forms the base for cognitive approaches to prejudice (Ross, 1977). According to the "fundamental attribution error," people have a tendency to emphasize dispositional attribution versus situational attribution in viewing others' behavior. Across varying types of situations, people tend to over-attribute actions to dispositions (Funder, 1987). However, does this tendency represent an "error" in the strict sense of an error (e.g., perceiving that a 200 pound person weighs 175 pounds)? No, as several scholars (e.g., Harvey, Town, & Yarkin, 1981) have argued, errors in social perception cannot be as readily established as they can in object perception (when pounds or inches, for example, can be measured with their meaning given ordinal values). Rather, what is occurring in the research on the "fundamental attribution

error” is establishing what more accurately could be called a “fundamental bias toward dispositionality in attribution.”

#### **1.10.2.2.4 Social perception and prejudice**

Jones and colleagues’ work also has formed a base for contemporary cognitive approaches to prejudice. These investigators have analyzed correspondence in attribution mainly in situations in which people are asked to infer others’ attitudes based on their behavior. A ready application to prejudice is the hypothesis that prejudiced people will make negative dispositional attributions regarding a target group’s behavior (e.g., “They are on welfare because they are lazy”). Research in this vein, however, has shown more subtle prejudice effects. Hart and Morry (in press) found that white observers made different dispositional inferences about African-American speakers than they did about white speakers as a function of the speaker’s nonverbal behavior, with the attributions about African-American speakers being more polarized in negative as well as positive directions. Relatedly, in an attitude attribution paradigm in which a speaker takes a certain position, Linville and Jones (1980) found that social evaluations of outgroup members (opposite sex) were more extreme than evaluations of ingroup members (same sex) in the direction being taken by the speaker. Such a finding may be readily linked to the evidence of bias in social perception demonstrated by Hastorf and Cantor (1954).

More generally, a defining characteristic of findings in cognitive social applications of attribution theory is the attribution of extreme, less complex and varied, qualities to outgroup versus ingroup members (Linville, 1982). As noted by Devine (1989), low prejudice individuals appear to be able to replace stereotype-congruent thoughts with thoughts reflecting equality, whereas high prejudice individuals do not appear to be able to inhibit stereotype-consistent thoughts (or have the motivation to do so).

#### **1.10.2.2.5 Kelley**

Kelley (1967, 1972) introduced the idea that sometimes people engage in a detailed attributional analysis not unlike the analysis of variance in statistics, and on other occasions they engage in quick deliberations about causality using priming cognitive sets that he referred to as causal schemata. His analysis was broader than Jones and Davis’s model, referring both to social and self-perception as well as attribution. His conception suggested a number

of hypotheses including that extreme outcomes will necessitate multiple necessary causal schemata (i.e., when something extreme happens, people tend quickly to embrace the idea that there were multiple causes for the event). Kelley’s treatment also systematized many of Heider’s ideas, such as the concept that behavior sometimes engulfs the field, meaning that people’s behavior is so salient that it swamps out perception of situational impacts on their behavior. This idea is consistent with the “fundamental attribution error” and with Bem’s (1972) integration of self-perception into attribution theory. Another major contribution by Kelley (1979) was the introduction of attribution theoretical reasoning to understanding close relationships. As shown, for example, by Orvis, Kelley, and Butler (1976), couples often diverge in their attributions about the causes of conflict in their relationships. This divergence, and whether or not it can be affected by therapy, may play a significant role in whether or not their relationship can be sustained over time.

#### **1.10.2.2.6 Bem and Schachter**

Bem (1972) and Schachter (1964) were not explicit attribution theorists. Yet their ideas have been assimilated into the attribution perspective, and each has presented concepts that have had currency in clinical science. Bem argued that people use their behavior, and the context in which it occurs, to infer their internal states, including attitudes. He suggested that they use the same information they would use in making inferences about others’ internal states to make inferences about their own. This description of a backward-type attribution/perception process has been influential in showing that people do not always have clearly in mind their attitudes about social issues, nor readily understand why they are acting as they are (Nisbett & Ross, 1980). The power of looking back and seeing ourselves engaged in some line of action (e.g., a fight with our spouse) may be great in determining what we think, our next emotional state, and subsequent behavior. Although Bem’s analysis is viewed as extreme in its rendition of how people make inferences about their own attitudes and feelings (Weary et al., 1989), it has had an impact on contemporary social perception work and on practitioners’ emphasis on getting people to act first as a way of constructively changing attitudes and behavior (as in stopping smoking before a stop-smoking attitude is firmly in place).

Schachter’s (1964) theory of emotion has been strongly linked to clinical issues such as phobias. This theory argues that perceptible

physiological arousal and labeling of this arousal in accord with situational or cognitive factors are necessary for the subjective experience of emotional stress. For example, suppose a person stutters rather badly in various social interactions. In a particular situation involving public speaking, this person may perceive that he or she is aroused and attribute the arousal to fear of public-speaking. If there is no plausible, outside cause of the arousal, the individual may become even more anxious. As suggested by Schachter's students Valins and Nisbett (1972), the person may exacerbate the stuttering in this situation when no other source exists for the anxious arousal. However, if this person were induced to attribute the arousal to some other source (such as a drug taken earlier in the day), it is possible that the real arousal associated with public-speaking could be lessened by virtue of the misattribution of anxiety to the drug.

In an indirect way, this hypothesis was confirmed in a finding by Storms and Nisbett (1970) who used placebos in a study with insomniacs to induce some participants to believe that the drug would relax them, while others were led to believe that the drug would arouse them. The investigators reasoned that the participants who were naturally aroused at bedtime but who could attribute the arousal to the drug would get to sleep more quickly than would those who believed that the drug was supposed to relax them, yet as usual were aroused. This reverse placebo effect held sway in research concerned with application of attribution theory until the 1980s. From that point, however, there have been few further demonstrations. The reason probably is that there is always an ethical question associated with giving people who suffer from some ailment false information about what a supposed drug will do for them. Beyond that, the area never established the boundaries very well regarding when indirect versus direct placebo, or suggestion, effects would occur (Kellogg & Baron, 1975).

### **1.10.2.3 More Recent Applications of Attribution Theory**

#### ***1.10.2.3.1 Learned helplessness and depression***

In a very general way, attribution theory continues to be one of the most frequently applied approaches to clinical phenomena. The application is "general" because unfortunately the work sometimes does not involve much bridging between early, basic theory and contemporary theory. An example is the attributional analysis of learned helplessness and depression. Abramson, Seligman, and

Teasdale (1978) took Seligman's (1975) idea of learned helplessness in dogs and added attributional components to suggest that people who are highly depressed are prone to helpless behavior because of a particular attribution style they have developed for most important areas of action. In this depressed attributional style, it is hypothesized that individuals do not attribute positive outcomes to their own actions. These outcomes are perceived as external, global, and chance-oriented factors. If outcomes are negative, however, these individuals perceive themselves as responsible for the outcomes. In effect, they are showing helplessness and the accompanying sense of hopelessness, not unlike what Seligman showed with dogs who could not escape electric shock in a shock box situation.

There is general evidence to support the value of attributional style in understanding depression. As noted originally, however, no attempt has been made to link this work to its implicit base in Heider's (1958) analysis of the conditions of action, nor to the derivative theory and research by Weiner (1974) that posited and showed evidence of how people attribute causes for success and failure in achievement contexts.

#### ***1.10.2.3.2 Depression and correspondent inference***

Depression is the psychopathology that contemporary cognitive social psychologists most often have tried to understand. Weary and colleagues' program of work is illustrative. They argue that moderately depressed people (usually college students, since severely depressed or hospitalized persons have seldom been studied) are particularly sensitive to information about others that might render their worlds more understandable, predictable, and controllable. Such information has included social comparison feedback (Weary, Elbin, & Hill, 1987) and another's negative or unexpected behaviors (Weary, Jordan, & Hill, 1985). Weary and colleagues (1989) further contend that moderately depressed perceivers use a style of information processing that may be characterized as effortful, vigilant, and complex. Accordingly, Yost and Weary (1996) showed that moderately depressed persons were less likely than nondepressed persons to make correspondent inferences and more likely to process the available social information when they were not preoccupied with a cognitive overload task.

Going back to the extrapolations of Jones's ideas about correspondence to the "fundamental attribution error" and to our understanding of prejudice, Yost and Weary's

research implies that given the opportunity, moderately depressed people will be less likely to jump to correspondent inferences about others. Rather, they will engage in the effortful task of analyzing situations to determine how they may have affected behavior. It is not clear what severely depressed persons would do in this situation. They may not even care. As for nondepressed persons and the negative implication of this work for their ready correspondent inferences, the major question may be whether they are motivated to be concerned about how the situation affected another's behavior, or even to be empathic with another regarding the outcome.

#### 1.10.2.3.3 *"Sane in insane places"*

This was the title of a well-known study by Rosenhan (1973). In this study, eight "normal" persons gained entry to 12 mental hospitals, many of which were considered to be excellent teaching hospitals, that were located in different states on the east and west coasts of the USA. The "Patients" ranged in age from early twenties to middle age and held occupations such as psychologist, pediatrician, psychiatrist, painter, and housewife. They had no history of mental illness. To be admitted, they indicated that they had been hearing vague voices and that they felt their lives were empty and hollow. These symptoms were chosen because there was no indication at that time in the literature of what might be called existential psychoses. The pseudopatients also reported typical life courses involving both losses and joys and a variety of experiences that were fairly common for persons at their age. They then were admitted to a mental hospital with no knowledge of when they would be released. Their behavior in the hospitals was described as cooperative and friendly.

Rosenhan found that during the public "show" of sanity, when pseudopatients' histories were taken, there was no instance of detection by the admitting person(s). Nor was their possible sane condition questioned in any of the admitting reports. The length of stay ranged from seven to 52 days, with an average of 19 days. The pseudopatients were not carefully observed in any instance of hospitalization. Interestingly, 35 out of 118 patients on the admission ward did indicate suspicions about the sanity of the pseudopatients.

Rosenhan offered a number of conclusions and speculations regarding this evidence. First, he acknowledged that the tendency to call the healthy sick may be most pronounced during the early admissions stage, since there had been no opportunity to observe the pseudopatients

prior to this time. He also linked the data to work of Asch (1946) in the impression formation field, which showed that people often assimilate perceptions of others around strong, central traits ("warm," "cold," "crazy") that they impute to the others. Perhaps that is what the attending physicians, nurses, and attendants were doing with the pseudopatients: assimilating an interpretation of who they were around certain labels such as "someone who hears voices and who feels empty."

Beyond the implication of the "stickiness of the psychodiagnostic label," it was clear from Rosenhan's study that patients are not very carefully observed, at least not back in those years of mental healthcare. Why? Physicians may have been too busy. Or they may have simply assumed that certain behavioral patterns would be observed if, indeed, careful observation had been done. Yet, they did not even question the pseudopatients very thoroughly. "Patient engages in writing behavior" was the daily nursing comment on one of the pseudopatients, who indicated that he never was asked what he was writing or why he was writing it. Consistent with the "fundamental attribution error," behaviors that were consistent with diagnosis, such as nervousness, were attributed to the pseudopatient's personality. Rarely did the analysis of patient behavior look toward the complex stimuli surrounding the patient. Consistent with what has been referred to as the self-fulfilling prophecy in social perception (Darley & Fazio, 1980), it was evident to Rosenhan that the typical patient received similar treatment from the staff and sometimes reacted by showing a sense of powerlessness and resignation. Rosenhan also suggested that the hierarchical, often frenzied, atmosphere of the mental hospital encouraged a depersonalization of patients by the staff.

Related to Rosenhan's study is the work of sociologists on labeling theory. Sociological social psychologist Goffman (1959) wrote cogently about the presentation of self in everyday life. In this seminal analysis, Goffman posited the similarity between human social behavior and the production of a theatrical work, with tactical stage presentations, entrances and exits, and as much going on "behind the scenes" as was apparent in the presentations to the audience. Goffman (1961) also produced an influential work entitled *Asylums* that addressed the social organization of deviance based on his observations from playing the role of an inmate in an asylum (Goffman, 1961). Central to Goffman's thinking was the view that people readily apply labels to one another, and even to themselves, and then their behavior often is consistent with those labels.

The sociologist Thomas Scheff (1966) went further than Goffman in his labeling theory as it is applied to psychiatric diagnoses. He argued that psychiatric diagnoses were merely convenient labels attached to individuals who violated conventional behavioral norms. He suggested that the stigmatizing process sometimes produced disturbed behavior. Concepts of mental illness, according to Scheff, were part of an ideology embedded in the historical and cultural present of the white middle class of Western societies. Scheff believed that the functioning of psychiatric labels was to reify and legitimate the existing social order.

A lot of progress in diagnosis and treatment has been made since the early writings on labeling theory and Rosenhan's work were published. Still, this work shows the potential insidious nature of prejudicial social perception processes in influencing mental health diagnosis and treatment. The same type of process may be found in the education and prison systems, and in any institution, wherein people are expected to progress from one level to another, but are judged as unequally able or prepared for such progress. The reader also is referred to Rosenthal & Jacobson's (1968) demonstration of the effect of labeling students according to pseudo IQ scores on their "educational progress," as a related piece of evidence about the impact of labeling.

### **1.10.3 SOCIAL INFLUENCE AND PERSUASION**

#### **1.10.3.1 Theories of Social Influence**

Why and how are people influenced by the actions of others? An understanding of how social interactions affect subsequent behaviors can be invaluable to any clinician. Not only does research in this area help in analyzing troubling situations clients may face, but it also adds a depth of understanding to the social interaction inherent in any therapy session.

Social influence is triggered by the desire to be socially accepted and to fit into a group. Festinger (1954) proposed that people continually compare their attitudes and performance to those around them in order to verify their actions and monitor their relative social standing. Using this social comparison process, people adjust accordingly in order to gain approval or status. Social influence can alter behavior in both ambiguous situations with no clear objective answer, and in those where individuals are striving to be accurate or factually correct.

Social psychologist Turner (1991) proposes a social categorization model of social influence.

Individuals identify themselves by social groups or categories, and strive to exemplify the characteristics of these categories while also maintaining individual distinctiveness. Thus we are driven to emulate others whom we feel belong to our categories, and to differentiate ourselves from those whom we see as outsiders. Because social surroundings are in constant flux, so are the social influences being brought to bear at any time. As allegiances and the salencies of particular self-categories shift, so do the relative influential strengths of the individuals surrounding us.

There has been a strong negative connotation associated with the power of social influence. Possibly this stems from the pattern of research in this area which has studied influence primarily by investigating how social forces can increase individuals' errors in judgment or opinion. It is also possible that a Western cultural emphasis on the strength of the individual produces a distrust of any social situation which seemingly diminishes that strength. Social influences, however, are not inherently negative. While they may at times make us more susceptible to destructive norms or behaviors, they also provide the support, affirmation, and security that we, as social animals, need.

#### **1.10.3.1.1 Conformity**

Conformity consists of changing one's behavior to match a group's social norm. Sherif (1937) and Asch (1955) performed classic experimental work demonstrating that individuals confronted with a group norm will often conform to that norm, even if it is obviously incorrect. Sherif's studies utilized the optical illusion of the autokinetic effect, asking participants to judge the illusory movement of an actually stationary light. The participants' judgments converged over time, creating a group norm.

Asch, convinced that Sherif's effect was due to the ambiguous nature of the stimulus, attempted to demonstrate this using judgments of line length. One naive subject was placed in a group of confederates. Three lines of varying length were displayed to the group. The researcher then presented a fourth line which clearly matched one of the preceding three in length. The task was simple and unambiguous: choose the matching line. Asch expected to see Sherif's conformity effect disappear.

The researcher requested verbal responses, with confederates always answering prior to the naive participant. The participant thus heard all of the group's answers and knew they would hear his. On a certain percentage of the trials, the confederates all gave the same, incorrect

answers. Rather than defy the group, a third of the subjects conformed to the erring confederates, also giving the incorrect responses (Asch, 1955). In postexperimental interviews, these subjects often reported being confused about the group's behavior but not wanting to "ruin" the experiment by being wrong.

Asch and his colleagues conducted many variations of this paradigm, including changing the size of the confederate group and the magnitude of the confederate "error." There were always those who conformed. The variation that did succeed in virtually eliminating conformity was the inclusion of an "ally" among the confederates who gave a nonconforming response. Just one other rebel apparently gave the subjects courage to state their true judgments.

In an interesting sociological extension of the conformity work, Jacobs and Campbell (1961) used Sherif's paradigm, cycling new naive subjects into the experiment. They provided a convincing demonstration of how an erroneous social norm can be "passed down" through generations, as the new subjects conformed to the exaggerated responses of the "older generation" of previously influenced subjects.

These studies are especially revealing in that no overt social influence was aimed at the participants. There were no attempts to persuade subjects to "go along" with the group and no explicit threats of any kind associated with disagreement. The pressure to conform, to be part of the whole, is thus a powerful one, even when a situation is straightforward. This pressure only increases when events are uncertain or confusing, as most real world situations are.

Sociologist Phillips (1974) has studied imitative suicides. Phillips's research indicates that the suicide rate increases significantly following a news story on suicide, only in the area over which the news of the first suicide has been publicized. Presumably, these additional suicides are conforming to the example of the first. Incidences of single-car accidents and plane crashes which result in fatalities also appear to increase following the reporting of a suicide. Phillips's contention is that, these, too, are sometimes "copycat" suicides masquerading as accidents (Phillips, 1979). People who are already experiencing confusion or questions about social identities or situations may be especially vulnerable to conformity pressures and react with self-destructive behavior.

#### **1.10.3.1.2 Group influences**

Group influences may be more important to individual behavior than is overtly evident.

People naturally categorize themselves as belonging to one group or another, even when the groups are not clearly defined or even particularly meaningful. Assigning people to groups with meaningless labels, such as "overestimators" and "underestimators" causes individuals to align themselves as part of their "group" (Brewer & Weber, 1994). Even when subjects are told they are not yet part of the group, but are being considered for later membership, they respond to tasks as if they were already group members (Mackie & Cooper, 1984).

Once individuals identify themselves as members of a group, perceptions of self vs. others change. "Ingroup bias" develops, such that what one's own group does is seen as better, more important, and preferable to other groups' actions. Members of a majority group are perceived to be more heterogeneous in opinion and action than members of a minority group, who are all perceived to be much alike (Mullen & Hu, 1989). As the size of the minority decreases in respect to the majority, this perception of homogeneity increases, and vice versa. This appears to be true even of the perceptions of the minority group members; they themselves may perceive their group to be more alike in attitudes and behaviors than they do the members of the majority (Mullen, 1991).

These effects of group membership may partially explain what we view as "prejudiced" behavior, including the belief that members of an ethnic group are "all alike" or cannot be "told apart." This ingroup-outgroup phenomenon may also account for some of the anger that minority group members might sometimes feel toward others of "their" group who assimilate or appear to identify more closely with the majority group.

Group membership also impacts on individual decisions and attitudes. Group polarization is the tendency for the average individual group member's opinions to become more extreme after exposure to the opinions of the group as a whole. Once thought to be a unidirectional movement in favor of higher risk (and thus termed "risky shift"), psychologists now agree that polarization can occur in either direction, depending on the initial tendencies of the group. If the average member begins somewhat in favor of an idea, polarization will occur in the direction of extreme positivity. If the average member begins somewhat against, polarization will push the group in the direction of extreme negativity. There is considerable debate over whether this polarization effect is caused by persuasive arguments of the group majority, normative influence, or the desire to exemplify the group norm proposed by social categorization theory, (Turner, 1991).



Clinicians working with groups, either in a therapeutic situation or otherwise, should be aware of these group influence effects. Individuals may behave quite differently in a group than in an individual counseling session. Opinions and emotions may become more extreme in a group setting. Publicly acknowledging group membership has been used quite effectively in treatments for alcoholism or other addictions, in which admitting the problem is considered to be an important step in therapy. This strategy reminds clients dramatically of their membership. In other therapeutic contexts this may not be appropriate. Care should also be taken in labeling groups. A “cancer survivors” group is probably a better name than “cancer patient” or “cancer victim” support group.

The effects of group membership are quite powerful. A cohesive social group that promotes positive norms can be a valuable source of social support and strength for an individual. An equally cohesive group which focuses on less positive behaviors can be a destructive trap from which it can be difficult to escape.

#### ***1.10.3.1.3 Reducing the effects of negative social influence***

There are methods of reducing the effect of social influence which is contributing to negative or dysfunctional behavior. Social impact theory, conceived by Bibb Latané, attempts to predict the intensity of social influence effects. The intensity, number, and proximity of sources of influence predict the amount of influence directed at the target (Latané, 1981). Thus, to reduce the amount of influence, it is necessary to increase the distance (physical or psychological) from the sources, or diffuse their effect over a larger number of targets by enlisting allies. Even one partner in dissent reduced the Asch conformity effect to almost nil (Asch, 1955).

Another strategy for combating social influence is to make prior commitments before being exposed to the sources of influence. Individuals who write down their judgments or share them with others prior to an influence manipulation are much less likely to be influenced (Deutsch & Gerard, 1955). Deciding ahead of time how to cope with social pressure gives one leverage against it.

In group situations, a minority can effect change and override some powerful norms, if key conditions exist. A minority of one is generally ineffective in changing majority opinion; it is too easy to dismiss one person. A minority of two or more, however, can sometimes convince others to defect from the majority stance, beginning a “snowball” of change (Moscovici, Lage, & Naffrechoux,

1969). The minority is most effective if it begins by being flexible, not rigid, and overdemanding. The minority group also needs to be perceived, in some way, as being “part” of the majority; it must be perceived as a new viewpoint within the majority group, rather than an outside force fighting for inclusion (Turner, 1991). Finally, and perhaps most importantly, minorities must be consistent and confident in their dissenting opinions (Moscovici et al., 1969).

The work in minority influence highlights some of the difficulties in changing attitudes. Persuading others to alter opinions or to comply with certain behaviors is especially complex when competing social influences are strong. There are facets of human social responses that can be utilized in order to facilitate the process of persuasion.

### **1.10.3.2 Theories of Persuasion**

#### ***1.10.3.2.1 Cognitive dissonance***

Consistency in behavior is something humans also strive for (Festinger, 1957). In his work on cognitive dissonance, Leon Festinger demonstrated that when there is a conflict between attitude and behavior, individuals are driven to resolve that conflict. The “dissonance” or conflict between what we may believe and what we have actually done creates great tension. Our behavior appears inconsistent with our beliefs. When a behavior has been performed and can no longer be changed, we resolve any dissonance by altering our belief about the behavior. Thus, a boring task is afterwards described as interesting, if it was done for little reward.

We feel obligated to live up to prior commitments in order to preserve that feeling of consistency in our own behavior. Once we have made a small concession to a cause or have conditionally agreed with some form of behavior, we may feel compelled to comply with future, more extreme, demands that are consistent with this previous action. Cialdini (1993) calls this the “foot-in-the-door” technique of behavioral compliance.

Commitment is an important determinant both of cognitive dissonance and of self-perception (see earlier discussion of Bem’s 1972 conception). As an illustration, a friend may suggest to a person that it would be helpful if the person helps with the political campaign of a candidate in a local election. The person may make a commitment to engage in the campaigning despite having previously not liked the candidate that much. After engaging in the campaigning, the person may experience dissonance in connection with having publicly supported a candidate whom he or she did not previously like very much and try to reduce the

dissonance by deciding that he or she really likes the candidate. From a self-perception position, the person simply “looks back” and sees that he or she decided to do the campaigning without much pressure. Hence, the person may conclude that he or she “must really like the candidate.” Each of these conceptions may be viable in various arenas when people take actions that are counterattitudinal in nature.

The formal application of cognitive dissonance theory to clinical intervention has been proposed by Cooper and colleagues (Cooper & Axson, 1982). Dissonance was theorized by Festinger (1957) as an aversive motivation that will stimulate actions designed to re-establish homeostasis in the organism. In dissonance theory, a person's expenditure of effort on a task may cause dissonance, which then may be reduced by enhancing cognitions supporting the value of the effort expenditure. Cooper and Axson (1982) suggest that effort justification is a common ingredient in psychotherapy. Their thesis is that when a person expends major effort and spends quite a bit of money on therapy, therapeutic success will be facilitated.

The reasons for this enhancement include increased motivation on the part of the client and a more positive view about the likelihood of therapeutic success. Feelings of personal choice and responsibility are at the core of these reasons, and they represent essential conditions for dissonance to occur; people must feel that they had choice and were responsible for the action that led to the dissonance. Cooper and Axson report research using experimental designs with college students interested in weight control that reveal support for this dissonance explanation of therapeutic outcome. Only participants in a condition involving high dissonance (expenditure of effort via exercise and choice in this type of effort) showed weight improvement relative to comparison conditions involving different approaches to weight control.

As noted by Cooper and Axson, their dissonance analysis bears considerable similarity to other theoretical conceptions including Bandura's (1977) view that psychotherapy will be successful to the extent that it enhances people's feelings of personal efficacy. Also, this dissonance perspective is similar to Frank's (1974) proposal that the primary function of all psychotherapies is to restore a person's sense of mastery. Such conceptions are integrally related because of their common emphasis on the importance of perceived personal choice and responsibility in mediating thought processes over a variety of behavioral contexts.

There have been other applications of dissonance to the practice of psychotherapy.

Strong (1968) argued that when a therapist provides opinions that are discrepant from the client's, the client experiences dissonance. The client may reduce this dissonance by derogating and discrediting the therapist. When the therapist is perceived as an expert, this reduces the client's ability to discredit the advice given and enhances the likelihood of attitude change in direction of the therapist's message.

#### **1.10.3.2.2 Elaboration likelihood**

The elaboration likelihood model is one of the most influential approaches in the history of work on attitude change and persuasion. This model was proposed by Petty and Cacioppo (1981, 1986), and it has greatly reinvigorated work on attitude change since the early 1980s. As we will see, this model also has been applied to applications of clinical and counseling practice.

According to the elaboration likelihood theory, there are two distinct routes to persuasion. One, called the central route, views attitude change as resulting from a person's diligent consideration of information that is central to what people feel are the true merits of an advocated position. The second, or peripheral route, views attitude change as occurring when an advocated position has been associated either with positive or negative cues, or when the person uses a simple decision rule to evaluate a persuasive message. These cues and decision rules may shape attitudes or allow a person to decide what attitudinal position to adopt without the need for engaging in extensive issue-relevant thinking. Petty and Cacioppo also admit that sometimes attitude change processes may involve a combination of central and peripheral processing.

This approach to attitude change helps us understand why people may be influenced linearly by the attractiveness of the communicator, or by the communicator's status and presumed knowledge. It also helps us understand better why people sometimes resist persuasion attempts (see discussion below), since some factor such as forewarning of persuasion has activated their thoughtful analysis of the positions being advocated. In its generality and neutrality on whether people are driven by motives such as the aversive motivation presumably involved in dissonance, this approach can subsume processes such as dissonance and self-perception since either may be involved in a particular activation of central or peripheral processing.

Since factors such as level of perceived expertise of a therapist generally are believed to be critical variables in the outcome of

psychotherapy (Strupp, 1981), the elaboration likelihood model is quite relevant to the therapy process. If the person's motivation and ability to think are very low, then increasing the therapist's expertise should enhance compliance with the therapy by serving as a peripheral cue. If a person's motivation and ability to think are very high, however, then expertise should not affect attitudes or compliance since they will be based primarily on a careful evaluation of the issue-relevant arguments presented. Therapy analog studies by Heesaker, Petty, and Cacioppo (1983) have provided support for this general line of reasoning.

In addition, research that did not involve an elaboration likelihood explanation has been reinterpreted as revealing the role of this process. For example, in an analog research design, Heppner and Dixon (1978) exposed participants to an expert or an inexperienced counselor who attempted to change their opinions about their problem-solving abilities. The expert counselors produced greater change on an immediate assessment and an assessment taken one week later. In a delayed expertise finding, Strong and Schmidt (1970) found that although expert and inexperienced counselors did not differ in the amount of attitude change induced immediately after the very brief influence session, one week later the expert counselor had produced more change than had the inexperienced counselor. The result suggests that participants may have been motivated to reconsider the information provided by the expert after the therapy session.

The elaboration likelihood model has been extensively imported into extant work on psychotherapy and counseling (e.g., Heesaker, Conner, & Prichard, 1995). It is an important model for therapy considerations in part because of the value it accords central processing. This type of processing presumably leads to more persistent change and to behavior that is more consistent with the changed attitude.

### **1.10.3.3 Resisting Persuasion**

While other scholars were studying attitude change in the 1960s, William McGuire and his colleagues began an important research program on factors that contribute to resisting persuasion. This work is important for parents and practitioners alike, since it is critical that people learn early in life to withstand pressures to conform and/or act in ways that contradict their values. McGuire's interest in this topic was partially stimulated by the study of how some US prisoners of war had succumbed to the influence attempts of their North Korean captors during the Korean War.

More generally, McGuire's ideas relate to what threatens people and their attitudes and values. As has been shown by Brehm (1966) in his theory of psychological reactance, people cherish their freedoms—including their freedom to hold their attitudes and beliefs—and will react against attempts to limit those freedoms. But what are some of the most enduring ways that people can resist persuasion attempts?

In an early demonstration, McGuire and Papageorgis (1962) showed that forewarning an audience of an upcoming discrepant communication on an involving topic produces resistance to persuasion by stimulating counterarguments in anticipation of the message. In terms of the previously described elaboration likelihood model, such forewarning activates central processing. McGuire believed that attacks on people's attitudes and beliefs for which a person was not prepared would be the most debilitating to maintaining those attitudes and beliefs.

To test his ideas, McGuire (1964) extended his earlier line of work on forewarning by proposing a biological analogy for resistance to persuasion. Just as people can be made more resistant to a disease by giving them a small dose of the germ so that they can develop antibodies, perhaps their attitudes can be strengthened by giving them a mild form of attack of their position along with arguments that refute the attack. Presumably, this inoculation can then make them more resistant to stronger attacks on their position. From the position of elaboration likelihood, the attack on an important topic engages the central processing mode, and individuals are stimulated to think of counterarguments on their own.

McGuire (1964) also addressed the question of whether active or passive participation in an inoculation treatment is more effective when preparing people to defend their attitudes or beliefs. He found that passive defenses appear to be superior when the attacking message occurs very shortly after the defense (in which case people may respond in a rote fashion with the defenses they have just learned). However, active defenses are more effective when the attack occurs at a later point in time (e.g., after one week). As time goes on, people are motivated to develop their own case and to think up stronger arguments to support the case.

Overall, McGuire and his colleagues reported impressive evidence to support the inoculation model of resistance to persuasion. The research paradigm in this work involved attacking cultural truisms such as: "It is important to brush one's teeth after every meal." An attack on this truism might be that frequent brushing

causes gum irritation and eventually gum disease. Individuals presumably have not had practice defending cultural truisms. Hence, such an attack may cause them uncertainty about the value of regular teeth brushing. A refutational defense for an attack on teeth brushing might be to admit that some gum irritation may be produced by poor brushing habits, but that on the whole brushing is essential to preventing gum disease. Unfortunately, there has been little application of McGuire's logic to attitudes and beliefs that are very important to research participants (e.g., regarding the merits of income taxes). This is because these topics do not lend themselves to investigation in an experimental situation. People often have given such topics considerable thought and hence are not readily susceptible to attack. Nevertheless, the basic principles of inoculation theory, especially when coupled with the reasoning of the elaboration likelihood model, appear to represent a valuable set of tools for practitioners and all persons involved in the process of socialization of young people. A fuller discussion of inoculation theory as it is integrated with the elaboration likelihood model can be found in Eagly and Chaiken (1993).

#### 1.10.4 INTERPERSONAL ATTRACTION AND CLOSE RELATIONSHIPS

The area of interpersonal attraction is a traditional one in social psychology, having been the topic of early work in the 1940s and 1950s and enjoying prosperous development until about the late 1970s. This research focused on factors involved in interpersonal attraction and very early dating behavior. These factors included physical attractiveness, similarity in attitudes and personalities, and physical proximity. Interpersonal attraction as a mainline interest in social psychology gave way to work on close relationships around the late 1970s. This latter focus subsumed interpersonal attraction but emphasized intimate, more long-term relationships. It also included how people maintain, and why they sometimes end, close relationships and the consequences of relationship breakdown (or divorce in the case of married couples). The close relationships area is burgeoning in popularity and involves a melding of fields including social and clinical psychology, along with such disparate additional fields as counseling psychology, family studies, sociology, communication studies, religion, philosophy, and traumatology.

Most of the work that will be discussed in this section has derived from research focusing on heterosexual, close relationships. There has

been a small amount of work comparing couples with different sexual orientations (e.g., Blumstein & Schwartz's 1983 "American Couples" study) that will be noted in the discussion. What will not be covered in this discussion are the literatures on friendship and on family relationships.

##### 1.10.4.1 Forming Relationships

Three main factors have been identified as involved in contributing to the formation of interpersonal attraction and then possibly later close relationships between two people. These factors are physical proximity, physical attractiveness, and similarity in attitudes and personality, each of which can be described via a classic study (see Hatfield & Rapson, 1993, for a review).

Festinger, Schachter, and Back (1950) investigated proximity by studying the pattern of how people formed friendships in a new housing development. Festinger et al. found that functional proximity was critical in determining the growth of relationships. For example, people became friends with others whom they encountered doing their normal chores such as putting out the trash. Monge and Kirste (1980) suggested that what is most important about proximity is whether or not there is an opportunity to communicate.

The classic study for physical attractiveness was conducted by Walster (Hatfield), Aronson, Abrahams, and Rottman (1966). They held a college computer dance and paired men and women randomly. Each individual's personality, intelligence, social skills, and physical attractiveness were measured (coders judged the individuals' attractiveness when they showed up at the dance). Walster et al. found that only attractiveness mattered in terms of partners liking one another. Of course, this situation was that of strangers meeting strangers. Still, this finding reveals the potency early on of physical attraction in the liking and dating process.

Hatfield and Sprecher (1986) review a large body of work that shows how differently people are treated when they are perceived as attractive than when they are perceived as unattractive. There appears to be a beauty stereotype in Western societies that has changed little over time. Actress Candice Bergen said of looks:

My looks grease the palm of life, but I resent that they're so important in our society. I resent that people are excluded ... But I'm certainly not talking about myself, because it's clear I've gotten the long end of the stick. (interview in *The Kansas City Times*, April 14, 1984, C-2)

Physical attractiveness has also been implicated in the self-fulfilling prophecy phenomenon that was discussed in the section on social perception. In a provocative demonstration, Snyder, Tanke, and Berscheid (1977) recruited men and women for a study on "the acquaintance process." Men then were given a photo and biographical information about their partners. The photo was a fake. It depicted either "a beautiful" or "an unattractive" woman. Men were asked their first impressions of the woman they saw and about whom they read. Those who believed they had been assigned a beautiful partner expected her to be sociable, poised, humorous, and socially skilled. Those who thought they had been assigned to an unattractive partner expected her to be unsociable, awkward, serious, and socially inept.

In the next phase of the study, men were asked to get acquainted with their partners via the telephone, without actually seeing them. Male expectations had a dramatic impact on the ways they talked to their partners in the telephone calls. That telephone approach, in turn, created a correspondingly major impact on the response of the women. Essentially, the women became what the men expected them to be. If the women had been perceived to be attractive, they indeed talked on the phone as if they were poised and so on. If the women had been perceived to be unattractive, their conversation showed the awkwardness with which the men already had labeled them. By studying the phone recordings, it was found that the males had elicited these divergent lines of behavior either by showing a warm, accepting tone (for the woman perceived to be attractive), or by a less inviting tone (for the woman perceived to be unattractive).

Newcomb (1961) studied the role of similarity in the development of relationships. He studied a group of college men who were given free housing in return for providing ratings of their friendship choices and attitudes. After the men had become acquainted, Newcomb found that liking was greatest among those who had similar attitudes and beliefs. This emphasis on similarity in attitudes and beliefs also was found in a series of laboratory studies by Byrne (1971). In comparison to other kinds of similarity (e.g., in personalities), similarity in attitudes and beliefs appears to be a fairly strong "glue" for continued development of a relationship. Why?

One possibility is that people like other people who are similar to them because that similarity validates their own attitudes and beliefs. It feels good to encounter others who have come to lines of logic about the world that are similar to our own. Further, people who have such similarity often have similar socioeconomic backgrounds, which tends to make them more

comfortable than less similar backgrounds or which tends to elicit less conflict over time.

What value does the foregoing evidence have for the practitioner? Factors such as similarity and physical attractiveness operate pervasively in our social relations. To be ignorant of them is to be ignorant of part of the basis of our achieving or not achieving satisfying relationships with others. In addition, one of the most common concerns on the part of single persons in dealing with dilemmas in their lives is how they can meet others for closeness. The practitioner needs to be aware of these basic findings, which have relevance to strategies for meeting others. Increasingly, people are becoming activists in developing their own networks of single persons who are similar in age and who have similar interests and affiliate in order to extend their opportunities for interaction and possible closeness (Harvey, 1995).

#### **1.10.4.2 Maintaining Close Relationships**

Therapists are centrally involved in how people maintain close relationships over time. Berscheid (1994) noted in a major review of the literature on close, personal relationships that despite this burgeoning literature, we still have little evidence about the processes that are involved over time in determining the progression of close relationships. We simply do not have strong information on questions such as: What determines how close and satisfied couples will become, and what determines whether they will maintain high levels of satisfaction and closeness? This gap in our knowledge contrasts with the extensive theoretical and empirical work both on the determinants of interpersonal relationships and the determinants and effects of breakdown in such relationships.

One relevant topic is that of courtship stages and progression toward marriage (Cate, Huston, & Nesselroade, 1986; Huston, McHale, & Crouter, 1986). This work has involved following a sample of college students (who were matriculating at Pennsylvania State University in the mid- to late 1970s) from the time of becoming engaged to several years into their marriage, or after they had divorced. The investigators inquired about these young people's feelings of satisfaction, and factors involved in their level of satisfaction. Often respondents were first asked to chart their progression toward marriage on graphs. Among the findings deriving from this program are: Significant conflict in courtship is strongly, positively correlated with major conflict developing around the one to two

year mark in later marriage; for example, major early periods of ambivalence and conflict often were related to postmarriage conflict that threatened the relationship. The progression and possible deterioration of relationships are affected by numerous factors including chance and individual participants' networks of friends who either approve or disapprove of the relationship.

Harvey and Omarzu (1997) have developed a process-oriented theoretical analysis of relationship closeness and satisfaction that incorporates other major approaches to the development of these states. They refer to this analysis as "minding the close relationship" to emphasize the integral role of each partner's mind in the flow of interaction in a close relationship. Harvey and Omarzu theorize that to achieve and maintain a high level of satisfaction and closeness, a couple must mutually engage in a set of continuous activities involving: seeking knowledge about their partner and the relationship; self-disclosure; attributions of positive intentions and motivations to their partners for the relationship-relevant actions they observe their partner taking; acceptance of, and respect for, what they learn about their partners over time; and other forms of behavior that are meant to be constructive in their impact on the relationship. These last forms of behavior can include both small acts such as feet rubs and large acts such as ongoing problem solving in parenting and coordination of home and work activities. Critical to the minding process is the meaning imputed to relevant acts by the partners. Within a strong, growing relationship, most acts will be construed to reflect positively upon the partners. Further, such positivity will be based at least to a considerable degree upon sharing of information, perspective, and careful listening and analysis of information.

One of the benefits of Harvey and Omarzu's analysis is that it integrates earlier theoretical conceptions on social penetration which is based on self-disclosure (e.g., Altman & Taylor, 1973; Derlega, Metts, Petronio, & Margulis, 1993), intimacy processes (e.g., Prager, 1995; Reis & Shaver, 1988), love (e.g., Hendrick & Hendrick, 1992), the expansion of self in love (Aron & Aron, 1996), empathy (e.g., Ickes, 1993), and positivity and other illusions in close relationships (e.g., Murray, Holmes, & Griffin, 1996; Swann, 1996). We believe that this minding the close relationship approach has considerable implications for scholars and practitioners interested in relationship progression issues. Nonetheless, this approach still must be subjected to careful study that will better reveal its conceptual and applied value.

#### 1.10.4.3 Dissolving Close Relationships

Fincham and Bradbury (1991) have conducted important research on marital distress that is based on attribution theory in social psychology. According to their contextual model of marital interaction, when one spouse behaves, the partner attends to and perceives that behavior, assigns some meaning to it, and exhibits a response. Between the spouse's behavior and the partner's response is the processing stage. Bradbury and Fincham (1992) argue that the processing stage involves reactions to the stimulus, momentary or transient thoughts, and personality factors such as mood state and attitudes or beliefs about relationships. They also argue that this approach expands the behavioral model of marital interaction beyond its strong emphasis upon behavior and satisfaction by incorporating various cognitive, affective, and personality mediators.

One of the most consistent findings from Fincham and Bradbury's research program is that distressed couples tend to make attributions that blame their partner and exonerate themselves for negative outcomes (such as major embarrassments regarding the state of the relationships occurring in public). On the other hand, nondistressed couples tend to make attributions that do not blame the other for negative outcomes, or that emphasize the joint responsibility for such outcomes (Fincham & Bradbury, 1993). These researchers have showed that to the extent that spouses make nonbenign attributions for negative outcomes at one point in time, their marital satisfaction is lower a year later.

This latter finding is important in making stronger a cause-effect role for attributions in the process of close relationship dissolution. Attributions may serve as a valuable index of relationship distress, but are they causal elements in the chain of factors involved in the ultimate breakdown of relationships? Fincham and Bradbury's (1993) results suggest that attributions at least may become contributory causal factors during the period of breakdown, if not involved from the inception of the process.

Other researchers also have implicated attributions in relationship distress and dissolution. Holtzworth-Munroe and Jacobson (1985) reported that women tend to make attributions about the state of their relationships throughout its course. Men, however, tend to make attributions about the state of their relationships mainly when trouble has arisen. Presumably, women may be more socialized to engage in relationship caretaking than men, and hence women may be more alert and active in inferring

the social psychological dynamics of relationship events. This argument is consistent with other findings and general theorizing on gender differences in dissolution (Bernard, 1982; Vaughan, 1986). It may be true, however, that as people increasingly have learned from therapy and social science's work on relationship problems, many men may have become more active processors of their relationship dynamics (Harvey, 1995).

The idea of attributional divergence has been prominent in the application of attribution theory to close relationship distress and dissolution. Orvis et al. (1976) and Harvey, Wells, and Alvarez (1978) reported that couples experiencing major distress not only made different attributions about the causes of their distress, but also they could not predict their partners' attributions. Such findings suggest that distressed couples often have distanced themselves from dialogue about relationship problems to the point that they are not cognizant of each other's explanations for conflict events. Hill, Rubin, and Peplau (1976) reported a similar kind of attributional divergence among college dating couples who had terminated their relationships. These investigators found that there was disagreement about who left whom, with each respondent claiming responsibility for being the leaver. Hill et al. also noted that other evidence suggested it most often was the woman who had been the leaver (a finding replicated by Vaughan, 1986).

#### **1.10.4.4 Grieving and Recovering from the Loss of Close Relationships**

##### **1.10.4.4.1 The account**

In a book aimed at people who are grieving the ending of their close relationships, the sociologist Weiss (1975) suggested that people use accounts to help them cope with loss in marital separation and divorce. Although "the account" was only generally defined by Weiss, a definition consistent with his statement is: a storylike construction of why the relationship breakdown occurred, who was responsible, key events, and consequences. He argued for the importance of the account in a time of high distress for the individual as follows:

The account is of major psychological importance to the separated, not only because it settles the issue of who was responsible for what, but also because it imposes on the confused marital events a beginning, middle, and end and so organizes the events in a conceptually manageable unity. Once understood in this way, the events can be dealt with: They can be seen as outcomes of identifiable

causes and, eventually, can be seen as past, over and external to the individual's present self. (Weiss, 1975, p. 15).

Weiss used narrative evidence collected from a group of persons attending "Parents without Partners" meetings to evaluate the role of the account in adjustment to marital dissolution. He found that those persons who had well-developed accounts managed the often convoluted and debilitating period of separation prior to formal divorce much better than did those who did not have very well-developed accounts. The latter took much longer to recognize that the relationship had ended and that they needed to grieve and move on. Weiss said about this group of persons who do not readily begin account making, "Those who cannot construct accounts sometimes feel that their perplexity keeps them from detaching themselves from the distressing experiences. They say, 'If only I knew what happened, if only I could understand why'" (p. 15).

Weiss' work has considerable applicability for therapists concerned with helping persons in the throes of separation develop their understandings or stories. As Weiss suggested, an account is not necessarily an objective and impartial interpretation and description. In fact, it may be highly biased and distorted. Nevertheless, these constructions may have powerful psychological truth for the individual.

Weiss' seminal observations had considerable influence on the development of theory and research about how people cope with and recover from major losses including close relationship dissolution. Harvey, Weber, Galvin, Huszti, and Garnick (1986) refined Weiss' logic of the account to define account as a mental construction containing explanations and descriptive material for events and states of being of self, other, and the world. In this conception, account making involves both private and public activities. Harvey and colleagues argued that there were several dynamics of account making, including: search for understanding and meaning associated with a loss, achieving completion regarding a loss, achieving catharsis in venting about the loss, presenting themselves in a certain light relative to responsibility for the loss, self-esteem enhancement, and persuading others about the nature of and reasons for the loss. Several of these functions are inherently social activities (e.g., persuasion of others), and later work focused on the importance of the social interaction component of account making.

This extension of account making and accounts logic was amplified by Weber, Harvey, and Stanley (1987), Harvey, Weber, and Orbach

(1990), and Weber and Harvey (1994) into a model of the role of account making and accounts in people's reactions to major stressors and loss events in general. This model also represented a revision of Horowitz's (1986) stage model of how people react to stress. In this model, account making is the process of working on an account, which is the product of account making. It starts to occur somewhere in a sequence that often involves these phases (that may occur in this order, or in a less linear fashion): a feeling of being numbed by the loss; an outcry about the loss; denial; intrusive imagery; eventually private work at constructing a story of what happened and why; public confiding in a close other (or others) who listens and possibly offers reactions; a feeling of working through the account, acceptance of the loss, and completion of the major grieving; learning from the loss; and a major element of identity change associated with the loss and this processing (e.g., the person no longer sees herself as "John Smith's wife," but now as "John Smith's widow who will go on alone and make a new life for herself"). In studies based on this model, the empathic response of a dependable confidant to a person's loss has been found to be vital in the recovery process (e.g., Harvey, Orbuch, Chwalisz, & Garwood, 1991).

Appreciation of work on the role of account making in relationship conflict and dissolution may be beneficial to practitioners in several ways. It is important to know what is the accounts-narrative truth to a client. While recognizing that a client's story may represent only one of many interpretations of an event or situation, a practitioner can appreciate that the client still may be acting on the basis of this interpretation. Further, the interpretation may need to be challenged. People may be open to challenge of their story and may learn from the feedback and questions posed by a therapist. A practitioner also can help a client recognize the value of developing a story and the functions of story development and the confiding of them to close others. As psychiatrist Robert Coles (1989) has cogently argued, a person's narrative and willingness to share it should be respected as a universal gift.

#### **1.10.4.4.2 Shattered assumptions**

Janoff-Bulman's (1992) work on shattered assumptions provides an invaluable perspective on adaptation to loss. She argues that the psychological disequilibrium that ensues from major losses such as divorce or death of a spouse or loved one often stems from the shattering of our fundamental assumptions about the world.

These assumptions include that our world is secure and benevolent. Of course, over the course of living many humans discover that such assumptions are not tenable. Like Frankl (1956), Janoff-Bulman suggests that even more basic is people's quest for and assumption of meaning and personal control in their lives. Janoff-Bulman contends that the victim's seemingly paradoxical and maladaptive response such as self-blame for a traumatic event is in fact a strategy, possibly unconscious, to restore the illusions of comprehension and control.

Janoff-Bulman also argues that the wide differences in how people respond to traumas such as death of a loved one are due in part to the nature of the event, and which assumptions are most threatened. Survivors of a human-induced trauma, for example, are posited to view themselves more negatively than are survivors of a natural disease process. The idea is that intentional harm carried out by a human perpetrator has a negative impact on the self-worth of survivors, even if this reasoning is irrational. It is an amplification on the "Why me?" question survivors often pose, but with special pertinence to the human-caused trauma.

One of the major contributions for practitioners deriving from Janoff-Bulman's work is the analysis of major tasks with which survivors are confronted in order to adapt to their loss and begin to heal. Like the account-making logic, Janoff-Bulman emphasizes the reframing of interpretations that is required in the recovery process. Included is a rebuilding of a victim's basic assumptions about the world and self. Somehow the survivor needs to integrate the loss into new schemas of understanding that make the loss event less frightening (which may be associated with intrusive, debilitating images and thoughts, as was suggested in the account-making model). The integration may involve reprioritizing of values and goals in living—lessons learned from the loss—and trying to find redeeming value in the suffering (as in the generative act of giving back to others who may suffer similar plights).

Beyond cognition, Janoff-Bulman contends that victims must not be passive, but active in confronting loss. Simple activity such as gardening, caring for friends and pets, and work on hobbies may help. Support groups and close, loving friends may help people "get out of the house" and begin telling their story to others, or if that is too difficult, listening to others' stories. Also, similar to the account-making model, Janoff-Bulman emphasizes the role of close, caring others in a victim's recovery. She indicates that there is considerable evidence that people's adaptation to loss is most often facilitated rather than impeded by the presence



of close others in their lives. Finally, she warns of others' tendencies to blame the victim. Lerner (1980) has theorized a "just world hypothesis" that suggests people believe others deserve their suffering and eventually get what they deserve in life. The tendency to blame victims is central to the phenomenon depicted by this hypothesis.

As Janoff-Bulman notes, in the stress and trauma area the emphasis on people's interpretations and assumptions devolves principally from Lazarus's (1966) pioneering conceptual work and research on how people's interpretations, or appraisals, mediate their reactions to stress. Inherent in this reasoning is that the person has the capacity after a major stressor to reassess interpretations and assumptions. Some losses are so profound that they severely test this capacity.

Examples of losses that may be too overwhelming for any ready cognitive adaptation include: wartime combat veterans' experience of personal terror and the deaths of close friends (e.g., see Shay's discussion of Vietnam veterans' reactions in *Achilles in Vietnam*, 1994); individuals' reactions to the untimely and deeply, personally devastating loss of a spouse (e.g., Lewis' powerful book of grieving "notes" in *A grief observed*, 1961); and the horrific experiences of World War II concentration camp survivors who lost close loved ones to brutality and lost self-respect in bearing degrading, inhuman conditions (e.g., see the powerful stories told in Langer's *Holocaust testimonies*, 1991).

These works help the practitioner define the boundaries of possibility for intervention by interpretation, story development and telling, and reframing of assumptions. The therapeutic relevance of stories of loss and trauma also is realized in family therapy approaches that follow Frankl's (1956) ideas and embrace the empowering value of people's search for meaning in times of great stress (e.g., Lantz, 1993). Increasingly, therapists are advocating the value of people telling their own stories of loss as a means of adaptation (e.g., Howard, 1991).

How do we know when recovery from a major loss has been achieved? Weiss (1988) offers the following set of criteria:

- (i) ability to give energy to everyday life;
- (ii) psychological comfort, as demonstrated by freedom from pain and distress;
- (iii) ability to experience gratification: to feel pleasure when desirable, hoped-for, or enriching events occur;
- (iv) hopefulness regarding the future, being able to plan and care about plans; and
- (v) ability to function with reasonable adequacy in social roles as spouse, parent, and member of the community.

This last criterion encompasses the idea of generativity that has been emphasized by many scholars, most notably Erikson (1963), which may involve a human's most important work both on behalf of others and oneself.

### 1.10.5 ALTRUISM AND HELPING BEHAVIOR

#### 1.10.5.1 Psychology's Role in Promoting Human Selfishness

Before discussing contemporary social psychological work on altruism and helping behavior, we will consider a basic question for the field of psychology that has been raised in this area: What role has psychology played in promoting excessive self-absorption and egoism as compared to concern for others in society? Personality-social psychologists Michael and Lise Wallach (1983) wrote an important book on people's nature, that has much relevance for the area of altruism, entitled *Psychology's sanction for selfishness*. In this, the Wallachs present arguments to the effect that many of the most influential workers in the history of personality, social, and clinical psychology have committed the "error of egoism" in the positions they have advocated.

The Wallachs are suggesting that major schools of thought in psychology offer a particular view of the human that embodies an egoistic concern for self and too little concern for other people. What is this selfishness and "error of egoism" that the Wallachs describe? They do not offer precise definitions, but do offer suggestions and examples. Selfishness is equated with an "emphasis . . . on narcissism, self-concern and preoccupation with 'me'" (Wallach & Wallach, 1983, p. ix), and "with achieving satisfaction for our needs and desires" (p. 18).

The Wallachs suggest that the basis for this portrait of the human as guided by self-centered motivation is found in Freud's emphasis on the importance of satisfaction of bodily needs. The Wallachs point out that within Freud's position, direct gratification of such needs can be most optimally achieved when individuals are free of prescription, restraint, and determination from the outside. Beyond Freud and neo-Freudians such as Sullivan and Horney, the Wallachs also suggest that humanistic psychologists such as Maslow and Rogers play into the selfishness theme. "When we reach Maslow and Rogers, much of society and our relations with other people is seen as obstructing our growth and the development of our potentialities. Self-determination becomes a matter of overriding significance" (p. 152).

Wallach and Wallach take on the influential social psychological analysis of interpersonal relations by Kelley and Thibaut (1978). Kelly and Thibaut's interdependence analysis is concerned with the structures of ongoing situations. The structures are defined in terms of patterns of positive and negative outcomes controlled by the joint actions of interdependent people (i.e., people such as couples in close relationships whose behavior, thoughts, and feelings are intertwined in significant ways over time). Wallach and Wallach suggest that this approach implicitly prescribes mainly a self-concern. They say about the Kelley and Thibaut logic, "Life with others is viewed as a negotiation process, using the carrots and sticks at one's disposal in pursuit of reciprocity . . . Commitment to another person . . . becomes a gambit or move that is tendered to induce reciprocation" (pp. 177–178).

Overall, the Wallachs believe that work such as the foregoing has furthered a cynicism about the nature of humans, with little weight given to the importance of people's commitment to others as an end in itself, rather than as a means to a personal goal. The theoretical work on altruism by social psychologists that will be described below would be viewed by the Wallachs as also embracing self-interest as the basis of most altruism, or helping of others in time of need. Readers who are interested in counterarguments about the Wallachs' position are referred to the *Journal of Social and Clinical Psychology* special issue on this topic (#1, 1985). A major theme by critics of the Wallachs' position in that set of papers is that their analysis often was too black and white and simplistic in representing complex analyses by other scholars. Regarding the Wallachs' critique of Kelley and Thibaut, Kelley and Thibaut (1985) disputed that their theory was prescriptive of a "win at all costs" rationale. They also argued:

it will be apparent that we see no incompatibility between deriving altruism from self-interest, on the one hand, and the existence of genuine or authentic altruism, on the other . . . the Wallachs make the mistake of putting our theory into the heads of the prosocial actors . . . we view altruism . . . as adaptations to problems of social interdependence . . . it seems clear that the Wallachs regard altruism and any logic or sign of self-interest as incompatible . . . we do not . . . Historically and developmentally, self-interest underlies altruism and other interpersonal tendencies. Contemporaneously, prosocial behavior is sometimes promoted by thoughts of self-interest, but it also often reflects direct responsiveness to and concern for other persons' interests. (Kelly & Thibaut, 1985, pp. 29–31).

Beyond such rejoinders, many of the responding analysts indicated a belief that the Wallachs had performed a major service in focusing on psychological views of human behavior and the extent to which behavior is motivated by selfishness. Further, the Wallachs' theme about cynicism regarding psychology's role in modern society was seconded and embellished by an interdisciplinary team of scholars, Bellah, Madsen, Sullivan, Swidler, and Tipton (1985), in a well-known book entitled *Habits of the heart: Individualism and commitment in American life*.

#### 1.10.5.2 General Principles of Altruism and Helping Behavior

There is an extensive literature on altruism and helping or prosocial behavior in social psychology. Among the questions of interest to this area are: What are the conditions that stimulate and impede people's coming to the aid of others in distress? Is there an altruistic personality, and is this personality operative across diverse situations in which helping others may bring great danger to oneself? We will deal with these questions in this section.

There are two major reasons why this literature should be of considerable interest to practicing psychologists. First, it is in the interest of society that people be socialized to help one another and to be genuinely committed to rendering help in others' times of distress when they can be of assistance. Second, as has been cogently argued in many classical psychological analyses, people can gain perspective, courage, and strength to face their own dilemmas when they extend themselves to others who are in distress. Frankl (1956), for example, used his own experience in a Nazi concentration camp to argue that people often create or find meaning in their lives to the extent that they focus on the needs of others and are sincerely concerned about their fellow humans. The term "caretaker" increasingly has been a part of psychologists' vocabulary. In general, it has been found that acting in this role makes people feel good (Williams & Clark, 1989). The general questions here concern when and how people can be effective caretakers of others, without reducing such others' own capacity for self-accomplishment or -empowerment (Steele, 1990).

Altruism theory and research were given a major stimulus by the publication of Latané and Darley's (1970) *The unresponsive bystander: Why doesn't he help?* This book won awards for its contribution to the field of psychology and has become a classic regarding social conditions for helping behavior. In the book Latané and

Darley discussed the case of Kitty Genovese who in 1964, while touring New York, was stabbed to death on a New York City sidewalk (Rosenthal, 1964). The murder was carried out over a 45-minute period and was witnessed by 38 people who heard Genovese's screams, and even observed some of the struggle, from their windows in the nearby apartments. Yet, amazingly, no one tried to intervene or otherwise help Genovese. Why such indifference?

Two principal insights were tested by Latané and Darley. One was that there was pluralistic ignorance: "No one else is reacting; it must not be an emergency after all." Two, there could have been a diffusion of responsibility: "I'm sure someone else already called the police." Latané and Darley tested these ideas in the lab and field by creating fires, accidents, thefts, fights, and seizures. For example, in one procedure, they let smoke fill the room where subject participants, either alone or with others, were completing irrelevant tasks. Would the participants take some action to notify authorities that a fire might be occurring? Or in another situation, a person in a nearby room was overheard by participants alone or in groups apparently having a seizure. Would the participants go to this person's assistance?

As with the work of Milgram (1974, see Section 1.10.6), at the present time it is not clear that such procedures would stand the test of "human subjects review committees" who evaluate research proposals for potential harm to participants. But the results were fascinating and generally have met the test of time and other workers' examination of similar questions.

Latané and Darley repeatedly found that as the number of bystanders in a situation increased, the percentage of participant subjects who helped the experimental accomplice decreased. Among those who did respond, an increase in the number of bystanders led to increased delay in taking action. Thus, Latané and Darley concluded that the "bystander effect" was a powerful phenomenon in influencing helping behavior in emergency situations. These investigators and their colleagues subsequently elaborated on what they viewed as the cognitive/decision-making steps that bystanders experience in an emergency situation. These included: recognizing and attending to the emergency, checking the interpretation that an emergency exists, deciding whether personally to assume responsibility, deciding whether to take some line of action to help, and if so, deciding what line of action is feasible.

An extensive literature on conditions of helping behavior and personality types associated with helping behavior has developed in social psychology since the late 1970s. We will

only highlight some of the major directions. One area of work concerns the impact of momentary emotional states on helping behavior. Surprisingly, it has been found that depressed people are especially likely to help others in need if what they have to do to help is interesting or pleasant (Cunningham, Shaffer, Barbee, Wolff, & Kelley, 1990). This general finding has been linked to the negative state relief model (Cialdini, Kenrick, & Bauman, 1982). This model suggests that helping makes people feel good, and those in a negative mood are motivated to do anything to make themselves feel better. As for positive emotions, it often has been found that people who are made to feel momentarily happy are inclined to help strangers in need. The state of happiness has been created in a variety of ways such as by receiving cookies (Isen & Levin, 1972), by listening to a comedy album (Wilson, 1981), or even going out on a day when the sun is shining (Cunningham, 1979).

Batson and his colleagues have conducted notable research on what has been referred to as the empathy-altruism hypothesis (e.g., Batson, Duncan, Ackerman, Buckley, & Birch, 1981). This hypothesis states that when people perceive an emergency in which they have the potential to render assistance, empathy is aroused and then helping occurs. If empathy is not aroused and if people can avoid the situation, helping will not occur; if it is difficult for people to avoid the situation, helping may occur in the absence of empathy.

To test the empathy-altruism hypothesis, Batson, O'Quinn, Fultz, Vanderplas, and Isen (1983) devised an experimental procedure in which they manipulated empathic concern for the victim and ease of escape from the situation. Participants were informed that they would either have to perform a task while receiving random electric shocks while an observer watched on closed-circuit television, or be the observer while another participant received the shocks. The real subject participant always served as the observer. As the observation began, the accomplice pretended to be in pain when shock was given. But she announced that despite having had a traumatic experience with electricity as a child, she could go on with the study. The experimenter then asked the subject participant if he or she was willing to take the victim's place. Empathic concern was manipulated by the investigator informing the subject participants that the victim had values that were similar to their own (high empathy) or very dissimilar (low empathy). The investigator also manipulated the ease with which the subject participants could escape from the situation by informing them either that they could leave after watching only two shock trials (easy escape) or

that they had to watch 10 such trials (difficult escape).

Batson et al. (1983) found that when empathy was low and escape was easy, subject participants tended to leave rather than engage in prosocial behavior by taking the place of the victim. When empathy was high, subject participants volunteered to take the victim's place, regardless of how easy it was to escape. It also was found that highly empathic subject participants volunteered even when they believed that neither the investigator nor the victim would ever know that they had declined to help. Hence, their helping was not due to evaluative apprehension in the situation, but rather appears to have been mainly influenced by empathy for the victim.

The above genre of research on altruism and helping behavior has focused mainly on factors affecting helping in artificial experimental contexts and in interactions among college students who were not acquainted with one another. In the real world, the opportunity to help others in need often occurs in situations in which people know one another. Is there a personality type associated with helping? This is a question that has been tested via focusing on those who have engaged in helping behavior in situations involving real emergencies or great danger to the helpers.

For example, Oliner and Oliner (1988) obtained personality evidence on people from various European countries who actively intervened to try to rescue Jews during World War II. Oliner and Oliner found considerable similarity among the helpers. They had an internal locus of control (i.e., they believed that their own actions made a difference in changing their environments). They also believed in a just world, which meant that they believed that people who do good deeds will be rewarded for them. They felt socially responsible and believed that people should do their best to help others who are in need. They felt empathy for persons who were in distress, and were not egocentric in general in their approach to other people.

Another powerful presentation of humans' caring spirit is contained in Eva Fogelman's book *Conscience and courage* (1994). She, too, interviewed persons who had rescued Jews during the Holocaust. Her treatment highlighted the small events that often shaped long-term, complex sequences of helping. One of the people she interviewed was a small businessman in Warsaw, Poland. When the Nazis conquered Poland and created the Jewish ghetto, this man said that he was curious about what was happening in the ghetto and found a way to visit it. He said that one transforming moment in that visit motivated him to spend the next

four years risking his life and the lives of his family to help Jews hide from the Nazis. The moment was the sight of flies swarming over the bodies of dead and dying Jewish children who had been killed by the Nazi's brutal treatment of them.

### 1.10.6 CONCLUSION: SYNTHESIS OF THE "SOCIAL ANIMAL IN A COMPLEX WORLD"

Elliot Aronson wrote a popular introductory social psychology textbook entitled *The social animal* (1995). In this book, Aronson described why humans may be conceived as social animals, and began this thesis with Aristotle's statement:

Man is by nature a social animal; an individual who is unsocial naturally and not accidentally is either beneath our notice or more than human. Society is something in nature that precedes the individual. Anyone who either cannot lead the common life or is so self-sufficient as not to need to, and therefore does not partake of society, is either a beast or a god. (*Politics*, c. 328 BC, reprinted Aronson, 1995, p. xvix)

While we may not be beasts, if we are reclusive we still are influenced by our society. The reclusive "Unibomber" was an example of a person who was greatly influenced by outside events and who sought to influence them in his own deadly way, yet he also was extremely reclusive in his behavior. Aronson contended that humans are more rationalizing animals than rational in their decision making (Aronson argued from a cognitive dissonance position). He cited many examples from reckless behavior in driving, smoking, and use of weapons, to warfare and genocide. He also argued that "People who do crazy things are not necessarily crazy" (p. 9). By this, Aronson meant that social conditions such as those studied by Milgram (1974) in his work on obedience may exert a powerful influence on human behavior.

In studies that were designed to examine how people might be induced to obey authority figures, somewhat similar to what might have occurred in Nazi Germany in the 1930s and 1940s, Milgram developed an experimental situation at Yale University that was advertised as involving the study of associative learning. If typical members of the community show up at the Yale psychology laboratories and are told that the study they are volunteering to do concerns learning behavior, why should they doubt the investigator? That was the type of situation set up by Milgram. The researchers were "official-sounding and -looking" in order

to try to determine if ordinary people would obey an authority and do something that they thought might be harming another person. In the course of the experiments, these individuals were “required” to give what they thought were increasingly intense shocks to another person (actually an experimental accomplice). Many of the participants did so, even to the point of the accomplice feigning a heart attack. Milgram’s methods were questionable in terms of potential harm to the participants and could not be carried out today, but they show the great power of social situations.

Situations such as those studied in the obedience work are only illustrative of the diverse social situations that people encounter on almost a daily basis. These situations may involve close others, acquaintanceship groups, work associates, or strangers, but whatever their nature they may totally engulf a person’s field of thought and action. People may be induced to act in a way that is opposed to their personality and moral position because of the nature and influence of the situation.

We believe that a broadly construed social psychological point of view in the last part of the twentieth century represents a complex view of human life that is appropriate to the intricacies of human behavior, behavioral disorders, and psychopathology. This approach points toward many of the types of social variables and situations that influence people’s behavior. As was shown by the 1996 Olympic Park bombing deaths in Atlanta, USA, in many ways we live in a terrifying world. Terrorism and other kinds of daily violence show the propensity of people to kill others in an instant with little guilt. Why? Inevitably the best available answers to such questions take us on an odyssey into work on social psychological findings and theories. These findings and theories are insufficient, in and of themselves, to answer such global questions. But they do sensitize us to the many nuances of understanding that may be tenable in considering causation. Coupled with the work from related fields in psychology, this body of work frames an understanding of some of the major dynamics behind these seemingly inane acts.

Contemporary social psychology is well integrated with the fields of personality, health psychology, and research in clinical psychology, all of which form an indispensable base for the practice of clinical psychology. This melding together of fields in the pursuit of knowledge and relevant application is illustrated in all of the sections of this chapter. It leads to a synergy in research that is quite positive and exciting for progress in understanding better the “social animal.”

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# 1.11

## Cognitive Psychology

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### 1.11.1 INTRODUCTION

#### 1.11.1.1 Cognition and Clinical Psychology

Cognitive psychology is that specialty area of psychology concerned with the examination of

how knowledge is acquired, how it is internally represented, how it interacts and modifies or is modified by previous experience and existing information, and how acquired knowledge affects feelings and behavior. The subject matter encompasses not only the process of thinking

(attention, concentration, consciousness, memory), but also an examination of emotions and motivation in relation to cognition. There has been a dramatic shift in the theoretical base of clinical psychology since the 1940s from psychoanalytic and behavioral models to cognitive models as the task of clinical psychology has changed to encompass more than simply disturbed behavior and maladaptive habits. Clinical psychologists now concern themselves routinely with such aspects of human behavior as the emotional state of their patient, their patient's ability to cope with difficult situations, their interpersonal problems, their sense of well-being or self-esteem, their memory and concentration, their thoughts and thinking process, and their physiological health status (Brewin, 1988).

In many respects this is a golden age for cognitive psychology. The rapid development of noninvasive neuroimaging technologies in the mid-1980s has allowed researchers the unprecedented opportunity to confirm some hypotheses of cognitive functioning derived from laboratory learning experiments by looking at neural function at the time that the subject performs the experiment. The use of positron emission tomography (PET), functional magnetic resonance imaging (fMRI), event-related potentials (ERPs), electroencephalography (EEG) and magnetoencephalography (MEG) technologies allows researchers to look at real-time "pictures" of the human brain in the process of cognition through measurements of blood flow and brain electrical activity. The reader is referred to Posner & Raichle (1994) for an overview of this fascinating area and Posner & Raichle (1995) for further commentary and critique. Noninvasive neuroimaging has led to increased collaboration between cognitive psychologists, neurologists, neuropsychologists, clinical psychologists, psychiatrists, mathematicians, physicists, and neuroscientists of all sorts. The point is being rapidly reached that brain, mind, and behavior need not be studied in isolation, and emerging electrophysiological technologies such as multiple single unit recordings and ERPs will only add to the available tools at the disposal of future cognitive scientists. Models of cognitive processes, neural networks, and mathematical models of cognitive function have also become highly developed since the late 1980s (Taylor, 1997), there have been enormous strides in our understanding of memory functioning from lesion studies (Zeki, 1993), and hundreds of studies have been published in the areas of mood, motivation, and cognitive social psychology. Within this same time span, cognitive behavior therapy and rational emotive behavior therapy have become highly refined therapeutic modalities that address psychological problems

ranging from anxiety and depression to panic disorder, anger problems, obsessive-compulsive disorder, and suicidal behavior (Ellis & Dryden, 1997; Freeman & Reinecke, 1993; Yankura & Dryden, 1997).

This chapter will provide an overview of some of the current literature in cognitive psychology that is particularly germane to the practice of clinical psychology in general and the practice of cognitive behavioral therapies in particular, with the clear understanding that this is a highly condensed and highly selective sampling of a rapidly expanding area. A major focus will be an examination of the origin and modification of emotional states, particularly anxiety and depression, due to their central role in contemporary clinical psychology.

#### **1.11.1.2 Models of Cognition, Learning, and Memory**

Since the 1970s, a central tenet in cognitive psychology has been that much, if not all of cognition can be conceptualized as information processing, and a great deal of work has been done since that time to identify component cognitive processes that underlie cognition. Early models of cognition were mainly derived inferentially by systematically varying stimulus properties and carefully measuring responses. The availability of high-speed computers allowed the later development of more sophisticated discrete stage linear mathematical models which, at least in a very limited sense, were capable of mimicking some aspects of cognitive function. The later use of more sophisticated computer systems in the 1980s dovetailed with the growing awareness that functional brain systems and complex neural circuits rather than linear modules were involved in higher brain function. Since that time, computer simulations of cognitive processes have been built that allow hypothesis testing of complex interactive systems and model the possible effect of localized brain damage on subsequent brain function. Computational models have been built which examine acquired disorders of reading (Hinton & Shallice, 1991; Mozer & Behrmann, 1990; Patterson, Seidenberg, & McClelland, 1990; Plaut & Shallice, 1993) as well as disorders of perception, attention, memory, frontal lobe function, and language (Feinberg & Farah, 1997).

Computational models of cognition have influenced the field of cognitive psychology since the 1970s by providing meaningful heuristics to link environmental events and information stored in memory to subsequent behaviors. The earliest such models were based primarily on

digital computer processing methodology. These models used processing algorithms based upon the premise that environmental events trigger the retrieval of symbolically coded events from a discrete physical memory location. These events are operated upon by some central processor according to previously stored instructions, and information about the subsequent behavior is fed back to memory stores by this same central processor (Newell & Simons, 1972). More recently, computational models of cognition have been developed that more closely model brain function and rely upon parallel distributed processing (PDP) architecture. These have provided the methodology for artificial neural networks as cognitive processing analogues (Anderson, 1995; Rumelhart & McClelland, 1986). Because the human brain is a functionally and anatomically distributed and highly interactive system, PDP modeling has provided a conceptual framework and concrete tools to begin to explain the effects of local lesions on cognitive function as well (Farah, 1997).

Briefly stated, PDP systems consist of large numbers of neuron-like units multiply connected to each other by weighted connections that determine the amount of activation or neural transmission that flows from one unit to another. Each unit or collection of units functions locally and in parallel with other units, with no true central processor governing the behavior of the network. An event is represented as a pattern of activation distributed across units. PDP networks can accommodate multiple types of learning depending upon the computational properties of the network. These computational properties in general are a function of the activation rule of that network (e.g., linear vs. nonlinear activation), the type of connectivity within the network (e.g., unidirectional vs. bidirectional), and the learning rule imposed by the architecture of input units vs. output units. Long-term memory is represented by the encoding of a pattern of connection strengths distributed within a population of units or neurons.

A basic premise of most PDP models of memory is that because every part of the brain or all collections of neural units possess the ability to learn, memory cannot therefore be housed in a specific structure or structures of the brain as was the case in classical theories of memory, but rather expressed as memory processes or circuits which are related to those areas processing that information. This is consistent with the observation that areas of the brain which process auditory information house auditory memory and areas that process visual information also house visual memory,

for example, and is consistent with the observation that functionally similar areas of the brain may contain multiple memories of any specific event based upon their specific function (color, form, size, motion, etc.), all or some of which may be recalled when a memory of the event is recalled (Kolb & Whishaw, 1996).

The process of memory recall can be divided into two broad classes or types with different underlying neural structures. Explicit memory refers to the conscious recollection of previously experienced events, whereas implicit memory is a process that is automatic, occurs at a level outside of consciousness, and is typically inferred from the effect that experience or practice has on the performance of a particular task (Moscovitch, 1984). Implicit memory is encoded in much the same way that it is perceived, whereas there is some active reorganization of data that occurs in explicit memory.

Explicit memory can be further subdivided into two subtypes, strategic and associative/cue-dependent, based upon those types of tests used to elicit explicit memory. Associative/cue-dependent explicit memory is a relatively automatic response that is elicited following a cue that is sufficient to bring that memory to mind. Strategic memory is that in which cuing does not elicit a specific response but rather initiates a memory "search" not unlike problem-solving (Moscovitch, 1992). Petri and Mishkin (1994) have identified four major limbic structures involved in the explicit memory system: the rhinal cortex, the amygdala, the hippocampus, and the prefrontal cortex. These structures have reciprocal connections with the medial thalamus, the basal forebrain and orbital frontal cortex, and sensory areas of the neocortex. Experimental results suggest that the rhinal cortex is involved in object memory, the hippocampus in spatial memory, and the amygdala in emotional memory (Kolb & Whishaw, 1996). Evidence both from animal models and lesion studies in humans has demonstrated sparing of old memories and implicit memories following damage to medial temporal structures, but severe impairments in the formation of new explicit memories.

Implicit memory can be further subdivided into two subtypes, procedural and item-specific, based upon those types of tests used to elicit implicit memory. Procedural memory refers to the acquisition and retention of general skills, procedures, or rules including learned motor tasks. Item-specific tests, in contrast, are concerned with the acquisition and retention of a particular type of information and the increased accuracy or speed with which a previously seen item identified on repetition is a function of the repetition priming effect (Moscovitch, 1992).

Petri and Mishkin (1994) have identified the caudate nucleus and putamen (basal ganglia) as those brain structures centrally involved in the implicit memory system. The basal ganglia receive projections from cells in the substantia nigra and all regions of the neocortex and send projections via the globus pallidus and ventral thalamus to the premotor cortex. Animals with damage in basal ganglia circuits demonstrate preserved recognition memory, spatial memory, and emotional memory, but demonstrate impairments in learning motor skills, learning to make appropriate responses to cues, and demonstrate poor performance on association tasks (Petri & Mishkin, 1994).

Finally, fundamental to the understanding of conscious and unconscious processing is the inter-relationship between networks of attention and short-term memory or "working memory." Working memory refers to the form of memory used to hold words, names, digits, or other items in memory for a brief period of time (Baddely, 1986). Working memory appears to be a function of the prefrontal cortex which allows the maintenance of information in short-term memory that is later used to guide action (Goldman-Rakic, 1987; Moscovitch & Umiltà, 1991). Short-term or working memory has a different structural basis from long-term memory. The parietal-frontal spatial system, which includes Brodmann's frontal cortex area 8 and projections from the parietal cortex, is involved in short-term memory for spatial location of objects and in searching for an object when a stimulus is presented. Frontal cortex areas 9 and 46, with projections from the inferior temporal cortex (the inferior-temporal dorsolateral frontal system), are involved in the short-term memory for visual objects and remembering objects that are identified sequentially (Petrides, Alivisatos, Evans, & Meyer, 1993; Wilson, Scalaidhe, & Goldman-Rakic, 1993). The available evidence suggests that working memory and long-term memory are parallel mechanisms in which material is processed separately and simultaneously, although information can be pulled from long-term memory and used in working memory for short-term problem-solving.

Electrophysiological evidence from primate studies suggests that there are at least three different types of attentional mechanisms in the mammalian brain (Moran & Desimone, 1985; Petersen, Robinson, & Morris, 1987; Spitzer, Desimone, & Moran, 1988). There is evidence for an attentional mechanism in the parietal lobe that enhances spatial attention, one in the inferior temporal region that selects objects, and one in the visual and posterior temporal cortex that selects object features. In addition, cells in the frontal eye fields have been demonstrated to

select movement. PET and ERP data with human subjects are consistent with these findings (Corbetta, Miezin, Dobmeyer, Shulman, & Petersen, 1991; Corbetta, Miezin, Shulman, & Petersen, 1993; Naatanen, 1992).

Regions of the frontal lobes are also activated in tasks requiring response selection. For example, the anterior cingulate cortex is activated in divided attention tasks as well as the dorsolateral prefrontal cortex (Corbetta et al., 1991), and verb-generation tasks activate the inferior frontal cortex (Posner & Raichle, 1994). Posner and Petersen (1990) have proposed that the frontal lobe therefore contains a second attentional system or executive attentional system which plays a major role in the activation of selective attention systems of the posterior cortex and which is closely related to the working memory functions of the frontal lobe. In this model, the frontal lobe is responsible for the programming of mental operations, and the content of consciousness consists of information within working memory that is operated on by the executive attentional system.

Clinically, it is not always easy to separate attention and working memory. Damage to prefrontal areas of the brain, if extensive enough, causes deficits in both attentional circuits and working memory. Also in some instances, observed deficits which appear to represent functional disorders of working memory may instead be residual or secondary to damage to attentional mechanisms. An assessment model of attention has been proposed by Mateer and Mapou (1996) that at least partially addresses this by carefully delineating the boundaries of attention and separating it into two component parts, deployment and capacity/encoding. Deployment is the ability to channel and focus attention, which includes the assessment of arousal, focused attention, and sustained attention. Arousal is assessed by observation, focused attention is assessed by tasks that require the rapid scanning and identification of targets, and sustained attention is assessed by continuous performance tests. Capacity/encoding is the ability to hold information and process it even if distracted or required to divide attention, and includes tasks that measure span of attention, resistance to interference, and mental manipulation.

### **1.11.1.3 Conscious and Unconscious Processing**

An understanding and appreciation of the unconscious has been one of the central themes of clinical psychology historically, and certainly a key concept in psychoanalysis and psychoanalytic psychotherapy. A companion theme is the emphasis placed upon gaining an awareness

of the internal state of the patient, an awareness both on the part of the patient and on the part of the therapist. Patients and nonpatients alike respond to situations and other people in ways that seem disconnected to the current situation and have day-to-day experiences that seem to be neither desired nor clearly volitional. Is this a result of experiences or learning that are below the threshold of awareness or in some way processed differently than those experiences of which we are fully aware?

Early observations of symptom remission in hysterical patients under hypnosis led Freud, Breuer, and later Janet to develop a model of repressed emotions and cognitive content to explain the seemingly miraculous disappearance and reappearance of all manner of dissociative symptoms (blindness, paralysis, anesthetics) in response to hypnosis and suggestion (Brewin, 1988). It was thought that such hysterical symptoms could be the result of traumatic experiences that could not be readily recalled by patients due to the protective psychological mechanism they labeled repression but which, under special circumstances, could be reexperienced or reexamined to the patient's benefit. Working through the patient's resistance to experiencing painful affect and unacceptable thoughts from unconscious to preconscious to conscious awareness, then becomes a central tenet of the therapeutic process. It was felt that the content of dreams and parapraxes or slips of the tongue were further "evidence" of unconscious processes. This model of repressed traumatic emotional events leading to symptoms and seemingly being responsible for day-to-day behavior requires some examination of a mechanism or mechanisms to explain information processing that occurs out of conscious awareness. There has been a great deal of evidence from the laboratories of both cognitive psychologists and cognitive neuropsychologists, particularly since the late 1980s, to confirm a central role of the automatic processing of information without conscious representation.

Early evidence of cognitive performance without awareness can be found in studies in the neuropsychological literature describing patients with amnesic syndrome and so-called "blindsight," reported in the 1960s and 1970s, although there are anecdotal reports dating back at least to the late nineteenth century. Although it is beyond the scope of this chapter to take an exhaustive look at this fascinating area, it would be remiss not to report some examples in detail which illustrate not only preserved discrete cognitive functioning without the awareness of the patient that this capacity remains, but also preserved discrete cognitive functioning that occurs in the total

absence of any awareness on the patient's part that they are even responding in any meaningful way to external events, events that they seemingly cannot discriminate or detect. For a thorough review of this rich area and the relationship of such syndromes to consciousness, the reader is referred to the publication by Weiskrantz (1997).

One of the first well-documented cases of learning in the absence of awareness was the famous patient H.M., who was tested extensively over the course of several years by multiple investigators (Corkin, 1968). H.M. became severely amnesic following the removal of structures in the medial portions of both of his temporal lobes to treat intractable epilepsy. Although H.M. had severe memory difficulties following this neurosurgical procedure, he was nonetheless subsequently able to master pursuit rotor tasks and learn mirror drawing tasks and retain them from session to session, despite the fact that he had no memory of the experimental sessions or memory of ever having seen or used the experimental apparatus. This same sort of motor skill learning and retention of training without any conscious awareness of the learning event has been documented in other densely amnesic patients by multiple authors (Brooks & Baddeley, 1976; Schacter, 1994; Schacter, McAndrews, & Moscovitch, 1988).

Classical conditioning has also been demonstrated in the absence of awareness. In a series of experiments reported by Weiskrantz and Warrington (1979), a severely amnesic Korsakoff patient was conditioned for anticipatory eye closure with a neutral light and tone preceding an air puff to the eye. Despite the fact that he reported no recall between conditioning trials of the air puff or what the apparatus that was in front of him was being used for, the conditioned stimulus reliably elicited the conditioned eyeblink.

Perhaps more surprising has been the observation that densely amnesic patients are able to learn and retain verbal information without explicit recognition or recall. In a series of clever experiments, severely amnesic patients were shown a series of words and tested for recognition after several minutes (Weiskrantz & Warrington, 1970). If asked to respond yes or no to whether they had seen specific words, their performance was at a chance level. If instead they were provided with a very fragmented drawing of a target word and asked to guess the identity of the word, they were much more able to identify those fragmented words to which they had been previously exposed than those to which they had not been exposed, despite having no recollection whatsoever of having seen any of the words.

This sort of "priming" effect to enhance recall in the absence of awareness has also been demonstrated by providing densely amnesic subjects with the first few letters of target words, which increased the facilitation of retention induced by the previous exposure, the so-called stem completion task. Retention intervals as long as four months have been reported following priming in severely amnesic patients such as H.M. (Milner, Corkin, & Teuber, 1968), demonstrating that priming is capable of revealing implicit memory (implicit processing) in the absence of explicit memory.

Experiments with cortically blind patients have revealed that the phenomenon of information processing without awareness is not simply confined to memory systems. It has been demonstrated by several investigators that patients who have sustained damage to the striate cortex of the brain at Brodmann area 17 (the V1 projection area) and who are cortically blind can nonetheless demonstrate the ability to detect the presence of stimuli, to locate them in space, to discriminate direction of movement, to discriminate the orientation of lines, and to discriminate between different wavelengths of light, despite having no awareness that there is any stimulus present in their blind hemifield (Blythe, Kennard, & Ruddock, 1987; Cowey & Stoerig, 1991; Weiskrantz, 1986, 1995).

These seemingly impossible feats of visual perception are explainable due to multiple cortical connections to the human optic nerve and retina. The major input from the optic nerve goes to the dorsal lateral geniculate nucleus and ultimately to V1, and if this circuit is intact patients cannot only "see" objects in the visual field but also have the awareness of "seeing." About 10–15% of the output from the optic nerve goes to nine other visual pathways or connections (superior colliculus, ventral lateral geniculate nucleus, pre-tectum, nucleus of the optic tract, dorsal terminal accessory optic nucleus, lateral terminal accessory optic nucleus, medial terminal accessory optic nucleus, inferior pulvinar, and suprachiasmatic nucleus) which affords the possibility of retaining some aspects of the ability to "see" but without the awareness of seeing (Sahraie et al., 1997; Weiskrantz, 1997).

If the phenomena of "blindsight" and memory retention in severely amnesic patients are to have relevance to the everyday practice of clinical psychology, similar processing of information without awareness would need to be documented in nonbrain injured individuals as well. Although an area not without considerable controversy, investigations in the area of subliminal perception and subliminal semantic activation provide additional evidence that

processing of information can and does occur in the absence of conscious awareness, a level of processing that has been referred to as unconscious cognition (Greenwald & Draine, 1997).

Merikle and Joordens (1997) have used a version of the Jacoby exclusion task (Debnar & Jacoby, 1994; Jacoby, 1991) with short stimulus onset asynchronies to determine the critical stimulus duration at which the unconscious influence of priming exceeded the conscious influence of priming in a word stem completion task. This same sort of priming effect (very similar to the priming effect observed in amnesic patients) has been demonstrated in the Stroop color word task (Logan, Zbrodoff, & Williamson, 1984; Merikle & Cheesman, 1987), in both visual and auditory word recognition tasks (Groeger, 1988; Marcel, 1983), and in visual search tasks (Schneider & Shiffrin, 1977). Multiple neural pathways for processing these types of information are the likely neuroanatomical substrate to account for these types of processing independent of awareness.

Several lines of evidence from numerous investigators in cognitive psychology suggest that there are two general types of information processing: conscious or control processes which appear to be clearly under the control of the individual and automatic processes which are triggered by a stimulus and operate outside of awareness and are therefore not under the control of the individual (Posner & Snyder, 1975). Control processes by definition would be limited by the attention span of the individual, could be characterized as deliberate, effortful, adaptable, and to some extent highly flexible, but processes that could be easily disrupted by stimuli competing for that individual's attention. Control processes would be more likely to be elicited by novel situations which require more effortful cognitive processing (Brewin, 1988), but in highly complex or ambiguous situations the limitations imposed by attention and attention span may lead to decisions that are made by simple rules or *a priori* expectations or hypotheses. Automatic processes, in contrast, require minimal attention, may be activated without intention or awareness, are likely to be rapid and inflexible, and are more likely to be activated in familiar situations in which the individual has had considerable experience. Since automatic processes run in the background below the level of awareness, they are subject to misattributions of causality by linking two events together that occur close together in time and by judging more salient stimuli as causally prior. Automatic processing has an obvious correlate in what has been referred to by investigators as implicit memory in which prior experience influences later

processing without conscious or deliberate recollection of the experience (Schacter, 1987), whereas the correlative memory analogue for conscious processing is explicit memory. There is at least some evidence to suggest that conscious processing of information inhibits automatic processing of that and contiguous stimuli (Posner & Snyder, 1975), suggesting some inhibitory effect of conscious processing and inhibitory effect of explicit memory.

Cognitive models of conscious processing and explicit memory vs. automatic processing and implicit memory have a direct relationship to psychopathology and patient behavior. The excessive rumination of depressed patients may be a manifestation of the inhibitory effect of conscious processing such that a patient's narrow focus on perceived deficiencies or perceived slights by other people inhibits dissonant information that might be contradictory and of a more positive nature and excludes it from subjective experience (Beck, Rush, Shaw, & Emery, 1979; Brewin, 1988; Dixon, 1981). Negative self-talk and negative core assumptions can therefore be seen as not only tied to the content of information processing or cognition but also the particular process of cognition. There is likely also a companion narrowing of focus in major depressive disorder from processing external information and maintaining alertness to processing internally generated thoughts and feelings. PET scan studies of unipolar depressed patients reported by Posner and Raichle (1995) have documented striking increases in cerebral blood flow in the frontal cortex, particularly in the left frontal lobe and left prefrontal cortex, and decreases in blood flow in the parietal and posterior temporal lobes compared with matched control subjects. Even more striking, when normal subjects are asked to contemplate sad thoughts or memories to induce a sad mood, the same left prefrontal blood flow increases are seen.

One central focus in the cognitive-behavioral treatment of depression is to understand and uncover unarticulated core assumptions that guide patients' attitudes and beliefs about themselves and act as a filter or template to assign values to everyday experiences, core assumptions that typically patients are not aware or are only minimally aware of having (Beck et al., 1979). These core assumptions may be formed in large part by the automatic processing of information, and would therefore be expected to exist without the awareness of the patient. Selective attention or selective abstraction is seen in depressed and anxious patients in which a single detail taken out of context becomes the conceptual frame for the entire experience and other sometimes more salient

information is ignored. This phenomenon has a corollary in the laboratory in the automatic attention responses reported by Schneider and Shiffrin (1977) in which extensive experience with a particular stimulus in a visual search task led to subjects immediately attending to that stimulus irrespective of the complexity of the target display or the memory load of previous learning trials.

There have been a number of cognitive models that have been proposed which have elaborated upon or modified the simple, dichotomous conscious/automatic processing model (Schacter, 1989; Schacter et al., 1988) and taken into account the relationship between phenomenal consciousness, procedural memory and procedural systems, declarative/episodic memory, and executive functions, including models that embrace multiple levels and types of consciousness (Block, 1995). Since the late 1980s, numerous cognitive psychologists, neurophysiologists, mathematicians, and computer scientists have been actively constructing and elaborating a wide variety of computational, neural network models to account for the properties of consciousness, attention, memory, and higher cognitive processes (Alavi & Taylor, 1993; Gray, Buhusi, & Schmajuk, 1997; Rolls, 1997; Sun, 1997; Taylor, 1997) which may ultimately have a profound affect on the way clinical psychologists conceptualize psychopathology.

#### **1.11.1.4 Neural Network Models and Classical Conditioning**

The cognitive abnormalities characteristic of the acute phase of schizophrenic psychosis (hallucinations, delusions, fragmentation, loss of selective attention) have been conceptualized by Hemsley (1987) as reflecting a breakdown in the normal control of current information processing by disruption of the normal ability to use stored regularities as a guide to current information processing. Information that is typically processed in an automatic, nonconscious mode is then directly experienced and processed in a controlled or conscious mode (Gray, Feldon, Rawlins, Hemsley, & Smith, 1991; Schneider & Shiffrin, 1977). There is a body of evidence to suggest that a disruption in latent inhibition may account for this phenomenon.

Latent inhibition involves a classical conditioning paradigm which embraces elements of cognitive processing (Dunn, Atwater, & Kilts, 1993; Ellenbroek & Cools, 1990; Killcross, Dickinson, & Robbins, 1994; Weiner, 1990). Briefly stated, latent inhibition is the difference in efficacy of conditioning between conditioned

responses evoked by pre-exposed conditioned stimuli and conditioned responses evoked by nonpre-exposed conditioned stimuli (Gray et al., 1997). Latent inhibition is decreased or blocked by amphetamine treatment in animal models (Solomon & Staton, 1982; Weiner, Lubow, & Feldon, 1984) and in human subjects (Gray, Pickering, Hemsley, Dawling, & Gray, 1992; Thornton et al., 1996). This block is reversible by injection of haloperidol (Gray et al., 1997).

A mathematical neural network model for latent inhibition has been developed (Buhusi & Schmajuk, 1996; Gray et al., 1997; Schmajuk, Lam, & Gray, 1996) which explains the cognitive symptoms seen in acute schizophrenia as the result of a blockade of latent inhibition, which is thought to be related to disruption of dopaminergic pathways beginning in the nucleus accumbens and cascading through the nucleus reticularis thalami and thalamocortical sensory relay nuclei. This model mathematically describes elements of the neural network theory of latent inhibition reported by Schmajuk (1997), which involves interaction between trace short-term memory for conditioned stimuli, attentional long-term memory for conditioned stimuli, associative long-term memory for conditioned and unconditioned stimuli, and intermediate-term memories for conditioned and unconditioned stimuli and their predictions.

#### **1.11.1.5 Multiple-level Neural Network Models**

Taylor (1997) has modeled a two-stage neural network that accounts for blindsight and neglect which is very similar conceptually to previously described conscious/automatic processing models, and has proposed a third stage to this model (the ACTION net) roughly corresponding to those cortical areas involved in executive functioning, planning, reasoning, and working memory (Brodmann areas 46 and 9). This third network is of particular interest for clinical psychologists because it involves neuroanatomic structures related to affect as well as memory and executive functioning. This model is based upon the assumption of the basal ganglia (striatum and globus pallidus) acting in a disinhibitory manner on thalamocortical recurrent loops. The ACTION net encompasses five networks (corresponding to the five frontal loops identified by Alexander, DeLong, & Strick, 1986) involving motor, premotor, and supplementary motor cortex (action sequences), limbic (affect), orbitofrontal (social), frontal eye fields (eye movements), and dorsolateral prefrontal cortex (cognition and attention). Although such a model can account for emotional tone as global activation from the limbic system and does allow for some spec-

ulation about the relationship of awareness, attention, consciousness, memory, and mood, this model does not include true integration of limbic structures, hippocampus, and related areas. Of great value, however, is the compatibility of this model with neural net models of the amygdala (Monchi & Taylor, 1995, 1997), a cortical structure that appears to have a central role in depression.

PET scan studies reported by Posner and Raichle (1995) have confirmed increases in blood flow in depressed patients in a group of cells in the amygdala and the medial dorsal nucleus of the thalamus and decreases in the caudate nucleus. Examination of PET data from a second study of patients with previous episodes of severe depression now in remission (Drevets et al., 1992) revealed blood flow increases in the amygdala with normal flow in the thalamus and the caudate, and depressed patients in remission who were also actively treated with antidepressant medication had normal blood flow in the amygdala. One role of the amygdala in cognition appears to be the affective labeling or assignment of emotional significance to experiences in long-term memory. Posner and Raichle (1995) have concluded that if the increased blood flow in the amygdala seen in depressed patients represents a disruption in normal amygdala function, this dysfunction could result in a negative emotional state assigned to all experiences resulting in depressed mood or could result in anhedonia due to the prevention of positive emotional labels assigned to events, thus tying together neural substrate, memory, cognition, and mood.

As noted earlier, left prefrontal blood flow increases have been noted on PET scans during bouts of depression and also during induced sadness (Drevets et al., 1992). If the same dysfunction hypothesis is applied to this finding, there is again the implication that cognition, memory, and mood are inter-related. It is well known that patients with structural damage to the left prefrontal area demonstrate problems with set shifting and rigidity of thinking, and describe having difficulty shifting problem-solving strategies even when they know that a given strategy is incorrect. Posner and Raichle (1995) have suggested that disruption in left prefrontal blood flow in depressed patients could lead to this same sort of difficulty in the set shifting of emotions and thoughts, leading to depressive rumination. Memory and mood are again intertwined in this process. Induced negative mood increases in left prefrontal blood flow occur coincident with the conscious association of thoughts or emotion with information held in long-term memory. Depressive rumination and the experience of



incessant negative thoughts also likely involve making conscious associations in working memory with information retrieved from long-term memory. There is increasing evidence from a number of laboratories that memory and mood are inter-related.

## 1.11.2 MOOD AND MEMORY

### 1.11.2.1 Effects of Affective States

Cognitive models of negative mood (e.g., Blaney, 1986; Carver & Scheier, 1990) assume that there is a two-way interaction in cognitive processing involving memory and the regulation of emotions, that is, (i) the way an individual reacts to an affective stimulus is influenced by previously acquired memories, and (ii) the way we remember previous events is influenced by affective states that prevailed when we encountered those events.

Accumulated evidence suggests that memory processes are influenced by affective states during which memories were acquired. Since the 1970s interest in the study of memory performance in the context of negative mood and depression has been extended to laboratory settings by both cognitive and clinical psychologists. Laboratory studies usually involve experimentally inducing a depressed, elated, or neutral mood to observe its effect on memory performance.

The most frequent method used to induce depressed mood is the Velten Mood Induction procedure (Velten, 1968). In this method, mood is induced by having subjects read statements that are related to a depressed or elated mood. Other methods, such as hypnosis, imagery, or music, have also been used in the experimental induction of mood.

The effect of a depressed mood on recall has been demonstrated in experimental settings by several researchers. Leight and Ellis (1981) conducted two such experiments. In the first they investigated the effect of depressed mood on recall and transfer of training. Subjects received depressed, elated, or neutral mood induction. Then, they were trained on a letter sequence task. Twenty-four hours after the training session, subjects received the same training plus a transfer task in which they were presented with similar letters, but different spatial groupings. Overall, subjects' recall improved over trials. However, subjects in the depressed condition showed a slower improvement rate than subjects in the elation condition. In the transfer phase, subjects who previously received depressed mood induction recalled significantly fewer letters than subjects who never received depressed mood induction.

Subjects who received neutral mood induction were able to recall more letters than subjects who received depressed mood induction. Depressed mood during learning in other experiments led to a lower rate of recall, a lower rate of learning of letter sequences, and lower number of letters chunked when compared with neutral mood. These findings were interpreted by Leight and Ellis as an indication of deficits in strategy learning (Leight & Ellis, 1981).

The effect of depressed mood induction may also be dependent on the nature of tasks being performed, for example, difficulty levels. In a series of experiments, Ellis, Thomas, and Rodriguez (1984) found poorer performance in free recall among subjects who received depressed mood induction vs. subjects who received neutral mood induction. A sentence completion task was used. In this task subjects were asked to complete a sentence with a suitable word. The sentences were either easy or difficult. It was found that depressed mood induction was associated with worse performance relative to performance during a neutral mood condition when subjects were asked to recall words used with difficult sentences. These authors also found that when subjects had the opportunity to process the information semantically using strategies such as elaboration, they were able to show better performance, supporting a resource-allocation model (Ellis et al., 1984). The model suggests that depressed mood has debilitating effects when the encoding processes required are demanding, difficult, and complex.

Similar conclusions were reported by Hasher, Rose, Zacks, Sauft, and Doren (1985), who indicated that memory deficits in depression may be attributed to the ineffective use of processes that require cognitive effort, such as organization of information, imagination, rehearsal, and other mnemonic devices. According to these authors, encoding operations require attentional capacity which could vary, depending on whether these operations are automatic or effortful. Depression is one of the variables thought to limit the availability of attentional capacity to perform effortful operation. Effects of depressed mood would therefore be evidenced on memory tasks that require effortful processing.

Hertel and Hardin (1990) proposed that memory deficit in depression may be attributed to the lack of cognitive initiative in depressed individuals, due to less initiative in using strategies that would help them remember. When these subjects are directed to use such strategies, memory performance should improve. To test these hypotheses, Hertel and Hardin (1990) conducted three experiments. They compared the performance of college

students who received depressed mood-induction with that of subjects who received neutral mood induction. The task used in these experiments involved presenting subjects with two lists of homophones in the context of questions that reflected the less typical meaning of the homophones, and with added questions to disguise the target words. Spelling and recognition lists were constructed from the two lists of homophones. After receiving the mood induction procedures, subjects were asked to spell new and old homophones to test their memory of the old homophones. When subjects who received depressed mood induction were provided with spelling strategies, they performed as well as those who were in the neutral mood condition. In addition to their argument about the role of cognitive initiative in memory, Hertel and Hardin (1990) suggested that motivational differences between groups may exist. Subjects who received depressed mood induction may have had an excuse not to try hard (Hertel & Harden, 1990).

Failure to find memory deficits among subjects who received depressed mood induction was reported by other authors, however (e.g., Hasher et al., 1985; see also Ellis, 1985).

In summary, three main hypotheses have attempted to address the effect of depressed mood on memory. The first is the hypothesis that poor recall of information learned when subjects have a depressed mood can be attributed to poor learning strategies and lack of flexibility (Leight & Ellis, 1981). The second hypothesis indicates that people who are in a depressed mood have less resources to process information, that is, according to the resource allocation model (Ellis et al., 1984), when the task requires more processing effort, depressed subjects show poorer performance than if the task is easy. Third, Hertel and Hardin (1990) suggested that low level of cognitive initiative is what causes poor memory performance in depressed individuals.

Although the experimental literature seems to support a detrimental effect of depressed mood on memory, some questions still need to be addressed. For example, how can we differentiate between memory deficits in depression and state-dependent effect? Also, to what extent could a state-dependent effect account for the effect of depressed mood on memory? These questions seem to be important when we take into consideration the fact that studies have assessed memory performance in states that may have been different from the state when the learning took place. That is, subjects who learned materials while they were depressed and then were tested in a neutral mood may have shown a poor memory performance as a

result between the mismatch of the mood state under which they recalled and the mood state under which they learned these materials.

### **1.11.2.2 State-dependent Learning**

In this section hypotheses are discussed that address the effect of mood on memory in the context of mood state-dependent learning (e.g., Bower, 1981). One hypothesis that has been introduced to account for poor memory performance in depression is the mood-congruence hypothesis. This hypothesis has received intense attention from scientists interested in both cognitive processing and emotions (see Blaney, 1986; Matt, Vazques, & Campbell, 1992; Singer & Salovey, 1988). In its basic form, the hypothesis states that learning and remembering stimuli will be easier when there is a match between affective tone of the learned materials with the receiver's current mood state than when there is a "mismatch." That is, performance is more efficient when the recall occurs in a mood state that is similar to the mood state when the learning process took place (Singer & Salovey, 1988), possibly due to efficiency in mnemonic processing (Blaney, 1986).

#### **1.11.2.2.1 The Network Theory of Affect**

The Network Theory of Affect, introduced by Bower (1981), attempts to account for the mood-congruency effect. One of the salient factors of this theory is the importance of mood state during the learning process. The mood state is thought to serve as a cue when recalling this information and included in associations with coincident events. When this emotion unit is activated, it is used as a cue to retrieve its associated events.

According to this theory, emotions may be viewed as nodes providing points of contacts within a semantic network. Nodes related to a particular affective state are connected to other nodes of related moods that provide representations of events or connections that were formed during learning. In this manner, nodes related to anxious state, for example, are more likely to be connected with anxiety-related concepts and experiences (e.g., fear, heightened autonomic arousal, and muscular activity, etc.) During a particular prevailing mood, nodes that tap these mood states are activated, and they in turn activate other related nodes. Information congruent with prevailing mood states acquires stronger processing than incongruent information according to this theory. This leads to elaborate processing at both encoding and retrieval phases, and this in turn contributes to a better ability to recall mood-congruent than

mood-incongruent information (Gilligan & Bower, 1984; Singer & Salovey, 1988). Encoding information that is congruent with current mood state supposedly occurs through nodes that connect a widespread network, which leads to a dense and elaborate representation of incoming information. In contrast, information that is incongruent with current affective state taps a relatively less elaborate and narrower scope of network representation. A similar pattern of processes occurs during retrieval, where retrieval of mood-congruent information is facilitated by biased search of relevant activated nodes, which leads to increased accessibility of mood-congruent materials. Incongruent materials therefore are relatively less accessible for search and retrieval (Bower, 1981; Riskind, 1989).

Studies conducted to evaluate this hypothesis have observed effects of mood-congruency on retrieving learned information (cf. Riskind, 1989; Singer & Salovey, 1988). These studies were done both in normal participants exposed to different types of mood induction procedures and in individuals who were classified as depressed (cf. Blaney, 1986).

Bower (1981) conducted a related study with college students. Either a depressed or a happy mood was induced using imagination guided by hypnosis suggestions. The main finding of this study supported the mood state-dependent hypothesis. Subjects who learned a word list in the depressed mood were able to recall more words if they were tested in the same depressed mood than if they were tested in the happy mood. A similar pattern of effect was found with subjects who learned the list in a happy mood.

Eich and Metcalfe (1989) went further to suggest that mood state-dependency influences one's memory for internal more than external events. Memory for external events is defined as a memory based on sensory stimuli that are brought into awareness from external sources through perception. Memory for internal events, on the other hand, is defined as those events created through mental processes such as imagination, reasoning, thoughts, etc. (Eich & Metcalfe, 1989). They conducted an experiment with college students in which they asked subjects to generate or read 16 items while in either a happy or a sad mood. The mood-induction technique used in that experiment was continuous music. Subjects were tested two days later. The mood during the testing session was either matched or not matched with the mood during learning. The findings showed a significant impairment in remembering generated items when the mood was shifted. The effect of the mood shift was significantly less when recalling read items. The authors indicated that

relying on internal rather than external resources in creating and processing cues for retrieval makes memory more mood dependent.

Mathews and Bradley (1983) used Velten mood induction and music to induce either a depressed or a neutral mood. Positivity of personal memories was used as a dependent variable in that study. Subjects who received depressed-mood induction recalled more negative memories than subjects with a neutral mood. Similar results were found by Forgas, Bower, and Krantz (1984) who used hypnosis to induce either an elated or a depressed mood. Subjects were asked to remember an interaction task they had recently completed. Subjects who received a depressed mood induction recalled more unpleasant and stressful aspects of that experience than subjects who received an elated mood induction. On the other hand, subjects in the elation condition recalled more memories related to the friendly and relaxed interaction they had during that task.

Riskind, Rholes, and Eggers (1982) asked 52 undergraduates to recall four pleasant and four unpleasant life experiences. Then mood induction procedures were administered using the Velten mood induction technique. Subjects received either depressed or elated mood induction. The latencies for recalling positive and negative experiences were compared across groups. Subjects in the elation condition were significantly faster in recalling positive experiences than subjects in a depression condition.

While many studies have provided data to support the mood state-dependent hypothesis, some studies failed to provide such support (e.g., Schare, Lisman, & Spear, 1984; see Blaney, 1986). Schare et al. (1984) conducted two experiments using mood-induction procedures to induce depressed and neutral mood in a sample of college students. They used a word list learning task. Subjects were tested in either a matched or mismatched mood state compared to the original state in which they learned the lists. None of the experiments demonstrated a state-dependent effect.

#### **1.11.2.3 Depressed Mood and Cognitive Performance**

Studies conducted both in experimental settings with normal subjects and in clinical settings with depressed patients have provided evidence to support a negative effect of depressed mood on memory performance. Several theories have attempted to explain the cause of this effect. The main themes of these theories indicate that memory deficits in depression could be attributed to the mood state-dependent effect and the severity of the

psychopathology of depression (Johnson & Magaro, 1987). The state-dependent theory attributes the poor performance in depressed individuals to the incongruity of the mood state under which subjects recall materials that may have been learned in a different mood state. Other theories propose that poor learning strategy and lack of cognitive initiatives may be the reason for the poor memory performance. The mood state-dependent effect is explained by pointing out the importance of contextual cues that could be provided in a particular mood state. These cues help in activating particular memory associations that are congruent with the current mood state. It is also believed that the depressed mood state activates a negative self-schema that results in selective encoding and retrieval of materials that match the mood state. Depression may affect memory through disrupting the "normal" pattern of organizing and processing information, the low level of effort exerted when encoding and storing information, or may be a product of a conservative response style and a lack of motivation to learn and process information efficiently.

The state-dependent effect may contribute to memory deficits in clinically diagnosed depression. Weingartner, Miller, and Murphy (1977) used a free association task with eight manic-depressive patients to investigate state-dependent memory in depressed patients. Subjects were asked to recall associations in either the same or a different mood state. Patients showed better performance when they were asked to recall the associations in a similar state to their learning state. According to these authors, mood state did not only affect the way these associations were encoded but also provided help to determine how subjects could retrieve the relevant cues of that state.

The mood-congruence hypothesis (discussed earlier) has also been tested in studies conducted with depressed patients (e.g., Clark & Teasdale, 1982; Gotlib, 1981; Matt, Vazquez, & Campbell, 1992; Tham et al., 1997). In some of these studies, depressed individuals were provided with different types of feedback about their performance. Subjects given negative feedback were able to remember these tasks better than when tasks were followed by positive feedback (Calev, 1996; Channon & Baker, 1996; Singer & Salovey, 1986). Johnson, Petzel, Hartney, and Morgan (1983) asked depressed and nondepressed students to recall the content of tasks on which they had been successful or unsuccessful. Both depressed and nondepressed groups had a similar amount of success or failure, since the success or lack of success was under the experimenters' control. The authors found that depressed subjects were more able to recall

failure tasks than nondepressed subjects. Nondepressed subjects compared with depressed subjects remembered more "successful tasks."

Another type of material used to investigate the mood-congruence hypothesis is self-reference statements or positive/negative materials. Clark and Teasdale (1982) conducted a study with depressed patients who were tested twice on different occasions to tap the diurnal variation in their mood. Materials with a depressing tone remembered while subjects were more depressed were compared with similar materials remembered while patients were in a less intense depressed mood. The results showed that more "unhappy" and less "happy" materials were remembered while patients were in the more depressed time. When patients were in the less depressed occasion, the results were reversed with the more happy and less unhappy materials remembered by patients.

Why do depressed patients show memory deficits? Weingartner, Cohen, Murphy, Marrello, and Gerdt (1981) investigated the strategies depressed individuals use to process information. In one experiment they found that depressed patients did not use elaborative strategies to encode information. Based on this and other experiments, these authors concluded that depressed patients were unable to benefit from encoding operations that would help them organize material and remember it. This conclusion indicates that a processing deficit may exist in depressed individuals. This deficit may be attributed to ineffective organization in encoding (Johnson & Magaro, 1987). This conclusion is similar to the conclusion reported by researchers who have investigated this issue in laboratory settings (Hasher et al., 1985).

Some authors have argued that short-term memory deficit may be the cause of overall memory deficits in depression (e.g., see Koh & Wolpert, 1983). This argument was based on the finding that deficits were found in short- but not long-term memory in depressed patients compared with nonpsychiatric patients (see Johnson & Magaro, 1987). However, inconsistent results were also reported. For example, Henry, Weingarten, and Murphy (1973) found long- but not short-term memory deficits among a group of bipolar and unipolar depressed patients when they performed tasks while they were depressed compared with their performance when they were not depressed.

Other factors not related to cognitive processes were also suggested to explain memory deficits among depressed individuals. One of these factors is the apparent lack of high motivation in at least some depressed individuals (Cohen, Weingartner, Smallberg, Pickar, & Murphy, (1982). Effort is necessary to perform

memory tasks that require elaboration and active rehearsal (Johnson & Magaro, 1987), but depressed individuals seem to lack the ability to maintain a high level of persistency (Miller, 1975) which may affect their performance. To investigate the relationship between memory deficit in depression and motivation, Cohen et al. (1982) conducted a study with 11 depressed patients and five normal controls. They demonstrated a negative correlation between memory performance and severity of depression and also between severity of depression and the tendency to exert effort to perform this task. A positive correlation was found between memory performance and the effort spent to perform the task.

Another factor that has been suggested to account for memory deficits among depressed individuals is the tendency to be cautious in giving responses when they are examined. Johnson and Magaro (1987) argued that this is not a cognitive deficit but rather a tendency to respond with a certain level of confidence that may make depressives conservative in their response to testing situations. According to this argument, memory may be intact and the information needed may be available in the memory store. The problem is instead related to the restrictions depressed people impose on what they should report, and how sure they are regarding the “accuracy” of the information they report (see Johnson & Magaro, 1987).

Theories that focus on the state-dependent effects and the mood-congruency effects assume that learned materials are available in memory. The failure to remember these materials could be attributed to the lack of sufficient retrieval cues that subjects used when they encoded these materials. When subjects are in a mood state that is similar to the mood state in which they learned materials, they may benefit from this mood by getting better access to associations in memory. Similarly, materials that are congruent with a particular mood may be processed more effectively because of the availability of many cues that could enhance the encoding process and retrieval. One could also infer that subjects who learn materials which are congruent with their mood would tend to have a lower threshold for encoding these materials and for chunking them in units that are easily accessible.

Theories that emphasize the effect of poor encoding strategies and low effort to process information effectively assume that materials cannot be remembered because they were not learned or they are not available in memory. Therefore, a depressed individual who could not recall particular events from a previous day may not have this information in their memory any more, or may not have stored this information in the first place. On the other hand, a state-

dependent theorist may argue that this individual may have been in a different mood when they learned about these events, and that they would more likely to remember them if they were in a similar mood state. All these inferences still need to be directly tested.

In summary, to understand the effect of depressed mood on memory, it may be useful to consider both the state-dependent hypothesis and theories that focus on encoding deficits. It is also important to consider the severity of depressive symptoms as a contributing variable. That is, while the mood state-dependent phenomenon may explain some of the findings when using mood induction with normal subjects, memory deficits among severely depressed individuals involve more complex mechanisms. These mechanisms may include poor learning strategies, less effort exerted in encoding information, lack of cognitive initiative to use strategies, and a conservative response style, all of which are associated with the level of severity of depression.

### 1.11.3 COGNITION–EMOTION INTERACTIONS

#### 1.11.3.1 Cognitive Factors

The cognitive model of behavior emphasizes the contribution of cognitive functioning to any emotional distress. Cognitive processes such as perception, reasoning, and thinking, represent substrates for mental health. Faulty processes, such as misperception of social situations, negative thinking, evaluation of oneself without sufficient data, and patterns of attributing failures to oneself, contribute to negative affect and increase the risk for psychopathology.

In addressing cognitive contributions to emotional behavior, it is important to distinguish between various terms used to describe cognition, including such terms as cognitive processes, cognitive structure, cognitive content, and cognitive products (Ingram & Kendall, 1986). Cognitive processes are the operations performed to receive, store, transform, and coordinate information. Cognitive structures are the organization of information. Cognitive structures influence our perception of surrounding situations and events. The cognitive content is the actual information that we store and process, including what we tell ourselves via self-talk. Cognitive products are those outputs that result from the various cognitive processes, including conclusions one may reach (Kendall & Hammen, 1995).

Relating a certain style of processing to a mood state may be better accomplished with the distinction between these terms in mind. For

example, the way one explains an event (i.e., attribution) is a cognitive product that may result from various cognitive processes. The way these cognitive processes are conducted would depend on the content and the cognitive structure for that person. The attribution therefore may vary in persons who are anxious, depressed, or in a happy mood.

This pattern of interaction between cognition and emotion is well accepted. However, the question of whether cognition and emotion should be considered as separate systems is still strongly debated. Gray (1990) articulated a model in which he described neurobiological systems that may underlie both cognition and emotion. He argues that the overlap between the two systems is very strong, to the extent that it may be impossible to distinguish between substrates of cognition and substrates of emotion.

Other emotion researchers, on the other hand, argue that separate pathways can be delineated to account for cognition and emotion, respectively (Izard, 1992; LeDoux, 1992; Panksepp, 1992). This school of thought also acknowledges the close interaction between these two brain systems. Indeed, the separation between the two tends to be blurred in certain conditions (LeDoux, 1989, 1992). One example of such conditions is when rapid unconscious computations of a situation that is survival-related have to take place. Such a situation, while it involves the emotional system, is also accepted as a type of cognition. The distinction is more pronounced when the process involves rational, analytic processing not related to basic survival efforts.

Most of the actively researched theories in clinical psychology deal with both cognitive and emotional aspects of behavior in a complementary fashion. To this end, several cognitive theories of emotion have been proposed and have enjoyed significant attention. These theories generally agree that a stimulus when perceived is linked or elaborated on by previous experiences, and based on this elaboration an emotional response ensues. An emotional response can be discrete or a mix of multiple emotions, with the most widely accepted emotions being anger, happiness, sadness, fear, and disgust (Ekman, 1973).

### **1.11.3.2 Cognitive Appraisal**

Lazarus (1991) discussed the generation of emotions as a result of cognitive appraisals. Lazarus' model of emotion proposes that events we encounter are thought over, and responses to each event are selected consciously from a range of options. Lazarus' model of emotion empha-

sizes perceived control over the environment as a critical determinant of the psychological impact of events.

Lazarus and Folkman (1984) have described how interactions with the environment generate emotions, noting the continuous evaluation process of events that people encounter. This evaluation, termed appraisal, is classified as familiar or unfamiliar and as threatening or nonthreatening. Various cognitive and behavioral strategies to deal with these challenges are also developed. Emotions experienced in these situations provide information about whether these strategies are successful and motivate additional behaviors.

According to Lazarus and Folkman (1984), there are two stages of appraising situations. The first, called primary appraisal, involves evaluating the threatening aspects of the situation and is geared towards developing a plan to deal with the situation. The second stage, called secondary appraisal, involves evaluation of the options available for dealing with the challenging situation. This appraisal determines the cognitive, behavioral, emotional, and physiological responses to environmental events.

Factors that influence primary appraisal of an event include one's beliefs about how the world should work and one's own commitments to given courses of action. Events that are incongruent with beliefs and commitments in life are perceived as threatening events. When an event is appraised as threatening, it invokes a simultaneous emotional reaction. This reaction signals alarm and motivates various behavioral and psychological responses along with physiological activation to enable one to undertake planned coping behaviors (Lovullo, 1997). On the other hand, the process of secondary appraisal focuses on efforts that might be used to "cope" with situations at hand. The efforts may include coping strategies and coping behaviors, and can be overt or covert.

Lazarus and Folkman (1984) classify coping responses into two types: problem-focused and emotion-focused strategies. Problem-focused strategies are directed at the source of the problem itself, with efforts geared towards gaining information, changing events, and modifying beliefs and commitments. Problem-focused strategies enhance one's awareness and knowledge about the situation, and therefore enhance one's cognitive and behavioral coping resources. These resources help in reducing the threat value of the event. On the other hand, emotion-focused strategies direct efforts towards changing the psychological reactions to an event. This change is geared towards limiting the degree of emotional distress that may be caused by a stressful event.

### 1.11.3.3 Attribution and Affect

For a subjective emotional state to take place, both peripheral and central inputs contribute. In the early 1960s Schacter and Singer (1962) described how these sources of physiological change contribute to emotion. According to these authors, an emotional response is a product of two factors: perception of peripheral physiological arousal and the label given to this arousal. The specific emotional response is then based on the cognitive appraisal of the source of arousal, whether it is attributed to external or internal stimuli.

In their original study, Schacter and Singer (1962) independently manipulated epinephrine and autonomic activation on the one hand and manipulated attribution of the source of arousal on the other. Subjects were injected with either epinephrine or placebo to induce physiological arousal. Subjects who were injected with epinephrine received no information or were misinformed. Subjects were not told that they would feel either happy or angry. The prediction was that subjects who were aroused but were not able to attribute this arousal to epinephrine would express stronger emotions. The findings supported the model, although not definitively.

In the late 1970s, subsequent studies showed that increased sympathetic arousal contributes to evaluating situations in a negative manner (Marshall & Zimbardo, 1979). However, the role of Schacter and Singer's (1962) two factors in predicting interactions between situations and perception of autonomic state has also been criticized (Erdmann, 1983). One area of criticism has been the supposed overestimation of the role of arousal in the interaction between arousal and attribution. The role of arousal seems to be more accepted when it is perceived as providing feedback which intensifies the experience of emotion (Reisenzein, 1983).

### 1.11.3.4 Psychobiological Substrates

The study of cognition–emotion interaction may be further advanced by understanding the biological underpinning of such interactions. Recent efforts in cognitive neuroscience research have generated excitement about the value of a multidisciplinary approach to study emotion and cognitive processes. Indeed, most of the prominent theoretical accounts of emotion–cognition interaction acknowledge the importance of incorporating physiological knowledge in understanding this interaction and propose neurobiological substrates for it. The next section includes a brief discussion of an account of this interaction.

The way cognition influences our emotion, or emotion influences our cognition, is influenced by processing that takes place at multiple physiological and anatomical levels. Gray (1990) discusses the prominent role of the hippocampal and amygdalary systems. He proposes the presence of three basic emotional systems that include a behavioral approach system, a fight-or-flight system, and a behavioral inhibition system. Each system is influenced by a specific neuroanatomical structure. The hippocampal system (particularly the septal area) is the biological substrate for behavioral inhibition, while the amygdalary system is the substrate of the fight-or-flight system. Both biological systems, according to Gray (1990), may be involved in the behavioral approach emotion. Other systems, such as brainstem and neocortical systems, may also be involved in organizing approach behavior.

The anatomical delineation of these hypothesized interactions can be further appreciated by reviewing the interconnections of the hippocampus, the amygdala, and other emotion-related brain systems. The hippocampus has various connections with cortical and brainstem structures. The hippocampus is also a primary structure involved in the conscious recollection of past information (Squire et al., 1992), and in processing spatial, contextual, and configural information (Eichenbaum, Otto, & Cohen, 1992; Rudy & Sutherland, 1995). The hippocampus provides inputs to structures involved in emotional processes, particularly the amygdala (LeDoux, 1993). The hippocampus interacts extensively with the amygdala and other stress effectors, especially the hypothalamic–pituitary–adrenocortical (HPA) system (Chrousos & Gold, 1992). Projections from the forebrain to the hippocampal formation also indicate involvement of the latter in attention processes.

Advancing this information to explain abnormal behaviors, Gray (1990) notes that lesions in the septal area produce behavioral effects that resemble those seen as a result of antianxiety drugs. Based on this finding, it was proposed that anxiety may be related to functional changes in the septohippocampal system (Gray, 1990). Additional cited support for this hypothesis includes the heightened sensitivity of this system for signals predicting punishment and innate fear cues. This sensitivity results in inhibition of behavior and enhancement of autonomic activity.

The integration of emotion and cognition also involves the amygdala. The amygdala is viewed as the primary brain structure involved in emotional behavior and expression (Aggelton, 1992). It is a key structure in the memory of

emotionally-relevant information (LeDoux, 1993). It plays a significant role in providing emotional meaning to a stimulus. Research with brain damaged humans has demonstrated the importance of an intact connection of the amygdala with other parts of the limbic system for the perception and expression of emotion (Bechara et al., 1995). For example, bilateral lesions in the amygdala prevent conditioned autonomic responses to aversive stimuli, in spite of an intact memory of declarative information about the presented stimuli (Bechara et al.).

Like the hippocampus, the amygdala receives projections from and provides inputs to a wide range of sensory and cortical structures (Derryberry & Tucker, 1992). The amygdala exerts a facilitatory influence on HPA activity (Feldman, Conforti, & Weidenfeld, 1995) and on the autonomic and behavioral apparatus associated with stress (Weiss et al., 1994). It projects output to various brainstem structures including the pontine reticular formation and the brainstem aminergic nuclei. The pontine reticular formation facilitates the behavioral and biological response. The locus coeruleus and raphe nuclei in the brainstem send their aminergic fibers to the cortex, limbic structures, and to the spinal cord. This influences the level of cortical activation and the interaction of descending cognitive output, emotional experience, and autonomic and motor outflow.

Cortical structures also influence both cognition and expression of emotion. An important area of research that has received increasing attention is the effect of cortical hemispheric lateralization on emotional perception and expression. The right hemisphere is generally thought to play a more prominent role in nonverbal, spatial, and affective processes than the left hemisphere. The latter is more prominent in verbal, sequential, and logical reasoning (Borod, 1992). The dominance of the hemisphere in emotional processing seems to be specific to negative affect, although it has been suggested that this hemisphere is also involved in emotional perception and expression, regardless of the specific affective tone (Davidson, 1993). Relative to the left hemisphere, the right hemisphere has more direct connections with subcortical structures involved in emotional expression (Gainotti, Caltagirone, & Zoccolotti, 1993).

A further delineation of the role of right vs. left hemisphere assumes that the left hemisphere is more involved in the expression and experience of positive affect, whereas the left hemisphere is more involved in the perception of positive affect and both the perception and expression of negative affect (Borod, 1992). Related to this, the left hemisphere may be

responsible for affect-related approach behavior (Fox, 1991). This specialized dominance seems to occur particularly in the anterior area of the cortex, whereas the posterior region seems to be associated with cognitive-perceptual processes (Davidson, 1993). Consistent with this hypothesis, studies of right hemisphere lesions have documented a steeper decline in sympathetic responses to stimuli compared with those noted in studies of left-hemisphere damage (Tucker & Williamson, 1984).

This line of work has implications for understanding the association of mood states, cognitive performance, psychopathology, and frontal lobe activation. Intra- and interhemispheric differences in EEG activity have been found to be related to depressed mood disposition and withdrawal in clinical and nonclinical populations (Davidson, 1993; Fox, 1991).

#### **1.11.4 COGNITIVE MODELS OF ANXIETY**

##### **1.11.4.1 General Cognitive Models of Anxiety**

Behavioral techniques such as desensitization, flooding, and response prevention have been historically effective treatments for anxiety disorders, but recent cognitive-behavioral therapy methods, in combination with behavioral techniques, have demonstrated superior efficacy (Brewin, 1996). Purely cognitive techniques, however, may not be superior to purely behavioral methods (Rachman, 1993). There are some commonalities in the mechanisms underlying the effectiveness of cognitive-behavioral therapy for anxiety.

Rachman (1990) noted several ways of acquiring fear, either by direct exposure to aversive or traumatic stimuli, by observing others displaying fear, or by hearing or reading verbal or written information about fear and danger. Similarly, catastrophic misinterpretation of symptoms based on ignorance or incorrect beliefs can lead to panic disorder (Clark, 1988) and prolonged post-traumatic stress disorder (PTSD; Ehlers & Steil, in press). In these latter cases, the representations in memory giving rise to feelings of fear may be verbally accessible and can therefore be altered by new verbal information. Many contemporary clinical approaches are based upon appraisal theories of emotion such as those of Lazarus (1991) and Weiner (1985), which emphasize the role played by conscious assessments of the meaning and cause of internal and external stimuli and of the coping resources available to deal with any prospective threat.

Verbally accessible knowledge may also be subject to nonconscious automatic processing, particularly when it is primed by a previous



stimulus. As noted by Clark (1988), catastrophic thoughts may come to mind so rapidly that patients are unaware of the interpretive process, which may in part explain panic attacks triggered in sleep. Also, almost by definition, automatic processing does not differentiate between verbally and situationally accessible knowledge (Brewin, 1996).

#### 1.11.4.2 Specific Phobias

Specific phobias are among the most common circumscribed anxiety disorders. Phobias may be based on unconscious representations, independent conscious representations, or some combination of the two. The particular pattern of response to different treatment approaches depends on the nature of the memories.

Most cognitive-behavioral treatment approaches to phobia include an emphasis on helping individuals undermine habits of cognitive or behavioral avoidance and to achieve habituation to a feared stimulus. More sophisticated cognitive approaches emphasize the importance of factors such as attention and memory (Dickinson, 1987) and conscious revaluation of the unconditioned stimulus (Davey, 1992, 1995).

Lang (1979) proposed that the representations underlying phobias, which he termed fear memories, contain three kinds of information: details of the location and physical characteristics of the feared situation (stimulus elements); details of the verbal, physiological, and behavioral responses that occurred in the situation (response elements); and an interpretation of the stimulus and response elements and their significance for the individual (meaning elements). Foa and Kozak (1986) propose that cognitive therapy for phobias involves changing information in the fear memory by first activating the memory and then incorporating new experiences arranged by the therapist into that memory (such as within- and between-session habituation to the feared situation).

Brewin's (1989) dual representation theory proposes that in therapy new memories are laid down as "situationally accessible knowledge." Patients in treatment are encouraged to expose themselves to as good an example as possible of what they fear and to experience the same sensations. In therapy, however, this experience culminates in mastery and the habituation of fear rather than in avoidance. Patients exposed to these feared situations are subsequently more likely to access this new memory than they are to access the original memory containing more fearful response elements and a more threatening meaning (Brewin, 1996).

#### 1.11.4.3 Panic Disorder and Agoraphobia

According to Clark (1988), panic arises from specific catastrophic misinterpretations of sensations such as tachycardia or dizziness. Treatment therefore involves a variety of techniques aimed at eliciting and challenging these misinterpretations. In contrast, Wolpe and Rowan (1989) suggest that at least in some patients there are no misinterpretations and that panic is instead the result of conditioning.

Panic provocation studies using CO<sub>2</sub> inhalation provide an analogue model for naturally occurring panic attacks (Gorman et al., 1988; Sanderson & Beck, 1989) and demonstrate the importance of cognition in mediating the emotional response to physiological sensations secondary to hyperventilation. CO<sub>2</sub> inhalation studies suggest that panic attacks are more than mere conditioned responses to a conditioned stimulus. Panic disorder patients who panic in this situation experience cognitive symptoms (e.g., fear of dying or losing control) in addition to somatic symptoms, whereas nonpanicking panic disorder patients and controls experience only somatic symptoms (Sanderson & Beck, 1989). Direct manipulation of cognitive variables also affects the likelihood of a CO<sub>2</sub> provoked panic attack. Rapee, Mattick, and Murrell (1986) found that panic disorder patients who were told which somatic symptoms to expect prior to the inhalation of CO<sub>2</sub> reported significantly less intense somatic symptoms and catastrophic thoughts compared to those who were provided no such explanation. Sanderson, Rapee, and Barlow (1989) found that panic disorder patients who believed they could control the flow of CO<sub>2</sub> (although in reality they could not) experienced significantly less anxiety and panic during the inhalation compared to those who did not believe this.

Rachman, Levitt, and Lopatka (1987) provide further evidence for the importance of cognition in the production of panic attacks. "Threat-relevant" cognitions accompanied the majority (74.6%) of panic episodes reported by panic disorder patients. When combinations of two or three symptoms were examined, Rachman et al. (1987) found that "no panics were reported unless there was an associated cognition." On the other hand, when the same combination of symptoms were accompanied by a fearful cognition, the episode almost invariably resulted in panic.

The biopsychosocial model of panic disorder (Barlow, 1988) conceptualizes the initial panic attack as a misfiring of the fear system under stressful life circumstances in physiologically and psychologically vulnerable individuals. Physiological vulnerability is defined as an

overly reactive or labile autonomic nervous system which may in part be determined genetically, consistent with the higher concordance rates of panic disorder seen within first-degree relatives (Crowe, Noyes, Pauls, & Slymen, 1983; Moran & Andrews, 1985; Torgersen, 1983). Psychological vulnerability is conceptualized as a set of danger-laden beliefs about bodily sensations and about the world in general. The concepts of uncontrollability (Barlow, 1988) and anxiety sensitivity (Reiss, Peterson, Gursky, & McNally, 1986) are central in this model of panic disorder and panic disorder with agoraphobia. Persons who panic tend to have stronger beliefs and fears of physical and mental harm arising from specific bodily sensations (Chambless, Caputo, Bright, & Gallagher, 1984; Clark et al., 1988; Holt & Andrews, 1989).

Barlow (1988) and Wolpe and Rowan (1989) emphasize the traumatic nature of the first panic attack and the consequent learning that takes place. The traumatic nature of initial panic attacks is apparent in the relatively high frequency of individuals seeking emergency medical help due to their misappraisal of the panic attack as signaling impending death. Consequently, fearful associations are likely to develop with the situational context and the physical sensations present at the time of the first panic. The concept of learning fearfulness of bodily sensations is based on Razran's (1961) account of interoceptive conditioning. This is a form of conditioning that is relatively resistant to extinction, and interoceptively conditioned fear responses are not dependent on conscious awareness of triggering cues. Panic attacks may seem to be uncued when in fact they are triggered by benign and subtle fluctuations in physical state (Barlow, 1988). For example, a slight change in heart rate might cue or elicit fearfulness in some patients in anticipation of experiencing a panic attack. Such internal fear cues can trigger more abrupt, and less predictable fear, which in turn leads to greater anticipatory anxiety about the recurrence of fear (Craske, 1991).

Anxious anticipation of panic increases the likelihood of its occurrence, since anxious arousal increases the availability of arousal sensations that have become conditioned cues for panic, in addition to increasing the degree of attentional vigilance for such cues (Barlow, 1988). In this manner, a cycle is established between panic and anxious apprehension about panic. It is hypothesized that sex-role behaviors and expectations and associated situational demands influence the degree to which agoraphobia emerges as a behavioral style in response to panic anticipation (Craske & Barlow, 1988).

Craske and Barlow (1993) describe a 15-session cognitive-behavioral treatment protocol that is specific for panic disorder and agoraphobia involving self-monitoring, cognitive restructuring, breathing retraining, applied relaxation, interoceptive exposure, and *in vivo* situational exposure.

#### 1.11.4.4 Social Phobias

Beck, Emery, and Greenberg (1985) describe a cognitive model of anxiety and phobias, including social phobias. The schema, the basic cognitive structure that guides the processing of information, serves as the core concept in this cognitive model. Schemas are sets of rules that classify, prioritize, and interpret incoming information to the person as well as facilitate the retrieval of relevant information from memory. Schemas are grouped into modes that create a cognitive set or processing bias that operates across situations. Anxiety-disordered individuals, according to Beck et al. (1985), typically function in the vulnerability mode, that is, the individual sees the world as a dangerous place in which they must constantly be vigilant to potential threat. As a result, neutral or mildly positive cues are misinterpreted negatively, while positive or safety cues are discounted or ignored. Memories of past successes or available coping resources are also underestimated or overlooked.

Social phobics are hypervigilant to cues that denote the possibility of negative evaluation by others. These cues may be situational, interpersonal, or internal. Social phobics devote excessive attentional resources to the detection of potential social cue threats (Hope, Rapee, Heimberg, & Dombeck, 1990; Mattia, Heimberg, & Hope, 1993; Smith, Ingram, & Brehm, 1983).

The self-presentational model of social anxiety (Leary, 1988; Schlenker & Leary, 1982) states that social anxiety occurs when an individual desires to make a particular impression on others but doubts they will be successful in doing so. How one might appear to others must be of importance and there must be apprehension about one's ability to engage in sufficient impression management. Individuals may be excessively motivated to create a particular impression because of a high need for approval, and several factors may affect a person's expectations about meeting impression management goals, including perceived or actual deficits in social skills, low self-esteem, and low outcome expectancies.

Hope and Heimberg (1993) describe a treatment protocol for cognitive-behavioral

group therapy of social phobia which consists of simulated exposures to feared situations, cognitive restructuring, and homework assignments for *in vivo* exposure.

#### 1.11.4.5 Post-traumatic Stress Disorder

Creamer, Burgess, and Pattison (1992) have developed a cognitive processing model for reactions to trauma that includes a feedback loop among intrusions, avoidance, and symptom levels. The model views the successful processing or integrating of the trauma as central to successful recovery. This model sees the cognitive processing mechanisms involved in recovery over time as occurring in five stages. Stage 1 is objective exposure. The major fact at this stage is the severity of the traumatic stressor. Stage 2 is network formation. This is determined primarily by subjective perceptions and meaning attached to the experience. Stage 3 is labeled intrusion. During this stage, the memory network is activated in an attempt to process and resolve the trauma-related memories. Stage 4, avoidance, is characterized by the use of escape and avoidance as coping strategies in response to intrusions. During stage 5, which is labeled outcome, recovery is achieved through network resolution processing. Such factors as pretrauma functioning and biological processes are not incorporated into this model. Chemtob, Roitblat, Hamada, Carlson, and Twentyman (1988) have developed a similar information processing model of PTSD.

The Jones and Barlow (1992) model considers PTSD as a variant of other anxiety disorders, particularly panic disorders, with anxious apprehension modifying or amplifying a predisposition to respond to stress with chronic autonomic overarousal or noradrenal lability. Anxious apprehension involves distorted processing of information along with extremely negative affect. The individual perceives the traumatic event and subsequent reexperiencing as unpredictable, uncontrollable, aversive events and reacts with chronic overarousal, hypervigilance, and narrowing of attention. This sets up a feedback loop in which hyperarousal, hypervigilance, and narrowing of attentional focus increase intrusive thoughts and reexperiencing.

Foa, Steketee, and Rothbaum (1989) and Rothbaum and Foa (1992) have articulated a cognitive-behavioral treatment approach for PTSD based on Lang's (1979) model of the emotional processing of fear, proposing that PTSD results from inadequate processing of the trauma stimuli, responses, and the meaning associated with them. Foa et al. (1989)

suggested that traumatic events create especially large and complex fear networks that are activated readily because of the large number of interconnections formed through conditioning and generalization. Associations that were once considered neutral or safe may now be connected with fear. This leads to a sense of unpredictability and uncontrollability that is important in the development and maintenance of PTSD. Treatment requires activation of the fear memory and incorporation of new information incompatible with the current fear structure, so that new memories are formed. The PTSD victim is asked to recall the initial trauma in detail and helped to process the memory until it is no longer intensely painful. This is combined with *in vivo* exposure to feared but objectively safe stimuli. Cognitive processing therapy (CPT) is a therapy model developed to treat the specific symptoms of post-traumatic anxiety and stress disorder in victims of sexual assault (Calhoun & Resick, 1993; Resick & Schnicke, 1992), which combines cognitive restructuring with exposure-based therapy.

#### 1.11.4.6 Generalized Anxiety Disorder

In generalized anxiety disorder (GAD), there are often complex sets of negative beliefs about the self or the external world that are activated in a variety of situations, and the underlying memory representations are thought to contain more abstract information than the specific memories underlying phobias. These representations are thought to consist of summaries of numerous aversive experiences produced by complex unconscious computations that abstract common meanings from repeated experiences (Brewin, 1996). In contrast to other anxiety disorders, evidence suggests that GAD has a more characterological presentation, although fluctuations in the course of GAD are often noted corresponding to the presence or absence of life stressors.

Patients with GAD often present with a lifelong history of generalized anxiety. For example, several studies have found that many patients with GAD cannot report a clear age of onset or report an onset dating back to childhood (Anderson, Noyes, & Crowe, 1984; Barlow, Blanchard, Vermilyea, Vermilyea, & Dinardo, 1986; Butler, Fennell, Robson, & Gelder, 1991; Cameron, Thyer, Nesse, & Curtis, 1986; Noyes, Clarkson, Crowe, Yates, & McChesney, 1987; Noyes et al., 1992; Rapee, 1985; Sanderson & Barlow, 1990).

Two types of cognitive distortions involved in excessive anxiety and ruminative worry are

probability overestimation and catastrophic thinking (Brown, O'Leary, & Barlow, 1993). Cognitions involving probability overestimation are defined as those in which a person overestimates the likelihood of the occurrence of a negative event (that is actually unlikely to occur). Catastrophic thinking is defined as the tendency to view an event as intolerable, unmanageable, or beyond one's ability to cope with successfully. Also in the category of catastrophic thinking are thoughts that involve drawing extreme conclusions or consequences to minor, unimportant events. Cognitions reflecting a strong need for perfection or personal responsibility (and of drawing extreme negative conclusions of the consequences of not being perfect or responsible) fall under this category as well. Worry has been conceptualized as a negative reinforcer that serves to dampen physiological reactivity to emotional processing (Borkovec & Hu, 1990). In a sense, worry may serve to hinder complete processing of more disturbing thoughts or images.

Two principal targets of treatment intervention for GAD are excessive, uncontrollable worry and its accompanying persistent overarousal and central nervous system symptoms. These cognitive and somatic features have been addressed most frequently with cognitive therapy and some form of relaxation treatment. Targeting worry directly via an exposure-based paradigm has emerged as a potentially effective treatment component (Craske, Barlow, & O'Leary, 1992). Brown et al. (1993) outline a 12- to 15-session protocol for GAD involving self-monitoring, progressive muscle relaxation, cognitive restructuring (including worry exposure), worry behavior prevention, problem-solving, and time management.

### **1.11.5 COGNITIVE MODELS OF DEPRESSION**

#### **1.11.5.1 Cognitive Distortion Models**

The cognitive distortion model of depression is based upon the assumption that erroneous beliefs and maladaptive information processing play a key role in the onset and maintenance of depression (Beck, 1963, 1967, 1976). Individuals at risk for becoming depressed are said to possess a latent cognitive schema that becomes active under conditions of stress. Once activated, this schema influences the way information is processed and affects the nature of specific beliefs about the self, one's experiences in the world, and one's future (Kovacs & Beck, 1978).

In this model, a negatively biased cognitive set constitutes the core process (Beck, 1976; Beck

et al., 1979). When faced with stressful life events, depression-prone individuals experience negative thoughts. These thoughts typically consist of negative views of the self, the world, and the future which precede the experience of depression. To such individuals, the environment presents overwhelming obstacles that guarantee personal failure. These depressed patients view the environment as overwhelming, as presenting insuperable obstacles that cannot be overcome, and as continually resulting in failure or loss. They view the future as hopeless and they believe that their own efforts will be insufficient to change the unsatisfying course of their life.

Three major concepts in this model are the cognitive triad, negative schemas, and cognitive errors. The cognitive triad consists of three patterns of negative ideas and attitudes about the self, world, and future that are said to characterize people who are depressed.

The depressive view of the self includes negative evaluations of abilities and worth as a person. In comparing themselves to others, depressed individuals are conceptualized as seeing themselves as defective and inadequate. This negative self-view pervades virtually all aspects of life and results in an overwhelming sense of worthlessness. Beck and his associates (Beck, 1963; Beck & Hurvich, 1959; Beck & Ward, 1961) observed that clinical interviews with depressed patients were dominated by themes of failure and personal inadequacy. Hollon and Kendall (1980) have documented the association of depression with self-reports of frequent automatic thoughts of personal inadequacy and maladjustment. In laboratory tasks, depressed individuals consistently expected their performance to be worse than average, and when asked to provide evaluative self-ratings, people who were depressed selected trait descriptors with significantly negative connotations.

Second, Beck (1963, 1967) observed that depressed persons perceived their daily experiences as permeated by themes of loss and stress. Their view of the world appeared to be that life is an overwhelming burden filled with excessive demands and daily defeats, and consequently depressed individuals experienced a pervasive sense of helplessness. Depressed patients rate even the most common interpersonal problems as significantly more difficult to deal with than people who are not depressed (Funabiki & Calhoun, 1979), and individuals who have apparently recovered from depressive episodes continue to dream about problems and losses more often than nondepressed people (Hauri, 1976).

The third component of the cognitive triad is the depressive view that the future is hopeless

and that the current unpleasant condition will continue without any possibility of improvement. Researchers have consistently found a strong association between depression and measures of pessimism and hopelessness (Erickson, Post, & Paige, 1975; Gottschalk, 1974; Hollon & Beck, 1979). In addition, depressed persons tend to dwell on past failures rather than to look toward future possibilities (Miller, 1975; Shaw, 1979).

According to this model, most symptoms of depression may be attributed to the negative cognitive triad. Sad affect and depressed mood are assumed to be a direct consequence of negative cognitions. In support of this, Velten (1968), Frost, Graff, and Becker (1979), and Teasdale and Fogarty (1979) have demonstrated that having subjects concentrate on depressive thoughts can induce a depressed mood.

Other symptoms of depression are also viewed as a consequence of the depressed person's cognitions in this model. Decreased activity levels, for example, are thought to be the direct result of the pessimism and hopelessness that characterize a depressed person's thoughts.

A major component in Beck's model of depression is the concept of schemas. Schemas are stable, long-standing thought patterns representing a person's generalizations about past experiences. According to this model, schemas serve to organize information from past circumstances that appears to be relevant to a current situation and direct attention selectively to particular aspects of a situation. Depression-prone individuals develop schemas consisting of stable but negative views of themselves and their experience. The schemas that produce depression often involve the perception of a personal loss or damage to one's self-worth. Depression-prone individuals tend to respond to life circumstances in a fixed, negative manner, independent of what is occurring in their environment due to the negative nature of their internalized schemas.

Once learned, a schema is usually out of awareness until the person encounters circumstances reminiscent of the conditions in which the schema was learned. At that point, the person may employ the schema to organize and process information about the situation. Depressive schemas predispose the individual to distort events so as to maintain a negative view of the self, the environment, and the future.

Derry and Kuiper (1981) compared the memories of depressed and nondepressed patients for adjectives that were either meaningful to them or descriptive of them. Depressed patients recalled more adjectives with depressive content that they had judged descriptive of

themselves than nondepressed patients or normal controls, which lends support to the hypothesis that depressed individuals utilize a negative schema for processing and retaining personal information. Abramson, Alloy, and Rosoff (1981) examined the hypothesis that negative self-schema may prevent depressed persons from generating hypotheses about the contingencies between their behavior and possible outcomes, and found that when asked to develop generalizations for exerting control, depressed subjects underestimated the potential amount of control available to them.

In this model, stressful life events activate depressive schemas. These schemas, in turn, are responsible for distortions in the way depression-prone individuals perceive and interpret experiences. Although interpretations based on depressive schemas often result in conclusions that are logically inaccurate, depression-prone individuals maintain depressive schemas even in the face of evidence disproving their validity. Beck et al. (1979) contend that the maintenance of depressive schemas is a consequence of a faulty system of information processing in which the individual draws illogical conclusions due to basic cognitive distortions. According to Beck, depressed individuals make these errors in logic when evaluating experiences. Consequently, their thoughts are characterized by extreme, negative, categorical, absolute, and judgmental cognitions. Their distorted perceptions serve to maintain negative views of themselves, the world, and their future.

Beck (1967, 1976) has described a number of specific types of cognitive distortions that tend to be present in the thoughts of individuals with emotional disorders. "Arbitrary inference" refers to drawing a specific conclusion in the absence of evidence to support the conclusion. In "absolutistic, dichotomous thinking," only one or both of two extreme alternatives are considered. "Overgeneralizing" refers to a single instance being taken as representative of a broader class of situations or characteristics. In "selective abstraction," some aspects of a situation, typically negative, are attended to or remembered at the expense of other more positive aspects. In "mind reading," the attitudes or future actions of others are assumed without evidence. "Personalizing" refers to the assumption that an action is directed toward (or occurs because of) oneself rather than some other aspect of the situation. "Should statements" refer to absolute imperatives that are expressed regarding an individual's or others' behavior. In "catastrophizing," extreme negative outcomes are anticipated without substantial evidence. In "minimizing," the significance

of positive outcomes is downplayed, just the reverse of "catastrophizing."

Recent developments in this cognitive model suggest that the presence of very early schemas is an important predisposing factor for many patients with depression (Beck et al., 1990; Stein & Young, 1992; Young, 1990). Young identified a subset of schemas which he has labeled "early maladaptive schemas" (Young, Beck, & Weinberger, 1993). "Early Maladaptive Schemas refer to extremely stable and enduring themes that develop during childhood and are elaborated upon throughout the individual's lifetime" (Young, 1990, p. 9). Young has identified 16 of these early maladaptive schemas in the six following domains: instability and disconnection, impaired autonomy, undesirability, restricted self-expression, restricted gratification, and impaired limits.

According to Young, children learn to construct reality through early experiences with the environment, especially with significant others. Sometimes these early experiences lead children to accept attitudes and beliefs that will later prove maladaptive. These schemas are usually out of awareness, and may remain dormant until a life event stimulates the schema. Once this schema is activated, the patient categorizes, selects, and encodes information in such a way that the maladaptive schema is maintained. In this way, early maladaptive schemas predispose depressed patients to distort events in a characteristic fashion.

Early maladaptive schemas are defined as (i) *a priori* truths about oneself and/or the environment; (ii) self-perpetuating and resistant to change; (iii) dysfunctional; (iv) often triggered by some environmental change; (v) tied to high levels of emotion when activated; and (vi) usually are the result of an initial interaction between the child's innate temperament and dysfunctional developmental experiences with family members or caretakers (Young, 1990).

Cognitive-behavior therapy of depression is based on the premise that individuals can learn to recognize and modify their negative beliefs and maladaptive information-processing tendencies to prevent or alleviate depression (Beck, 1964, 1970; Beck et al., 1979). This approach is typically operationalized as a structured, didactic intervention in which clients are encouraged to test the accuracy of their own beliefs, often by means of modifying their behavior in a systematic fashion. Treatment is usually provided in a skills-training format, with the goal being to help clients acquire a frame of reference and a set of procedures that they themselves can apply to forestall future depressions.

Brewin (1989) suggested that rather than create new memories, cognitive therapy for

depression attempts instead to limit the ease with which these memories are activated by the current environment. One common feature of depression is that negative mood changes are elicited by a wide range of stimuli. Patients respond to many relatively harmless situations as though these situations contained enormous potential for psychological or physical threat.

By drawing attention to the patient's apparent assumptions and challenging these with the use of logic and behavioral experimentation, the patient can develop new rules for discriminating between situations that are truly threatening and those that merely elicit the feeling of being threatened, similar to the process of construct elaboration described by Kelly (1955). Practice in making these discriminations then changes the content of verbally accessible knowledge so that previously threatening situations are reclassified and automatic activation of the unconscious representations is decreased (Brewin, 1989).

In his activation-based model of cognitive therapy for depression (Brewin, 1996), relevant situationally accessible memories are not overwritten and remain available to be reactivated, which explains the high likelihood of relapse in depression. The more varied the negative experiences contributing to the formation of the memories, the fewer distinctive stimulus features the memories contain. This promotes overgeneralization and impedes the identification of relevant discriminations. According to Brewin, relevant cues associated with any event may activate or deactivate memories. Negative life events are therefore strongly associated with the onset of depression where there is prior low self-esteem (Brown, Andrews, Harris, Adler, & Bridge, 1986), and positive events are strongly associated with recovery from depression (Brown, Lemyre, & Bifulco, 1992).

#### 1.11.5.2 Differential Activation Model

The differential activation model of depression (Clark & Teasdale, 1985; Teasdale, 1983; Teasdale & Dent, 1987) attempts to account for vulnerability both to onset and persistence of depression. The differential activation hypothesis assumes that in addition to any differences in cognitive organization that may be apparent in the nondepressed state and in addition to idiosyncratic cognitive schemas that may be activated, vulnerability to severe and persistent depression depends upon differences in patterns of thinking that are activated while in the depressed state. It is suggested that the patterns of thinking activated in the initial depressed state will determine whether that state remains mild or transient, or whether it becomes more

severe (onset vulnerability). Differences in the patterns activated in more severe states will determine whether those states show remission or become persistent and chronic (persistence vulnerability).

According to this hypothesis, the original source of the depression may not matter too much. The crucial factor that determines whether the initial depression will intensify and persist is the pattern of thinking that exists when the patient is depressed. The way in which experiences are interpreted will be a joint function of the nature of the experience and the state of the information processing system that interprets them. The latter will determine the type of information that is selected for attention and the nature of the interpretative categories that are highly primed and most likely to be used to interpret experiences (Bargh, Bond, Lombardi, & Tota, 1986). Similarly, the type of information from memory that is processed will be a function both of what is available in memory and what is most accessible at a given time. Interpretative biases may also affect the interpretation of this material.

Teasdale (1983) has elaborated upon Bower's associative network theory (Bower, 1981) to explain the effect of mood on cognitive processing. Teasdale (1985) has proposed that faulty beliefs about symptoms (depression about depression) lead to the exacerbation of depression. Depressed mood increases the accessibility of representations of depressing experiences and the accessibility of negative interpretative categories and constructs. In depressed mood, there will not only be an increased likelihood that unhappy memories will come to mind, but there will also be a negative bias in the way situations are perceived and interpreted, and in the way in which inferences and predictions are made using information from the environment and from memory. As a result of these effects of mood on cognitive processing, once a person is in a depressed state any environmental input that can be interpreted in ways that will produce further depression will be more likely to be interpreted in that way. Similarly, the memories that come most easily to mind in the depressed state are those that are likely to perpetuate the depression. This reciprocal relationship between depression and cognitive processing creates the potential for a positive feedback loop. Depressed mood and cognitive processing can act to reinforce each other and to set up a vicious cycle that will tend to intensify and maintain depression.

Barnard and Teasdale (1991) and Teasdale and Barnard (1993) have elaborated upon the original differential activation hypothesis with-

in the framework of a comprehensive systemic model of the organization and function of resources underlying human cognition, the interacting cognitive subsystems (ICS) model. Their ICS model, an extension of Barnard's original ICS model (Barnard, 1985), proposes a framework of nine peripheral and central cognitive subsystems: the acoustic, morpho-lexical, articulatory, propositional, implicational, body state, visual, object, and limb subsystems. Each subsystem is hypothesized to contribute either directly or indirectly to the production or experience of emotion.

The ICS model characterizes persistent depression as a product of the establishment of self-maintaining, reverberatory patterns of cognitive activity within the implicational subsystem, with streams of negative thoughts that are experienced by depressed patients as markers of that activity. This theory suggests that the function of therapy is to disrupt the repeated synthesis of high-level schematic representations containing generic meanings prototypical of previous depressing situations (so-called depressive interlock). These representations contain idiosyncratic sensory, proprioceptive, and meaning information synthesized from past experiences of depression that cannot be fully captured by a verbal description. Interlock involves numerous cognitive subsystems, which include those responsible for processing sensory and proprioceptive data as well as those that extract meaning at propositional and higher-order (implicational) levels. Disruption of interlock may be achieved in a number of ways. For example, if the representations were continually being activated by ongoing stress, problem-solving training aimed at resolving that stress might effectively deactivate them. Consistent with this view, distraction procedures that interrupt the flow of negative thoughts may also lead to an alleviation of patients' depression (Fennell, Teasdale, Jones, & Damle, 1987).

### 1.11.5.3 Cognitive Appraisal Model

As noted earlier, the work of Lazarus has long been associated with cognitive appraisal processes mediating emotional responses to stressful stimuli (Lazarus, Averill, & Opton, 1970). This has provided a general framework for explaining depressive reactions to personally stressful events and circumstances (Coyne, Aldwin, & Lazarus, 1981; Folkman & Lazarus, 1986; Lazarus, Averill, & Opton, 1970; Lazarus & Folkman, 1984).

The central component of the cognitive appraisal model of depression is an emphasis

on person–environment reciprocal influences, cognitive appraisal processes, and coping. This transactional perspective emphasizes relational factors that are thought to “transcend the separate sets of person and environment variables of which they are comprised” (Lazarus & Launier, 1978). In Lazarus’ model, both environmental and personal factors are seen to be involved in an ongoing reciprocal relationship, which can be studied as person–environment units. Transactions between person and environment are mediated by two processes: appraisals and coping.

According to Lazarus and Launier (1978), “cognitive appraisal can be simply understood as the mental process of placing any event in one of a series of evaluative categories related either to its significance for the person’s well-being (primary appraisal) or to the available coping resources and options (secondary appraisal).” The emotional responses of sadness and depression are therefore a direct outcome of appraisals of the meaning of an event in relation to that person’s well-being. The emotional response may be immediate and nonreflective, so that thoughts and feelings are simultaneous, but appraisal is an essential component of emotion (Lazarus, 1982).

Primary appraisals may lead to judgments that a circumstance is irrelevant, benign or positive, or stressful. A stressful appraisal can typically be classified as one of three types: harm/loss (the damage has already occurred), threat (anticipated harm or loss), and challenge. The outcome of this primary appraisal, in this case the experience of depression, will be determined by the transaction between personal experiences, predisposition, and environmental factors. Coping appraisals or secondary appraisals of perceived coping resources and options reciprocally interact with the primary appraisal of the nature and magnitude of the stressor and its effect on the person’s well-being.

Similar to other cognitive models of depression, the cognitive appraisal model emphasizes the central role of cognitive processes in provoking depressive reactions to stressful events. The transactional emphasis, however, differs from perspectives that emphasize either the qualities of events (Paykel, 1979; Brown & Harris, 1978) or personal characteristics (Abramson, Seligman, & Teasdale, 1978; Beck, 1967). The Lazarus model views the individual as someone in a continuing state of appraising and negotiating stressful occurrences and everyday circumstances. Although threatening or stressful acute events or ongoing chronic stressors are seen as important antecedents of emotional reactions, everyday “hassles,” or more minor events can also be associated with

depressive symptomatology (Kanner, Coyne, Schaefer, & Lazarus, 1981).

#### 1.11.5.4 Learned Helplessness Model

The learned helplessness model was first described in the 1960s by Overmier and Seligman (1967) and Seligman and Maier (1967). They reported that dogs which experienced inescapable electric shock demonstrated significant motivational, learning, and emotional deficits and failed to initiate behaviors to terminate the shock. Seligman suggested that these deficits were a consequence of the animals learning that their behavior had no impact on the outcome of whether or not they received a shock. After experience with uncontrollable outcomes, they appeared to develop low expectancies for exerting control over later outcomes which could be controlled. These low expectancies appeared to produce motivational, affective, and behavioral deficits similar to depression observed in humans.

Seligman (1975) compared the responses of people experiencing feelings of helplessness as a result of experimental manipulations in the laboratory with the behavior of individuals suffering from naturally occurring depression. Both groups demonstrated similar response patterns including passivity, slowed learning, lowered aggression, loss of appetite, negative expectations, and feelings of helplessness, hopelessness, and powerlessness.

The original helplessness model did not specify which factors account for differences in the severity and chronicity of depression, however, nor did it address subtypes of clinical depression or explain such symptoms as guilt and blame (Abramson & Sackheim, 1977). In some instances, uncontrollability of outcome also not only failed to produce helplessness but actually facilitated subjects’ subsequent performance (Wortman & Brehm, 1975).

##### 1.11.5.4.1 Revised helplessness model

In response to the need for a more complex model of human depression, Abramson, et al. (1978) reformulated the helplessness theory, placing an emphasis on individual causal interpretation of uncontrollable events. They proposed that an individual’s attribution for loss of control serves as a mediator between the absence of control and the emergence of helplessness-related deficits and depression and that helplessness and depression are most likely to occur following either the inability to produce a highly desired outcome or the inability to prevent a highly aversive outcome. When confronted by such circumstances, individuals



attempt to explain why the circumstances have occurred and these explanations determine how they will respond to events.

In this revised model, it is assumed that attributions are the primary determinants of an individual's actions and affect. According to Abramson et al. (1978), the cause may be attributed either to the person or to the situation (an internal vs. an external explanation). The cause may also be viewed as either transient or persistent across time (an unstable vs. a stable explanation), and the cause may be perceived to have an impact on a variety of outcomes or may be limited just to that particular event (a global vs. a specific explanation). The revised helplessness model proposes that particular consequences are associated with attributions made across each of these three dimensions. For example, an individual who attributes a negative event to an internal cause is likely to experience a loss of self-esteem. The stability of causal beliefs is hypothesized to affect the persistence or chronicity of depression following negative events. If a negative event is explainable by a persistent cause, then depressive reactions are likely to be chronic. If an individual believes that a global factor has caused the negative event, then deficits associated with helplessness and depression are likely to occur in a variety of situations.

The revised helplessness model assumes that individuals demonstrate consistency in attribution and tend to invoke the same sorts of causal explanations for different negative events in their lives. An individual who typically invokes internal, stable, and global causal beliefs will therefore be at a high risk for developing depression when faced with uncontrollable negative events. Brewin (1985) and Sweeney, Anderson, and Bailey (1986) have reviewed the literature examining this relationship between measures of attributional style and depression.

According to the revised model, when an individual perceives negative events as beyond their control, the expectation of future uncontrollability is likely to occur. In such circumstances, the specific attributions that develop are influenced by the reality of the particular circumstances and the individual's explanatory style. The expectation of future uncontrollability is thought to be sufficient to produce most of the symptoms of depression, including a lowered rate of activity, cognitive deficits such as impaired learning, emotional responses of sadness and anxiety, and a lowering of appetitive drives. In addition, the expectation of uncontrollability is hypothesized to bring about physiological consequences such as neurochemical changes and an increase in susceptibility to disease.

#### 1.11.5.4.2 *Hopelessness theory*

Abramson, Metalsky, and Alloy (1988) present a further elaboration of the revised theory of human helplessness and depression which is relabeled as the hopelessness theory of depression. The hopelessness theory of depression specifies hopelessness as a proximal sufficient and necessary cause of depression beginning with the occurrence of negative life events and ending with the symptoms of a type of depression called hopelessness depression. It has been speculated (Seligman, 1978) that hopelessness depression may not directly correspond to current diagnostic categories of depression, and may even include psychological phenomena not currently contained in psychiatric nosologies. The hopelessness subtype of depression is hypothesized to be characterized by three primary symptoms and one secondary symptom. The major symptoms are described as the retarded initiation of voluntary responses (a motivational symptom), a difficulty in seeing that one's responses control outcomes related or similar to the outcome about which one feels hopeless (a cognitive symptom), and sad affect (an emotional symptom). The secondary symptom of lowered self-esteem is hypothesized to be related to the expectation that other people can attain the outcome that the patient feels hopeless to attain.

The causal pathway hypothesized by Abramson et al. (1988) which culminates in hopelessness suggests that at least two distinct variants of hopelessness depression may exist. The first theoretical variant of hopelessness depression is called attributional style-event hopelessness depression. This variant of depression would develop in an individual who exhibits a depressogenic attributional style and is confronted with a negative life event which by itself may not be sufficient to trigger hopelessness in most people, but when interpreted or explained by an individual with this attributional style is sufficient to do so. A second variant of hopelessness depression is labeled as event hopelessness depression. This theoretical variant of hopelessness depression would develop in an individual who does not exhibit the hypothesized depressogenic attributional style or other depressogenic cognitive styles, but who is confronted with an event sufficient to engender hopelessness in most people. It is thought that event hopelessness depression may overlap with the diagnostic category of acute dysphoria (Klein, 1974) and that attributional style-event hopelessness depression may correspond to chronic over-reactive dysphoria.

Hopelessness depression is conceptualized as the culmination of a series of events beginning

with a negative life stress and including depressogenic attributional styles, situational cues, stable and global attributions for the negative life event, and other contributory causal pathways such as deficits in social support networks. Intervention to reverse hopelessness depression or to reduce vulnerability to depressive episodes and depression-proneness can by definition occur at any point in the causal chain leading to hopelessness, and possible intervention strategies are reviewed by Alloy, Clements, and Kolden (1985), Beach, Abramson, and Levine (1981), and Halberstadt, Andrews, Metalsky, and Abramson (1984).

### 1.11.6 CONCLUSIONS

In addressing the cognitive basis of clinical psychology, several developments have been considered in the field of cognitive psychology, and how they may impact the development, presentation, and treatment of psychopathology. The focus was limited to salient topics that embody multifaceted influences of other scientific endeavors, including those of information networking research, emotion, and abnormal psychology. In doing so, other issues of importance to the field of cognitive psychology have not been addressed comprehensively. It is hoped that this approach, while relatively less inclusive, provides the reader with a flavor of how information processing can impact, or at minimum interact with, other factors in the development of psychopathology.

Broadly defined, cognitive approaches to emotional response focus on evaluation of information processing patterns that contribute to directing and organizing a behavioral/emotional response. The role of cognitive processes such as appraisal determine how we react at multiple behavioral and physiological levels, and how these reactions may contribute to maladaptive behaviors. In this vein, the role of cognitive distortions as important processing dispositions that may predispose an individual to emotional problems have been addressed.

The discussion of anxiety and depression as examples of emotional disorders directly impacted by cognitive factors provides an illustration of the nature of cognitive contributions in the cascade of events leading to the development of psychopathology. This discussion was preceded by an overview of how cognition and emotion interact in a dynamic fashion. The approach chosen treats cognitive and emotional change, not only as potential mediators for each other, but also as consequences of each other. Such may be the case when addressing cognitive deficits observed in depressed individuals.

A trend that has been noted here is of multiple disciplines addressing cognition-based questions. The increased volume of these interdisciplinary investigations is being nurtured by the growth in cognitive neuroscience, psychophysiology, and neurobiology. Methodological and conceptual advancements in these areas are being brought to bear to increase understanding of how cognitive processing affects behavior at the level of multiple systems. Some of these issues have been brought to the forefront in discussion of the interaction of cognition and emotion and the viability of integrating research in this area with research in neurobiology and neuroanatomy.

The challenge that lies ahead is in bringing about a coherent integration of these disciplines that is theoretically sound and empirically supported. Such a framework not only would allow efforts in these areas to be well focused and well guided, but would also foster further cross-disciplinary research on constituent questions within each area to address how cognitive factors contribute with other factors in precipitating psychopathology.

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# 1.12

## Epidemiology and its Rubrics

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### 1.12.1 OVERVIEW

With a heavy reliance upon quantitative methods, epidemiology functions as a lens to aid clinicians who wish to know the answers to five important questions about what affects their patients. The five questions are:

- (i) In the community at large, how many are affected?
- (ii) Where are the affected cases more likely to be found?
- (iii) What accounts for some people in the community becoming cases while others do not?
- (iv) What linkages of states and processes influence who becomes and remains a case?
- (v) What can be done to prevent and intervene?

These questions form the rubrics for epidemiology, that is, its major headings.

Using epidemiology as a lens, a clinician can see along dimensions that stretch beyond the individual patient, and beyond the threshold of the office practice, clinic, and hospital, toward the population from which the patient has surfaced. Viewed through this lens, the experience of individual patients is seen against a

background of the affected cases, frequently numerous, who never seek help or receive care. Epidemiology also provides evidence about the especially informative background experience of mentally and behaviorally healthy individuals in the community who might have become cases but did not. As discussed in this chapter, epidemiology provides a sharp focus and greater understanding with respect to any patient's clinical condition. More than any other scientific discipline, epidemiology involves population-level studies of each condition of health or ill-health. Epidemiology focuses upon the nature and extent of the occurrence of each condition, its distribution in the community, the causes and mechanisms of its development, and the well-established or hotly debated means for intervention, especially in terms of health promotion and disease prevention.

Among the most remarkable accomplishments of epidemiology has been demonstration of a counterintuitive idea. Namely, effective prevention and intervention strategies are often devised at an early stage of hypothesis testing, well before there is definitive and thorough evidence on the specific causes and on causal or

protective mechanisms (Wynder, 1994). This chapter draws attention to numerous examples of effective health promotion and preventive interventions before causes and causal mechanisms were known. The chapter also provides illustrations that effective prevention can elude us even when we have firm knowledge about specific causes.

As a discipline, epidemiology started to intersect with the content areas of clinical psychology and psychiatry in the early nineteenth century. Valuable field surveys of mental morbidity in circumscribed national and local populations were underway during the 1830s, as an extension of growing concern about how many people in the population were mentally ill, and whether the mentally ill were mistreated. These epidemiological field surveys on mental health were completed a decade before the European father of epidemiology, John Snow, launched his studies of cholera, and about 80 years before Joseph Goldberger embarked on pellagra research and became the discipline's American father.

In the mid-nineteenth century, while Snow was defining cholera epidemiology, his senior colleague in London's Epidemiological Society, William Farr, started a range of investigations on mental disorders as part of his general studies of mortality from all causes. For instance, using numerical methods he had learned during French medical training under Pierre Charles Alexander Louis, Farr compared mental hospital patients admitted to different asylums with respect to their chance of being discharged in a state of recovery vs. the risk of dying in the institution.

The transition of epidemiology from a prenumerate phase into numeracy actually might be seen best in relation to the study of causes of mental health and disturbances of mental life and behavior. Some of the nineteenth-century questions provoking this transition to numeracy were the following: (i) which nations and systems of government produced the mentally healthiest populations, (ii) was it true that slavery actually improved the mental health of the Africans transported to America, and (3) can't we reduce the number of cases of mental disorder by finding the families that produce these cases and doing something about them?

In numerate form, epidemiology has become a scientific discipline with specific guiding principles and concepts. It also has a unique methodology and associated jargon. Most theories of epidemiology can be expressed in a generic framework of interaction between agent, host, and environment (D.E. Lilienfeld & Stolley, 1994). Nevertheless, these theories often

remain specific to an object of study, in that it makes no more sense to speak of an "epidemiology of all mental illness" than it does to speak of an "epidemiology of all infectious diseases" or an "epidemiology of all cancer" (Anthony, Eaton, & Henderson, 1995). We have an epidemiology of cholera, which has some similarities but is not the same as our epidemiology of AIDS, although both are conditions of infectious origin. We have an epidemiology of brain infarction and hemorrhage (stroke), which is not the same as the epidemiology of neurosyphilis or the epidemiology of late-life dementia, even though all of these conditions involve the brain. The epidemiology of suicide attempts is not one and the same as the epidemiology of suicide (Borges, Anthony, & Garrison, 1995; Monk, 1987). The epidemiology of suicide can be distinguished from the epidemiology of major depression, although many cases of major depression become suicide victims (Moscicki, 1995).

There is an epidemiology for "mental health," an epidemiology for individual behaviors (e.g., suicide attempts), and for each of the categories of mental disorders listed in psychopathology's classifications and nomenclatures. For example, we have an epidemiology of schizophrenia, within which hypotheses of genetic causation contend with those of prenatal infections and insults, as well as hypotheses involving later conditions of environment or gene-environment interaction (Jablensky, 1995). We have an epidemiology of drug dependence, also with a new prominence of genetic hypotheses, but with undeniable demands for gene-environment interactions. For example, without encountering an opportunity to ingest cocaine, no one can become cocaine dependent. There now is evidence that genes may determine personality traits that, in turn, influence who seeks out novel opportunities, such as the opportunity to use cocaine. Other evidence points to genetic influences on who becomes cocaine dependent once cocaine is ingested (Uhl, Blum, Noble, & Smith, 1993). Cocaine, in turn, can influence the production of messenger RNA and other gene products of potential later importance to the development of cocaine dependence (Hyman & Nestler, 1996).

Given its orientation to substantive theory, epidemiology has a position in relation to clinical psychology and psychiatry that is analogous to the position of experimental pharmacology and therapeutics or human genetics. There is no unifying theory to master as there might be in physics or sociology, but there is a benefit for the practicing clinician to be familiar with concepts developed or refined within epidemiology, such as "epidemic," "risk

factor,” “odds ratio,” and “exposure opportunity.” These epidemiological concepts serve clinicians as well as do pharmacological concepts such as “the dose–response relationship,” “tolerance,” and “withdrawal,” or concepts from genetics such as “heritability,” the human genome, and genomic imprinting. In addition, to be able to use or simply to interpret scientific findings that emerge from epidemiology, the clinician should become familiar with its research designs such as the epidemiologic case–control study, and the pitfalls or threats to validity of results in epidemiological research. This familiarity complements what clinicians must learn about placebo-controlled double-blind randomized trials of experimental therapeutics, or comparisons of proband-wise vs. pair-wise concordance for monozygotic vs. dizygotic twin pairs in genetics research.

There are also important opportunities for collaborations between epidemiologists, clinical psychologists, and psychiatrists, and for borrowing aspects of methodology from epidemiology for the purposes of clinical research. As described in this chapter, there is a long tradition for these collaborations, and also for the borrowing of epidemiological methods by psychologists and psychiatrists, who have had no other way to test the psychopathological hypotheses they have framed.

Despite the early misgivings of some leaders in epidemiology (e.g., Frost, 1927; Levin, 1950), and unhampered by psychology’s internal debate about its idiographic and nomothetic traditions, there now is much traffic at the intersection where epidemiology meets with psychology and psychiatry. Until about 1980, two years of serious library work was more than sufficient for a scholar to master the history of work at this intersection. This could be accomplished by first developing an acquaintance with every noteworthy book and article on the topic published up to that date, and then by evaluating every new publication on this topic, year by year.

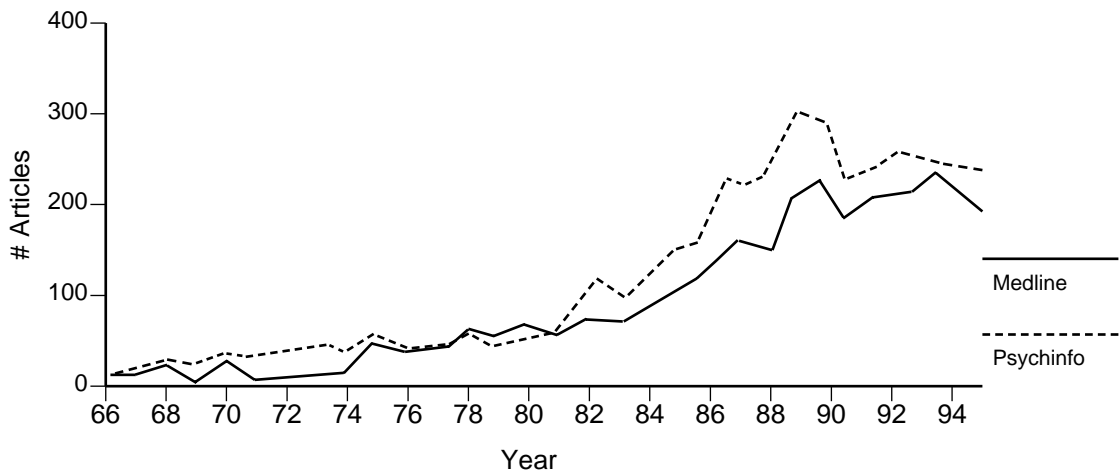
This kind of comprehensive view of the field no longer is within easy reach. Recent upward trends in the number of published articles, shown in Figure 1, have placed a comprehensive view outside the range of all but the most serious full-time scholars in the history of psychiatric epidemiology and epidemiological psychology. Adapting themselves, clinicians interested in epidemiology now generally approach the field as subspecialists. For example, many focus on one or two domains such as the epidemiology of depression, schizophrenia, or drug dependence; likewise for those who take graduate training in public health and epidemiology, but are also interested in psychopathology. Regrettably,

subspecialization in any field is associated with a narrowing of perspective on what has been learned. Important threads of historical origin can be lost. Early concepts and principles can be overlooked. This is what we trade in return for benefits that come with the focused attention of subspecialization.

This chapter is intended as a partial antidote to overfocused subspecialization, providing an overview of epidemiology and its origins while also introducing some selected examples of the intersection of epidemiology with clinical psychology and psychiatry. It is not a comprehensive review of all progress in this domain of epidemiology. Instead, the chapter introduces and reviews a useful selection of key organizing features, concepts, and principles in this field as they have been applied to the study of mental health and to categorical disturbances of mental life and behavior. The history of mental morbidity surveys is covered briefly, including longitudinal and prospective studies to estimate the incidence of mental disorders. It seems that these field studies retain a central position, and they are likely to remain central until still-controversial methodological issues have been resolved. By sorting out these methodological issues, greater attention can be given to what should prove to be a more productive debate on substantive hypotheses about causes, causal and protective mechanisms, and intervention strategies.

The final section of the chapter outlines future directions for research and scholarship in this field. It stresses the possibilities for collaboration between clinical scientists and epidemiologists, with recognition that some investigators will wear both hats. Whereas this chapter provides an orientation to background, concepts, and principles, a companion chapter in this collection provides a more detailed introduction to some specific methods of epidemiology as applied to the study of psychological and psychiatric disturbances (see Chapter 3.06, Volume 3).

All of these topics are covered more thoroughly in other works. There are textbooks of epidemiology, including Major Greenwood’s early classic *Epidemics and crowd-diseases: An introduction to the study of epidemiology* (Greenwood, 1935), as well as those more recently published by Morris (1957), A. M. Lilienfeld (1980), D. E. Lilienfeld & Stolley (1994), and Gordis (1996). There are parallel developments at the intersection of epidemiology, psychiatry, and psychology, including useful texts (e.g., Cooper & Morgan, 1973; D. Goldberg & Huxley, 1992; Tsuang, Tohen, & Zahner, 1995). Short review articles and useful brief monographs also exist (e.g., Bland, 1988;



**Figure 1** Time trend for number of journal articles published on the topic of epidemiological psychology/psychiatry during the period 1966 through 1995: results of Medline and Psycinfo searches. (Note: Boolean searches were used with the following text words: Epidemiolog\$ and Psychiatr\$ or Psycholog\$ or Mental.)

Cooper, 1993; Morrison, 1959; Reid, 1960; Robins, 1978; Rutter, 1981; Shepherd, 1985; Shepherd & Cooper, 1964).

When seeking examples to illustrate the rubrics of epidemiology, we often turned to the *Annotated bibliography of psychiatric epidemiology*, edited and published in 1992 by Michele Tansella, Giovanni de Girolamo, and Norman Sartorius. This useful resource represents a recent effort to compile articles and books reporting achievements in this field which 44 experts from 21 countries had ranked as “outstanding or of special relevance” (Tansella et al., 1992).

### 1.12.2 BACKGROUND

Epidemiology and its rubrics came to the surface during the nineteenth century, but our field has a long history in a prenumerate form. For thousands of years, physicians and merchants have written about the coming and going of human diseases in relation to months and seasons of the year, according to conditions of the land (e.g., swampy vs. arid), and across tribes or families of people. The most famous of these ancient notes on the history, medical geography, and anthropology of human suffering is a collection pulled together under the name of one person: *Of airs, waters, and places*, attributed to Hippocrates.

A physician of the late Renaissance, Girolamo Fracastoro, followed in these footsteps and observed that variations in occurrence of disease might be due to invisible but identifiable agents in air and water. In the late seventeenth century, Ramazzini differentiated diseases and causes of death found to be more common in

some occupations, less common in others. For example, the phrase “mad as a hatter” and Lewis Carroll’s “Mad Hatter” reflect an appreciation of the toxic effects of the mercury used to prepare felt for hats, which includes an intoxication characterized by prominent disturbances of mental life and behavior. As exemplified by this linkage between felt making and mercury poisoning, a common thread running through these early origins of epidemiology was an attempt to find understandable patterns in the occurrence of disease and death, expressed in relation to conditions of time, place, and person.

Epidemiology’s modern era and more quantitative orientation emerged from seventeenth-century advances in mathematics, including an early “life table” method developed by Edward Halley of comet fame, as well as more basic foundations of probability and statistics. While Europe passed through the Age of Enlightenment and the Industrial Revolution, these advances were refined and then applied in work to promote the public’s health and well-being. The inspiration for this work was drawn in part from the social philosophy of French mathematician Condorcet, eighteenth-century founder of the social sciences. The momentum carried over from mathematics and social philosophy into medicine—as expressed by the use of numerical methods by French physician Pierre Charles Alexander Louis and his students, in order to improve patient care and the health of the public (Farr, 1885; A. M. Lilienfeld & Lilienfeld, 1979). One of Louis’s students was William Farr of London, who defined the field of “vital statistics” during a long career of studying Great Britain’s

administrative statistics on births, deaths, and disease.

In early nineteenth-century Great Britain, waves of Asiatic cholera started to sweep through London and other European communities, regularly killing thousands. Responding to the cholera epidemics, Farr used Halley's life table and numerical methods taught by Louis to study cholera's patterned aftermath in mortality statistics. Based on this work, Farr helped to frame the majority position of London's Epidemiological Society: that breathing miasmatic vapors from unclean water or soil caused cholera. Anaesthesiologist John Snow thought otherwise, and chose to work against common opinion. Aided by Farr, Snow assembled the available data on cholera deaths, gathered his own data, and used numerical methods to marshal evidence that cholera was due to drinking contaminated water. Snow's thesis about cholera as a waterborne disease at first was rejected by his peers, including Farr. Nevertheless, in time, Snow's evidence became overwhelming; the miasma theory was overturned. England and the other industrializing countries began to prevent cholera epidemics by improving water sanitation—years before bacteriologist Robert Koch finally discovered the mechanism of action and identity of the cholera vibrio, which we now trace as the disease's specific cause. For this victory of research on cholera, John Snow is now known as a father of epidemiology.

The numerical methods of Louis also crossed the Atlantic to the Americas (Osler, 1897), where nineteenth-century diseases such as yellow fever and pellagra compromised the public's health and prompted major investigations, just as AIDS has done in our own time. The history of epidemiological research on these diseases teaches one of the most important lessons learned by Snow in his work on cholera: Effective prevention does not require thorough knowledge of specific causes nor causal mechanisms. Methods of prevention can be identified and put into place well before definitive etiological evidence is available. Furthermore, firm evidence about etiology and causal mechanism does not always translate into effective prevention.

In the case of yellow fever, many nineteenth-century authorities believed that the disease was spread by inanimate particles ("fomites") in the clothing, bedding, and other possessions of infected persons. In the name of prevention, millions of dollars worth of these possessions were tossed away or burned, until epidemiological and clinical experiments under the direction of Walter Reed's Yellow Fever Commission produced conclusive evidence that yellow fever

was mosquito borne. With this knowledge, the commission began an effective program of yellow fever prevention by draining puddles and swamps, installing nets and screens, and otherwise creating effective barriers between mosquitoes and their human hosts. Many years passed before the yellow fever virus was identified and an effective preventive immunization was created. Thus, effective yellow fever prevention was underway well before it became possible to trace the causal mechanisms and pathways by which the virus passes from mosquitoes to humans.

Pellagra is known as the disease of the three Ds: dermatitis, diarrhea, and dementia (e.g., see Macleod, 1978). Although pellagra was not mentioned by the ancients, it aroused attention during the nineteenth century, especially in the southern United States, Latin America, and other places where maize had become a staple cereal and where other nutrients were in short supply. At the dawn of the twentieth century, the connection with maize was overshadowed by the fact that many of the observed patterns of pellagra occurrence resembled those of an infectious disease with respect to matters such as seasonal variation and clustering of cases within vulnerable subgroups (e.g., the poor). Based on success with cholera, rabies, and other infectious diseases, there was a bacteriological optimism about pellagra, with hope for an early discovery of the responsible bacterium. Nonetheless, from the perspective of Joseph Goldberger, the American father of epidemiology (Goldberger, 1964), there was a different way to interpret the data. In Goldberger's mind, the evidence on pellagra as an infectious disease was just as readily interpretable as evidence favouring a nutritional deficiency—perhaps a deficiency of one of the "vitamines" named by Casimir Fund, a pioneer in the new science of nutrition. Goldberger wrote:

In view of the great uncertainty that exists as to the true cause of pellagra, it may not be amiss to suggest that pending the final solution of this problem it may be well to attempt to prevent the disease by improving the dietary of those among whom it seems most prevalent. In this direction I would urge the reduction in cereals, vegetables, and canned foods that enter to so large an extent into the dietary of the people of the South and an increase in the fresh animal food component, such as fresh meats, eggs, and milk (Goldberger, 1914a).

A year later, illustrating how initial epidemiological findings about the location of observed cases can lead toward investigations into prevention and causes, Goldberger explained his reasoning (Goldberger, Waring, & Willets, 1915):

In a paper published [last year] . . . , attention was called to certain epidemiological observations relating to pellagra which appeared inexplicable on any theory of communicability. These observations showed that, at certain institutions at which pellagra was either epidemic or had long been endemic among the inmates, the nurses and attendants, drawn from the class economically and socially identical with that most afflicted in the population at large, appeared uniformly to be immune, although living in the same environment and under the same conditions as did the inmates. Neither "contact" nor insect transmission seemed capable of explaining such a phenomenon. It was suggested that the explanation was to be found in a difference, which was believed to exist, in the diet of the two groups of residents. . . . It was suggested, therefore, that it might be well to attempt to prevent the disease, . . . by providing those subject to pellagra with a diet such as that enjoyed by well-to-do people, who as a group are practically free from the disease.

Thereafter, Goldberger conducted this very type of experiment, as well as other key studies. In so doing, he produced evidence necessary to confirm that pellagra and the pellagra psychoses are not communicable diseases, and were preventable by appropriate changes in diet such as he had described in 1914. Of course, for many, the evidence from his diet experiment was among the most compelling evidence in favor of his dietary hypothesis. Subsequently, prevention of pellagra became a topic of public activity and public policy, well before 1926, when the pellagra preventive factor was narrowed down to one of the B group of vitamins. Goldberger died in January 1929. Almost a decade passed before a research team at the University of Wisconsin identified niacin as the specific protective factor in relation to the etiology of pellagra.

In the meantime, pellagra has essentially disappeared from the United States, Canada, Western Europe, and other places where either high standards of living or other mechanisms promote niacin-containing diets. For the time being, it has become difficult for US-based psychopathologists to observe cases of pellagra psychoses except when the clinical picture is secondary to chronic alcohol dependence. To see primary pellagra psychoses, psychopathologists typically must travel abroad to countries where various constraints thwart good nutrition, and with much lower standards of living than can be found at home.

A more contemporary history lesson on our capacity to start effective prevention campaigns before full knowledge of causes or causal mechanisms is provided by the recent epidemic of human immunodeficiency virus (HIV) infections and the acquired immunodeficiency

syndrome (AIDS). Well before HIV had been isolated as an infectious agent, and before general concepts or diagnostic criteria for AIDS could be specified, epidemiologists and clinicians at the Centers for Disease Control were able to spot an unexpected occurrence of *Pneumocystitis carinii* pneumonia. They narrowed its predominate location to young homosexual men, some being consumers of amyl nitrite ("poppers") and other drugs, but also positive for exposure to a cytomegalovirus (Centers for Disease Control, 1981a, 1981b, 1981c; Goedert et al., 1982). As the story unfolded, the epidemiologists gathered enough information to shift gears from a search for locations (where are cases most likely to be found?) to a more focused search for causes (why are some affected while others are not?). In this process, they were able to identify inhalant and other drug use as a correlated behavior or cofactor lacking singular causal importance. While clarifying that drug use by itself was not a cause of HIV infection, these studies noted the high-risk status of unprotected receptive anal intercourse with multiple male sex partners, often in conjunction with the use of poppers and other inhalant drugs, or with crack or other psychostimulant drugs. Prevention campaigns to reduce the risk of AIDS in the homosexual community were underway before the specific viral etiology was understood, and before the risk to heterosexuals was appreciated fully. Because the prevention campaigns target the proximal developmental antecedents of high-risk HIV-related behaviors, these early campaigns retain utility for the prevention of HIV infection and AIDS even today, years after isolation of HIV infection as the necessary causal factor for AIDS (National Institute of Allergy and Infectious Diseases [NIAID], 1995).

Drawn from the history of epidemiology (Wynder, 1994), the lesson that effective prevention can precede firm knowledge of causes, causal mechanisms, and developmental sequences is of special importance in relation to disturbances of mental life and behavior. With few exceptions, it is unlikely that definitive and thorough evidence on the causal or protective mechanisms and developmental processes of these disturbances will be available in the near future. But there is no need for research on prevention of these disturbances to await such definitive and thorough evidence, even though it must be acknowledged that every important advance in understanding basic causal mechanisms and developmental processes typically reveals new targets for prevention and intervention. The value of these advances in public health terms is that each advance can lead to discovery of more efficacious interventions with

fewer unwanted side effects and at less cost. In the meantime, preventive trials can move forward and become part of the arsenal of scientific tools used to discover basic causal and protective mechanisms (Mrazek & Haggerty, 1994).

As it happens, we still do not have completely definitive and thorough evidence on the causes and causal mechanisms for any of the infectious or nutritional diseases mentioned above, even though we know how to prevent them, at least in theory. That is, the identification of specific etiological agents for cholera, pellagra, and AIDS did not constitute final brushstrokes in the total picture of disease etiology. In Farr's day, for example, the cholera death rate was lower for the well-to-do than for the impoverished people of London. Apparently, this in part was because wealth was protective: it coincided with access to clean water supplies before there was a complete appreciation of the link between water sanitation and cholera. The association with wealth also seems to be present in the recent upsurge of cholera epidemics during the late twentieth century. Nonetheless, it would be imprudent to delay our efforts to prevent cholera until we understand all the different ways that wealth might promote access to pure water supplies, or until we understand the determinants of the distribution of wealth in society as it might relate to health status. In public health work, the details of these causal mechanisms are of secondary importance when water sanitation, vaccines, and other effective preventive maneuvers are already known.

In Goldberger's day, the well-to-do rarely developed pellagra, as noted in his article from 1915, quoted above. Attempting to develop a more complete view of the epidemiology of pellagra, Goldberger used social science methods designed by a collaborator (Edgar Sydenstricker, an economist). In so doing, his research group was able to document a socioeconomic substrate which was one step back from dietary deficiency in the causal chain leading toward pellagra. In later years, Sydenstricker turned his attention to medical insurance plans and other societal-level means of promoting public health (Sydenstricker, 1964). Needless to say, this aspect of the causal network for diseases associated with socioeconomic status (SES) continues to be underinvestigated.

Turning attention from the more sociological aspects of causal mechanisms for disease to a fundamental biological aspect, we still are many years away from definitive evidence on the chromosomal locations of those genes responsible for our species' widely shared vulnerability to *Cholera vibrio*, or to niacin deficiency. These genes, combinations of genes, and their gene

products represent yet another unexplored part of the causal mechanisms and developmental processes leading toward diseases for which we now have at least some effective preventive interventions.

Finally, in our own time, except for a vocal minority in the scientific world, most scientists are confident that the underlying cause of AIDS is a virus, and not the inhalants, the drinking, cocaine use, or sex *per se*. But the person-to-person spread of this virus, by routes including unsafe sex practices and drug injection practices, is determined at least in part by drug-taking behavior and behavioral disturbances; by alcohol and cocaine use, as well as the sex-associated behaviors of inhaling poppers, or taking methamphetamine via inhalation of smoke ("ice") or injection. These behavioral characteristics by which HIV infection is spread are in turn determined by a broad array of influences that range from the domain of genetics to that of social policies toward drug use and treatment of drug dependence. Hence, even though HIV is confirmed as a necessary cause of infections that lead to AIDS, this evidence did not complete the picture of causal mechanisms for HIV infection and AIDS. In the present instance, knowledge of a necessary cause has not yet produced an effective means to block the now global epidemic spread of this infection (NIAID, 1995).

### 1.12.3 THE RUBRICS OF EPIDEMIOLOGY

Whereas the search for causes, causal mechanisms, and methods of disease prevention and health promotion are central to epidemiology, many in this field choose to concentrate on the other rubrics listed in Table 1. Indeed, epidemiology's first intersection with the subject matter of clinical psychiatry and psychology addressed pragmatic questions. How many persons suffer from mental maladies? Where in the community population are these cases to be found? Are they being treated badly or well?

#### 1.12.3.1 Rubric 1: How Many? A Question of Quantity

Many clinicians in the United States are interested in the mental and behavioral disturbances that affect adolescents and adults in the age range 15–54 years; fewer are acquainted with epidemiologic estimates on the prevalence and incidence of these disturbances (e.g., Kessler et al., 1994; Regier et al., 1993). Many do not appreciate that more Americans in this age range have been affected by a history of

**Table 1** The five main rubrics, subject matter, and questions of epidemiology.

<i>Main rubrics</i>	<i>Subject matter</i>	<i>Questions asked by epidemiologists</i>
(i) How many?	Quantity	How many in the population are affected as cases, or are becoming cases?
(ii) Where?	Location	Where in the population are affected cases more likely to be found, with respect to characteristics, conditions, and processes of person, place, and time?
(iii) Why?	Causes	What accounts for some people becoming affected while others are spared?
(iv) How?	Mechanisms	What sequences or linkages of states and processes influence who becomes and remains a case and who does not?
(v) What can be done?	Prevention and intervention	What can be done to prevent or delay the onset of suffering, to shorten its duration, or otherwise to reduce the burden of affected cases, their families, and society in general?

dependence on psychoactive drugs than by other psychiatric disturbances now accorded higher priority in mental health service delivery systems, prevention, and sponsored research programs. For example, the most recent nationally representative sample survey of 15–54-year-olds in the USA, indicates that cannabis (marijuana) dependence has affected about 4.2% in this population. By comparison, this survey, conducted between 1990 and 1992, found only 3.5% to have had a history of panic disorder.

Cocaine dependence was almost as prevalent as antisocial personality disorder (2.7% versus 3.5%) and was about 70% more common than bipolar disorder (1.6% prevalence). Alcohol dependence (14.1%) was somewhat more common than simple phobia (11.3%) and social phobia (13.3%), whereas tobacco dependence at 24.1% was more prevalent than major depressive disorder at 17.1%. (Anthony, Warner, & Kessler, 1994)

The prominence of alcohol and other drug problems in the United States can also be seen when epidemiology seeks to quantify how many persons are currently active cases of mental and behavioral disturbances. This is true whether the case assessment method involves computerized diagnoses based on information from standardized interviews conducted by lay persons (e.g., the Diagnostic Interview Schedule, or DIS) or standardized psychiatric diagnoses made by clinicians after a formal psychiatric assessment with opportunities for cross-checking and probing freely. For example, in the Baltimore (Maryland) arm of the National

Institute of Mental Health (NIMH) Epidemiologic Catchment Area community surveys carried out during 1981–1982, the DIS method found 3.6% of the study population with a history of recently active *DSM-III* alcohol use disorders (i.e., with symptoms present during the month prior to assessment). An estimated 1.1% had active drug use disorders involving marijuana and other internationally regulated substances. The DIS estimate for one-month prevalence of *DSM-III* Major Depression was intermediate, at 2.3%. By comparison, the corresponding one-month prevalence estimates based on standardized psychiatric examinations of the study subjects were 6.9% for alcohol use disorders, 3.1% for drug use disorders, and 1.1% for major depression. The most commonly found mental disorder by both methods of case assessment was phobic disorder: according to the DIS method, 11.2% of the Baltimore study population had phobic disorder with symptoms present in the month prior to assessment. The corresponding estimate based upon the psychiatric examination was 21.3% (Anthony et al., 1985). Other one-month prevalence estimates from the Baltimore study are presented in Table 2.

These recent estimates from epidemiologic surveys of community populations are not definitive for all persons, places, or times; variation in epidemiological estimates proves to be as important as constancy. Nevertheless, point estimates from community surveys of mental and behavioral disturbances have been indispensable when policy makers and planners have faced hard decisions about allocation of scarce health resources, and when society has to



**Table 2** One-month prevalence of selected *DSM-III* conditions, by two methods of ascertainment. Data from the Eastern Baltimore Mental Health Survey conducted as part of the NIMH Epidemiologic Catchment Area Program, 1981.

<i>DSM-III category</i>	<i>Prevalence estimate based upon standardized clinical diagnosis by study psychiatrist (%)</i>	<i>Prevalence estimate based upon Diagnostic Interview Schedule administered by lay interviewer (%)</i>
Phobic disorders	21.3 (1.9)	11.2 (1.2)
Alcohol use disorders	6.9 (1.1)	3.6 (0.8)
Drug use disorders	3.1 (1.0)	1.1 (0.5)
Major depressive episode	1.1 (0.4)	2.3 (0.4)
Schizophrenia	0.5 (0.1)	0.7 (0.2)
Manic episode	0.4 (1.2)	0.4 (0.9)
Obsessive-compulsive disorder	0.3 (0.1)	1.3 (0.3)
Panic disorder	<0.1 (0.07)	0.8 (0.2)

These results are based upon multistage sequenced sampling of adult household residents living in eastern Baltimore at the time of the survey. The diagnoses were from independently conducted standardized psychiatric examinations and Diagnostic Interview Schedule assessments of the same subjects, who were selected by probability sampling from the community and also by probability sampling for the diagnostic assessments. These estimates take the varying probabilities of selection into account, as reported in Anthony et al., 1985, from which this table is adapted. Numbers in parentheses are the standard errors of the observed prevalence estimates.

weigh the relative public health importance of one health or social problem vs. another. For many years the allocation of resources to the mental health field has been constrained by an overemphasis on the study of death certificates, the causes of death, and prevailing mortality rates. During the twentieth century, there has been an increasing appreciation that conquest of communicable diseases yields a worldwide increase in the prevalence of mental and behavioral disturbances that disable but do not necessarily kill (e.g., see Gruenberg, 1977; Kramer, 1983; Levav, Lima, Somoza, Kramer, & Gonzales, 1989). Recent evaluations of the global burden of diseases take both prevalence and disability into account, drawing estimates of prevalence and levels of disability from epidemiologic surveys. These evaluations show major depression and alcohol use disorders to be at the forefront of the major disabling health conditions that face countries worldwide, now that more humans are living into adulthood when they otherwise would have died during infancy and childhood (Murray & Lopez, 1996). The same process of reduced mortality with increased survivorship has given Alzheimer's disease and the dementias of late life a much more prominent aspect than human society has ever faced before (Gruenberg, 1977).

The history of epidemiologic surveys of mental and behavioral disturbances dates back to the early nineteenth century. A famous Professor Holst of Norway is cited as having been responsible for the first formal community survey of mental disorders. This survey was completed in 1835 in order to assess the number of citizens who were suffering from mental

disturbances and how they were being attended. The method involved two stages of case assessment. In the first stage, the clergy were asked to review each roster of parishioners, which included all citizens of the area served by the church without respect to religious preference, and to make notes about the mental and behavioral characteristics of each listed person. In a second stage, these notes were compiled and reviewed by a royal commission of alienists (psychiatrists of the time), who sorted the disturbances into categories of idiotia, dementia, melancholia, and mania. Based on these assignments, the survey team then produced estimates for the prevalence of each of these conditions in the urban and rural areas of Norway, and for men and women (Table 3; Holst, 1835, translated by Massey, 1852).

In the United States, the first formal epidemiologic surveys of these disturbances were the responsibility of the Census Bureau. In the 1840 census, the census takers were asked to identify cases of insanity as they went from community to community, household to household, and institution to institution, with case assessment either directly observed or reported by household, community, or institutional leaders. Edward Jarvis, who was William Farr's American counterpart, compiled many of the results of the survey, with both good and bad results. Jarvis, a physician, was an early leader in the American Statistical Association, and his name should be remembered in this context for two important reasons.

First, Jarvis studied patterns of admission to mental institutions, and found that the probability of being admitted to such an institution

**Table 3** Prevalence estimates from an early community survey of mental disorders in 1835 in Norway.<sup>a</sup>

<i>Prevalence (cases per 10 000)</i>	<i>Mania</i>	<i>Melancholia</i>	<i>Dementia</i>	<i>Idiotia</i>
All citizens	6.05	5.31	4.35	14.21
Males	6.20	5.19	4.46	15.12
Females	5.91	5.43	4.25	13.34
Citizens of towns				
Males	9.27	5.69	5.60	7.97
Females	9.03	6.66	3.85	5.03
Rural districts				
Males	5.84	5.13	4.31	15.96
Females	5.52	5.28	4.30	14.38

<sup>a</sup>These prevalence estimates (cases per 10 000 population) are from a report written by Professor Holst of Norway, 1835, translated by Massey, 1852. As described in the text, the clergy of each parish in the country made notes about the mental life and behavior of each area resident. These notes were reviewed by a panel of specialists, who sought to identify the mentally ill, and to sort their disturbances into the categories listed above.

varied inversely with the distance of one's residence from the institution (Jarvis, 1865–66). This relationship, now known as “Jarvis’s Law” still held when it was checked in the mid-twentieth century (e.g., Kramer, Goldstein, Israel, & Johnson, 1955). Second, Jarvis was proud of the results of the 1840 census, and praised them as a major advance in the understanding of insanity in the United States. Illustrating his point, he used the census data to calculate the prevalence of insanity among “colored” persons living in the slave states of the South vs. the corresponding prevalence among those living in the free states of the North. In so doing, he found and reported that the prevalence of insanity in this group was lower in the South than in the North, only to discover, after his publication of the results, that serious errors had produced an undercounting of mentally disordered slaves in the South. Despite repeated retractions, and to his great dismay, Jarvis was unable to stop use of this statistical evidence in the proslavery arguments that ultimately led to the great Civil War between the North and the South (Deutsch, 1944; Jarvis, 1843a, 1843b).

Another neglected classic survey was conducted during 1916 by Aaron J. Rosanoff, a psychiatrist who worked in Nassau County, New York. Rosanoff’s two-stage community survey involved a sophisticated first-stage assessment designed to screen for mental and behavioral disturbances via brief tests and item sets (e.g., cognitive tests of orientation and memory, and proverb interpretation). In the second stage, Rosanoff or another psychiatrist colleague examined every one of the individuals suspected of having some mental or behavioral disturbance. After this direct examination, they

sorted the disturbances into discrete categories from the classification of neuropsychiatric conditions of the era, and calculated prevalence estimates for each of the categorized disturbances. Though the results of this early sophisticated community survey of mental disorders were used to help plan for mental health services in the county, the survey had an unfortunate origin in the eugenics movement. One of its stated goals was to identify families who were responsible for bearing offspring with mental and behavioral disturbances, and doing something about these families (Rosanoff, 1917).

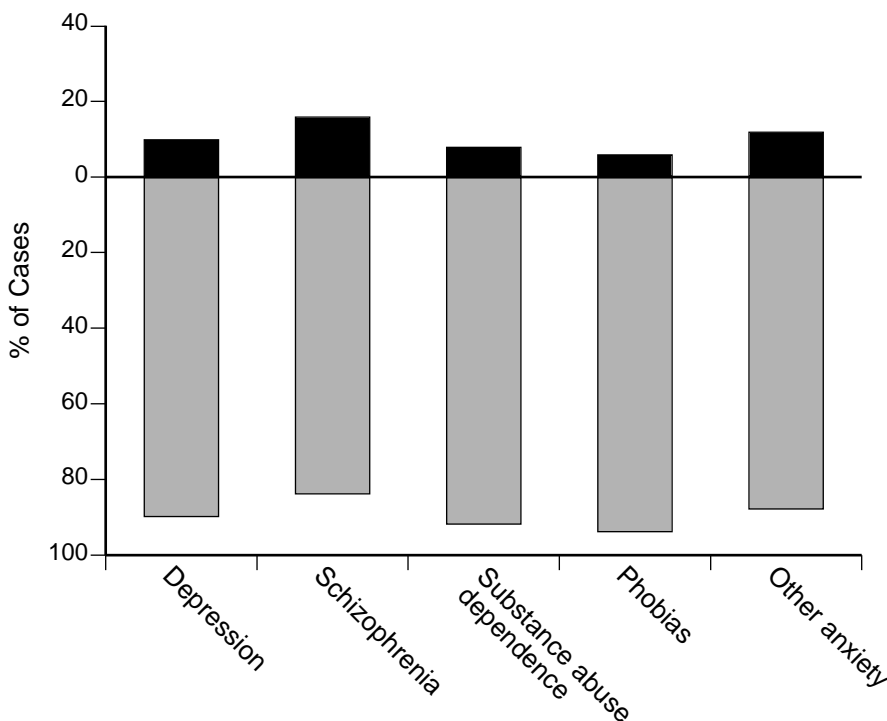
This chapter is too brief to provide a complete account of the history of community surveys of mental and behavioral disturbances. Many surveys of this type have been completed during the twentieth century, both in the US and abroad (e.g., see Cooper & Morgan, 1973; Dohrenwend & Dohrenwend, 1974; Lemkau, Tietze, & Cooper, 1942; Robins & Regier, 1991; Schwab & Schwab-Stone, 1978; Tansella et al., 1992). For the present purposes, it might suffice to say that the impulse to count the number of persons affected by mental and behavioral disturbances in the community was first expressed in scientific studies during the first half of the nineteenth century. This impulse grew to full strength during the twentieth century and continues unabated. First oriented to counting how many persons in the population are affected by each type of mental and behavioral disturbance (i.e., the prevalence of each disorder), this epidemiologic research has expanded to other domains. For example, via prospective and longitudinal research designs, epidemiologists now seek estimates for the risk of becoming a case for the first time (i.e., the

incidence of each disorder), and also seek to gauge the size of an “iceberg” phenomenon (i.e., the relative proportion of active cases who have come to the attention of health authorities vs. the proportion who remain without attention or care). Figure 2 illustrates the iceberg phenomenon, which might help convey how it is that epidemiology helps clinicians see beyond the horizons of clinical practice (Eaton et al., 1989).

Of course, once questions about treated and untreated cases of mental disorder are raised, it becomes necessary to account for why some cases are recognized and treated, while others are not. The transition from being a case in the community to being a recognized case and receiving clinical attention has been characterized in relation to filters that promote or impede these transitions. As identified by Goldberg and Huxley (1980, 1992), the progress of a case from the community toward psychiatric attention appears to be influenced strongly by the type and severity of the mental disorder (see Gallo, Marino, Ford, & Anthony, 1995; Marino, Gallo, Ford, & Anthony, 1995). In this expanded research mission, epidemiology turns to its second rubric.

### 1.12.3.2 Rubric 2: Where? A Question of Location

Either before or along with the capability to estimate how many persons in a population are dying or are falling ill, epidemiology can provide a means for making separate estimates for geographic locations or political jurisdictions, for different chronological ages or time periods, and for shared or individual circumstances such as social class and sex. In many circumstances, these estimates are presented in relation to dimensions of location. For example, one such dimension was the distance of one's residence from a mental hospital, as expressed in Jarvis's Law (Jarvis, 1865–66). Dimensions of socioeconomic status also have been prominent in epidemiological studies of mental disorders. For example, in one recent study, a research group led by psychologist Bruce P. Dohrenwend presented estimates for the occurrence of mental disorders in relation to a dimension of social status defined according to educational attainment. Separate estimates were presented for persons located at different positions along that dimension (Dohrenwend, et al., 1992).



**Figure 2** The “iceberg” phenomenon in the epidemiology of mental disorders. Data from the National Institute of Mental Health Epidemiologic Catchment Area (ECA) surveys, 1979–1985. The total area of each bar represents 100% of the active cases, in each of the listed categories of *DSM-III* disorders, found in the ECA surveys. The solid area above the *x*-axis represents the proportion of cases who had been seen by a mental health specialist (the “tip of the iceberg”), while the hatched area below the *x*-axis shows the proportion of active cases not seen by mental health specialists (Marino et al., 1995).

Often, the locational dimensions of time, chronological age, and stages of development have been of central importance in epidemiological studies. For centuries, epidemics have been observed to come and go, either varying in relation to the season of the year (e.g., influenza) or with much longer between-epidemic intervals (e.g., smallpox, cholera). Many communicable diseases such as measles, chickenpox, and mumps were discovered to affect mainly the young and rarely the old: a manifestation of durable immunity after an initial attack. While these epidemics raged among older children, newborn infants might be spared for a few months, due either to immunity acquired from the mother, or because of being swaddled or otherwise protected from contact with the infective agent. Those who managed to escape relatively mild disease during childhood might be faced with much more serious medical complications once the years of puberty were reached, as is the case with the mumps.

On occasion, the dimension of time serves as a marker for preventive actions, and early epidemiologists discovered that studies of disease occurrence over time might help them to understand the preventive value of these actions. For example, decades before Farr was able to rely upon Great Britain's national vital statistics data for calculation of mortality rates, Robert Watt had studied the burial registers of Glasgow, Scotland, to learn the annual numbers of smallpox deaths during the period 1783–1812. Watt observed generally declining numbers of smallpox deaths; proportionately, smallpox deaths became much less prominent among all deaths, despite generally increasing population size in Glasgow. Watt attributed the observed decline in the occurrence of smallpox deaths to the initiation of smallpox vaccination in that area (Watt, 1813, cited in Farr, 1885).

Nonetheless, in epidemiology, the question of where (and when) we are more likely to find cases occurring often runs ahead of questions about causes, mechanisms, and prevention or intervention, especially when there are no strong theories to guide more probing investigations. This has had some advantages, because intervention and prevention-oriented services can be mobilized toward the locations of incident and active cases, once there is some firm evidence about location, and even before there is good theory to guide causal investigations. This was true in the history of the HIV and AIDS epidemics, as noted above: interventions were directed toward the gay community, and especially toward those in the gay community who were engaged in high-risk sexual encounters tending to facilitate the spread of sexually transmitted diseases. The nature of the

connection with sexual encounters was not understood at all, but the capacity to start developing interventions directed toward apparently risky sexual activities was created early in the epidemic.

Sometimes we need to sustain basic descriptive efforts to learn where cases are more likely to be found, even when advances are being made in tests of competing theories about causes and causal mechanisms. This certainly is true in the instance of the epidemiology of suicide, which has been studied carefully for about 160 years, starting with William Farr's discovery of evidence both conforming with and contradicting Par M. Bronc's "social law" that "suicide is most common where education is most diffused: suicides and scholars increase in the same ratio." The supportive evidence was based upon a comparison of four jurisdictions in which it was possible to review marriage records in order to determine the proportion of persons who were able to write their names at the time of being married. Based on this indication of education in these four areas, it was found that the number of suicides per 100 000 population was 2.2 in the district with the lowest level of education and 10.9 in the district with the highest level of education. Of course, this type of epidemiological evidence was liable to the "ecological fallacy" in that it is operating at a unit of analysis that is more aggregate than individual. There was no assurance that it was the more educated persons in these districts who were committing suicide. Pursuing this line of reasoning, Farr found the contradictory evidence: namely, in a comparison of the number of suicides per 10 000 males, Farr found a value of 6.7 suicides per 10 000 among servants and coachmen, vs. a lower value of 4.9 per 10 000 among capitalists, professionals, and other educated men (Farr, 1838, reprinted 1885).

Later in the nineteenth century, Durkheim (1897) tested more complex theories of suicide in relation to social structural characteristics and social constructs such as anomie, using vital statistics data on suicide mortality rates. Nonetheless, we still have not come to a firm understanding of the causes and causal mechanisms of suicide (Monk, 1987). Moreover, in recent years, by plotting the age-specific suicide mortality rates over time, in the United States and in many other countries, it has been possible to spot an increasing occurrence of suicide deaths among young people and in late life, especially among elderly men in the United States (Moscicki, 1995). This work illustrates useful epidemiological efforts to identify the location of incident cases in terms of the increasing trends over time and in terms of these two age subgroups within the population.

With firm evidence that these are real and persisting increases, and not insignificant and transient phenomena, we have all the more reason to probe more deeply, to frame, and to test theories about causes, causal mechanisms, and the means of prevention.

Farr maintained his interest in suicide mortality throughout his career. Toward the end of his life, he completed an international comparison of suicide deaths and other deaths from violent means (e.g., homicides). In so doing, he found substantially greater numbers of suicides per million living persons in Switzerland (196), Prussia (134), Austria (113), and Bavaria (103), as compared to Ireland (21), Finland (34), Italy (37), and Scotland (37) (Farr, 1885). Farr was well aware that differences such as these might be explained by variation in the death certification process (Farr, 1885).

Complementing these nineteenth-century analyses to investigate where suicide mortality was more likely to be occurring, at that time there also were analyses on the location of active cases of mental disorders. We already have mentioned Jarvis's troubles in connection with his analyses of the 1840 US Census data on insanity among free and enslaved African-Americans in the various states of this country. The early nineteenth-century Norwegian survey included prevalence estimates for specific mental disorders, cross-classified for urban vs. rural parts of the country, and also by sex and age (Holst, 1852, translated by Massey), as presented in Table 3. In Wine's report on the 1880 US Census, detailed tables were presented to show both the numbers and proportions of US residents affected by insanity and idiocy in each state (i.e., the state-specific prevalence values), as well as the age of onset of these conditions, with cross-classification analyses according to the sexes, various ages, and social groups. The report also gave consideration to the excess frequency of insanity among foreign-born residents (3881 per million inhabitants) vs. native-born inhabitants (1509 per million), and to the hypothesis that foreign nations were shipping their insane citizens to the shores of the United States. Addressing this hypothesis, Wines reanalyzed the census data, showing that the excess was much reduced when a correction was made by subtracting persons under the age of 16 from the denominators of these prevalence values. This correction was motivated by his consideration of the age of first onset of insanity, which was observed quite infrequently prior to age 16 (Wines, 1888).

In the nineteenth-century literature on psychological medicine, there also were fairly sophisticated discussions of the methodological problems associated with making international

comparisons of mental morbidity statistics, as well as comparisons between the sexes and other variations in the liability to mental disorders (e.g., with reference to marital status). For example, in Tuke's *Dictionary of psychological medicine*, there is a serious critique of previous methods of calculating the relative liability to insanity of different communities, with an emphasis on the problem of whether the numbers of insane persons were counted in a methodologically comparable way across countries. In addition, considering whether there might be increases in the liability to become insane over time, Tuke (1892) noted:

A moment's consideration will show that the only proper test of the increase of mental disease is the proportion of first attacks to the population during different periods. First admissions [to mental institutions] are clearly not identical with first attacks, seeing that a patient may be admitted into an asylum for the first time, and yet have had one or more previous attacks of insanity.

From Rosanoff's survey in 1916 onward through the twentieth century, it has been uncommon for epidemiologists to estimate how many individuals are becoming affected by mental disorders, or how many individuals have become recently active cases, without also analyzing the variation to the prevalence or incidence of these disorders in relation to location in time, from place to place, or across circumstances. For example, in addition to reporting an overall morbidity risk estimate to age 56 for both sexes, in a nonconcurrent prospective study conducted on Bornholm Island (Denmark), Fremming (1951) estimated the morbidity risk of alcoholism for males as 3.41%, but was unable to do so for females because only two of the women in the study population had developed alcoholism.

In a comparison of this type, even when the goal simply is to locate where the cases are more likely to be occurring or more likely to be found, it is possible to take differences or to form ratios. Taking differences in Fremming's data on morbid risk of alcoholism for males vs. females, we can compute an excess morbidity risk of 3.0% or greater, given that the morbidity risk for males was 3.41% and that for women truly was too small to calculate. Alternatively, we can compute the ratio of the two morbidity risk estimates, which in this instance would prove to be far greater than a threefold excess in morbidity risk for males vs. females. In that the female morbidity risk value is close to zero, an estimate based upon the actual ratio of male morbidity risk to female morbidity risk would tend towards infinity.

In addition to these risk difference and risk ratio computations that are used to quantify the magnitude of variation from one subgroup to another, from place to place, and time to time, etc., we also can transform each morbidity risk estimate into an odds estimate, and then take the ratio of the odds. The term "odds," by conventional definition, refers to a probability divided by the complement of that probability. To the extent that we can ignore the fact that incidence rates are expressed in relation to units of time, if these units are kept the same for the groups being compared, we can convert the standard form of the incidence rate ("probability of becoming a case for the first time") into an odds value by treating its numerical value as a proportion, and by expressing the odds as that proportion divided by the complement of the proportion. Thereafter, we can divide the odds of becoming a case for one group (or time or place) by the odds of becoming a case for another group (or time or place). The resulting "odds ratio" also serves well as a statistical measure when studying variation in the location of occurrence of cases. Indeed, under some circumstances, the odds ratio approximates the risk ratio defined above (e.g., see Breslow & Day, 1980).

If the proportions under study are prevalence values from cross-sectional survey data, then it has become customary to use the terms "prevalence difference," "prevalence ratio," and "prevalence odds ratio" instead of "risk difference" or "incidence difference," "risk ratio," or "incidence ratio," and "odds ratio." Indeed, if the risk values are cumulative probabilities over some specified interval of time, then there is good reason to use the terms "cumulative risk difference," "cumulative risk ratio," and "cumulative odds ratio" in order to distinguish these statistical measures of variation from the corresponding measures based upon incidence rates.

The same comparative statistics to identify variation in the location of cases (e.g., estimates of prevalence odds ratios and cumulative risk ratios) can be derived from epidemiologic case-control studies, as well as from other comparative study designs related to the case-control paradigm. The case-control research design can be thought of as a special case of the sequenced two-stage (or multistage) survey sample design: in case-control studies we always sample on the outcome variable in the first stage of assessment. In a second stage of assessment, we measure locational grouping variables of interest in the cases and also in sampled controls (noncases). That is, we seek a sample of people who are becoming cases, and who seek a comparison sample of people we have not become cases.

In a case-control study, the values "observed" for the cases can be compared to the "expected values" observed for the noncases, much as a clinician might measure some characteristic of a case, and then make a comparison of this observed value to a table of reference values or "norms" for a reference population. If we are interested in making comparisons about the incidence of a specific condition, we see cases who have just become cases ("incident cases") and we compare them to individuals who have no past history of having become a case ("controls") or we compare them to the reference population out of which the incident cases have surfaced ("a sample of the study base"). In this instance, the magnitude of association between case status and the locational characteristic of interest is typically estimated using the odds ratio. However, it can be useful to think of the odds in a special way. For example, to conduct a case-control study of the association between sex (being female) and major depression, we might recruit a sample of incident cases of major depression as well as noncases with no history of major depression. Among cases, we estimate the odds that an incident case of depression is female, and among the noncases, we estimate the odds ratio that a noncase is female. Properly designed, the case-control study, with its sample based on the outcome variable (major depression), yields a ratio of these two odds that serves as a good approximation to the incidence odds ratio that would be derived from a prospective study in which the sample has been assembled with reference to the locational grouping variable of interest (here, "being female"), and with subsequent follow-up assessments to evaluate whether one group is more likely than another to become a new case of major depression. This is an important aspect of case-control study design that merits reflection: the odds ratio estimate from a case-control study often serves well as an estimate for the incidence odds ratio from a prospective study (Breslow & Day, 1980; Schlesselman, 1982).

Two central advantages of a case-control research design over a prospective study design are that the case-control study can often be completed with less time and effort, and a case-control study also allows testing of multiple hypotheses with a single sample. For this reason, the analysis of case-control study data is often organized as a test of one or more central hypotheses of greatest interest, followed by a more exploratory search for associations of less central interest. That is, once key hypotheses have been tested, analyses of case-control data can shift toward a more exploratory mode of investigation in which the aim is to screen for

new, previously unestablished patterns of associations that might serve as leads to causal hypothesis testing in future studies.

Nonetheless, the most powerful uses of the case-control study design have been observed when there is a need for rapid detection of leads for more probing causal investigations, when the supply of available cases is limited (i.e., sample size is small), and when there is no viable opportunity to use a prospective study design or a randomized controlled trial to test alternative hypotheses. This situation can arise when an epidemic outbreak of a disease occurs and there is an urgent need for a means of prevention or other public health action before a deliberate and long-term search for causes can be undertaken. In some instances, randomized trials are ethically impossible (e.g., to test whether fetal exposure to cigarette smoke via maternal smoking might increase a child's risk of later drug dependence). In other instances, the planning of prospective or longitudinal studies requires preliminary information best gained by relatively inexpensive and rapidly conducted case-control study methods.

Under circumstances such as these, and until effective means of public health action are well in hand, it makes good sense to be very enthusiastic about the prospect of completing multiple case-control studies that show where the new cases are located. At a minimum, these studies can yield new information and expertise which then can be harnessed within the framework of prospective studies to test causal hypotheses. The recent history of the international research program on Alzheimer's disease provides a useful example of this linkage from informative case-control studies toward more probing prospective investigations. Specifically, multiple case-control studies on senile dementia of the Alzheimer's type (SDAT) were conducted between 1975 and the late 1980s, and virtually all of these studies were based on cases referred to specialty clinics of neurology or neuropsychiatry. Estimated odds ratios from almost all of these studies pinpointed a past history of head trauma with loss of consciousness as a locational characteristic with excess occurrence of SDAT (Anthony & Aboraya, 1992; Breteler, Claus, van Duijn, Launer, & Hofman, 1992). Given the biological plausibility that head trauma at one stage of human development might cause an increased risk of SDAT at a later stage, and despite the clear possibility that referred cases might be a biased sample of all cases, investigators who learned the ropes of SDAT research via case-control studies now are conducting prospective and longitudinal studies, including longitudinal investigations of twins who are discordant for head trauma, in

order to probe the possible causal significance of this observed association. With the human species, these observational studies are the best we can do: a randomized controlled trial of head trauma as a cause of SDAT makes for an interesting thought experiment, but is out of the question.

### 1.12.3.3 Rubric 3: Why? A Question of Causes

The history of epidemiology's search for causes of mental and behavioral disturbances dates back to the prenumerate period when Ramazzini made observations about occupation-related diseases such as the ailments and premature mortality affecting hatters, medical workers, and others who handled mercury incautiously. As mentioned earlier in this chapter, in the middle of the nineteenth century, William Farr also turned his attention to the subject matter of psychology and psychiatry when he considered links between education and suicide, but stopped short of a probing causal analysis.

Nevertheless, by the middle of the 1800s, John Snow and others had taken Louis's numerical methods for the clinical study of patients and had adapted them for causal analyses of the probabilities and rates of diseases that affect populations. As practiced today, these adapted numerical methods encompass study design and analysis tools such as randomization, stratification, and matching. In the main, all of these refinements of numerical methods are used to create balance or comparability when we search for the causes of human suffering or the means to prevent or reduce this suffering.

For example, by randomly allocating patients to different treatment interventions, we seek to constrain the influence of possibly distorting influences such as sex or age in order to allow the effects of the various interventions to shine through. When we are careless in our work, we simply assume that randomization works to constrain the influence of these variables, even though randomization in any given trial actually might fail, and often does so. When we are careful, we check to make sure that randomization has worked to our benefit, looking to see that the distributions of potentially influential variables are balanced across our various intervention groups. Theory or prior evidence guides us when we choose which pre-intervention variables might be distorting influences on treatment outcome, and the means of assessing these variables. Checking to see that the distributions of these variables are balanced means that we look at statistical

indicators for each group in relation to the central tendency of the variables (e.g., mean, median), their dispersion (e.g., variance, standard deviation, interquartile range), skewness, and kurtosis, allowing comparisons within and between the groups to be made.

When we find that the hypothesized influential variables are not in balance across the groups to be compared (i.e., when the groups are “nonequivalent”), we have a number of options at our disposal. For example, we can rerandomize until balanced distributions are achieved. In this fashion, group equivalence can be achieved on all pertinent variables except for the interventions to be compared. Or, when rerandomization is not possible, we have the option of conducting a stratified analysis that will “hold constant” the influential variables when we seek to know whether the intervention had its desired effect. For example, if we think educational level might introduce a distorting influence on the trial results, and if we find out that one randomly allocated intervention group has proportionately too many college-educated patients, we can stratify in our analyses, making sure the distribution of the education variable is balanced within strata before making a contrast of intervention effects. Two special cases of stratification involve (i) matching, which is a kind of “fine-grained” stratification which we use to make sure that the matching variable has the same distribution within the groups to be compared, and (ii) exclusion, which amounts to stratifying and then eliminating from consideration any instances of a powerful distorting variable. For example, after completing all baseline assessments for a trial of a new psychotherapeutic intervention, we might find that, somehow, three individuals already taking a serotonin reuptake inhibitor slipped into one group, and two individuals taking this same antidepressant drug slipped into another group. In this instance, we might well decide to eliminate these individuals from the analytical sample, restricting our study and our analyses to the stratum of individuals not taking such antidepressant medicines.

All of these numerical methods for constraining the influence of possibly distorting variables were available to early epidemiologists and were used by them in their efforts to search for the causes of disease in populations. For example, once Farr had come to agree with Snow’s rejection of the miasma theory about the cholera epidemics, and to focus on water supply, he made a before-and-after comparison that sought to clarify the cause of an observed drop in cholera mortality rates, namely, a change in water supply between 1849 and 1854. In his reasoning, he illustrated methods of

bringing into balance and holding constant an area’s elevation above sea level (unchanged before and after), as well as the presence of poverty, crowding, and other potentially causal factors. As described in Table 4, unless taken into account, these factors (labeled **a**, **b**, **c**, **d**, **e**, and **z**) might have distorted Farr’s before-and-after comparison.

The twentieth century has added refinements to this application of numerical methods in epidemiological research, but it is hard to improve upon the logic of Farr’s original reasoning. Included among the more recent refinements are: a greater appreciation of the odds ratio as an indicator of how strongly one variable might influence another; widespread use of a multiple logistic regression model, that can be used to express the odds of disease occurrence in relation to set of potentially influential variables (corresponding to Farr’s **a**, **b**, **c**, **d**, **e**, and **z** in Table 4); improvements in the estimation of the odds ratio under conditions of matched samples; advances in statistical methods needed to estimate the odds ratio when multiple distorting influences must be kept in check; and software programming plus computational advances which now place these statistical innovations within reach of each individual investigator who has access to a personal or office computer, whereas less than 20 years ago large mainframe computers were required.

In concept, the multiple regression model serves a function analogous to that of stratification or matching in more elementary numerical methods. Namely, the idea is to apply multiple regression models in order to hold constant or adjust for the potentially distorting influence of other variables. Application of this method is not unique to epidemiology as a scientific enterprise, and some investigators in psychology have taken the multiple regression approach far beyond anything to be seen in the public health branches of epidemiology (e.g., see Bentler & Stein, 1992; Newcomb & Felix-Ortiz, 1992). Nonetheless, more than most fields, epidemiology now makes use of the multiple logistic regression model, fostered mainly by graduate training that teaches how to derive and interpret the odds ratio from fourfold cross-classification tables and to extract the same from logistic regression (e.g., see Breslow & Day, 1980; Hosmer & Lemeshow, 1989).

In its search for causes, epidemiology also has benefited from twentieth-century advances in the design of experiments, contributed by Sir Ronald A. Fisher and others. In this respect, epidemiology is no different from other scientific fields that make use of both experimental and observational research designs. However,



**Table 4** Epidemiologic reasoning in the nineteenth century.

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Farr's reasoning illustrated early methods of bringing into balance and holding constant an area's elevation above sea level (unchanged before and after), the presence of poverty, crowding, and other potentially causal factors which otherwise might have a distorting influence on the comparison. As expressed here in a reorganized outline, and with correction of two minor typographical errors, Farr summarized his reasoning as follows (excerpt from Farr, 1885, pp. 375–377):

- (i) The excess of mortality (**x**) by cholera in the Southwark and Lambeth water-fields in 1849 and 1854 was produced by one or other of all the possible causes existing in 1849, represented by **a, b, c, d, e, z**.
  - (ii) In the second period (1854), as well as the first (1849), all the possible causes remained unchanged, except the possible cause **z**, which varied.
  - (iii) With it varied **x** so as to diminish as **z** diminished. Therefore **z** was the cause of **x**.
  - (iv) If the enumeration of the possible causes is complete, and given the assumption that the forces of **a, b, c, d**, and **e** are substantially unchanged, it is difficult to resist the conclusion that **z** was the cause of **x**.
  - (v) The obvious possible causes are exposure to infection (**a**), bad drains (**b**), crowding (**c**), poverty (**d**), imperfect medical relief (**e**), impure water (**z**).
  - (vi) None of these, as far as it is known, differed, as far as Lambeth is concerned, in 1849 and in 1854, except the water (**z**), which was very impure in 1849, and much purer in 1854.
- 

the epidemiological case-control study design has been refined by epidemiologists through years of usage in this century, first in applications involving research on lung cancer, then on other cancers, mental retardation and other neuropsychiatric disorders of children (A. M. Lilienfeld, Pasamanick, & Rogers, 1955) and, more recently, on a broad array of diseases and health conditions, including schizophrenia, panic attacks, and major depression (Anthony, Tien, & Petronis, 1989; Dohrenwend et al., 1992; Link, Dohrenwend, & Skodol, 1986). Because it tends to be underappreciated outside of epidemiology, this particular research design merits some special attention.

As implied in our introduction to this topic, if the epidemiological case-control study design did not already exist, we would have to invent it. This is not mere boasting on behalf of epidemiologists and biostatisticians, such as Morton Levin, Abraham Lilienfeld, Nathan Mantel, Norman Breslow, and others, who have labored to develop and refine case-control methods. The case-control research design poses some of the most impressive challenges in epidemiology, but epidemiology could not respond to pressing public health problems without this design. An example might help drive home this point. The following illustration of the value of case-control research draws upon the experience of clinicians and epidemiologists who faced a recent epidemic of eosinophilia-myalgia syndrome (EMS), described in detail by Kilbourne (1992) and summarized here.

First identified as an outgrowth of the work of astute clinicians who compared notes on three

patients in October 1989, EMS was soon discovered to be affecting more than 1500 people in the United States alone, and with more cases abroad. The main laboratory finding on these cases was striking eosinophilia, increased eosinophils, and eosinophil precursors in bone marrow, but the main presenting ailment of affected cases was severe muscle pain, which generally was severe enough to constrain the patient's usual activities and often was disabling. A great majority of cases also complained of headaches, feelings of numbness or muscle weakness, and fatigue. As the case histories progressed, cognitive deficits associated with EMS were identified, as well as neuropathy due to axonal loss and documented by electromyography and nerve conduction studies. In some cases, the neuropathy was severe and progressive to the point of quadriplegia; some cases required mechanical ventilation to compensate for respiratory insufficiency secondary to the neuropathy. Over the course of a relatively brief epidemic period from 1989 to 1990, a small number of deaths attributed to EMS accumulated, but the epidemic was curbed and its apparent cause was identified in time to keep the EMS-associated mortality to a minimum. This rapid action is attributable to a series of very small case-control studies, all conducted within one month of clinical reports on the first EMS cases; that is, far more rapidly than it would have taken to mount standard longitudinal, prospective, or experimental investigations.

The clinicians reporting the first three EMS cases thought that the appearance of this syndrome had something to do with the patients' ingestion of oral L-tryptophan, an

amino acid precursor to serotonin, taken by them as a dietary supplement (e.g., with the intention of improving mood or reducing sleep problems). The reported syndrome brought other possibilities to the minds of epidemiologists at the US Centers for Disease Control (CDC). For example, an epidemic form of a very similar syndrome had developed in Spain during the early 1980s, ultimately affecting 20000 cases and accounting for 300 deaths. Traced by case-control and clinical methods, the Spanish epidemic ultimately had been linked to ingestion of rapeseed oil, though the specific causal mechanism or contaminant still remains unknown. Another suspected cause was the nematode *Trichinella spiralis*, which causes trichinosis when ingested via undercooked and contaminated pork. Indeed, a similar syndrome might appear as a consequence of neoplasms, and when no more than a handful of cases had been reported, this possibility could not be dismissed entirely.

Once the CDC investigators set up a system to collect clinical reports on cases of this syndrome, the known number of affected cases mounted. By July 1990, confirmed reports of EMS had been received from every state of the USA, and more than 1500 cases had been reported. Prevalence, calculated as the number of reported cases per million (cpm) population, was greatest in New Mexico and Oregon (>20 cpm population), but also was high in South Carolina (15.1 cpm) Minnesota (14.5 cpm), Colorado and Arizona (13 cpm), California, Idaho, and Washington State (9–10 cpm). The lowest value was from Louisiana, with only 0.2 cases per million population.

Two case-control studies, one in New Mexico and one in Minnesota, proved to be pivotal in establishing an empirical link between EMS and oral L-tryptophan, despite a background of observed associations with other factors, and suspected causes such as trichinosis and cancer. For example, from the early case reports, a female excess was apparent: 84% of the reported EMS cases in the US were women, 97% were Whites not of Hispanic origin, the median age of cases was 48 years, and few cases were under 25 years of age. In the New Mexico case-control investigation, the history of ingestion of dietary L-tryptophan and 32 other suspected etiologic factors was ascertained for 11 identified cases, along with 22 neighbors of the cases of the same sex and age groups; in the Minnesota case-control study, the characteristics of 12 identified cases were compared with 12 noncases matched on sex, age, and telephone exchange. In both studies, the associations between ingestion of tryptophan-containing nutritional supplements were expressed in

relation to huge odds ratios: all of the cases in both studies had ingested tryptophan-containing products; fewer than 10% of the noncases had done so. Furthermore, only one of the 85 cases reported to the CDC by early November 1989 was found to have no prior history of ingesting tryptophan-containing products.

On the basis of this evidence, because other suspected causes had been ruled out, and because no biases appeared to account for the observed EMS-tryptophan associations, the US Food and Drug Administration (FDA) asked manufacturers and suppliers for a voluntary recall of tryptophan-containing products, effective November 17, 1989—less than six weeks after the original report on the syndrome! Thereafter, the number of reported EMS cases declined sharply.

Although there were more than 1000 manufacturers of tryptophan at that time, it was discovered that six Japanese companies were responsible for essentially all of the dietary tryptophan consumed in the United States during 1989. Furthermore, adaptations of the case-control strategy narrowed the search to a single Japanese manufacturer and to retail lots of dietary tryptophan that had been produced from this manufacturer's supplies between mid-1988 and mid-1989. Whereas other manufacturers might have produced tryptophan that caused EMS, the risk of EMS was shown to be markedly greater among users of a single manufacturer's retail lines.

Securing the cooperation of the Japanese company, the epidemiologists sorted batches or lots of tryptophan in relation to whether they were found to be linked to one or more cases of EMS or to no cases of EMS. Tryptophan batches linked to the EMS cases were more likely to have involved changes in standard fermentation and purification processes. Two of several changes were implicated: less powdered activated carbon had been used in product purification, and in addition, a new strain of *Bacillus amyloliquefaciens* had been used for fermentation. Subsequent chromatographic studies were used to identify a possible contaminant, apparently the result of using the new strain of bacterium for fermentation, and this contaminant (EBT) is now regarded as a candidate for the etiologic agent in this epidemic of EMS. (However, subsequent to the end of the epidemic, randomized controlled trials of EBT exposure in Lewis rats showed that EBT can cause some but not all of the histopathological changes associated with EMS.)

Although some postepidemic cases of EMS have been reported, and it is claimed that there are other causes of EMS besides badly manufactured dietary tryptophan (Spitzer

et al., 1995), the 1989–1990 epidemic subsided to negligible levels within months of the FDA regulatory action (Kilbourne, 1992). Hence, dramatic reductions in the incidence of EMS following the FDA action constitute additional important evidence that the root causes of this epidemic involved dietary tryptophan in some way.

As exemplified by the story of the eosinophilia-myalgia syndrome, there often are public health emergencies that cannot be addressed quickly without case-control methods. Because they seem to be developmentally backward and sample on the outcome, epidemiologic case-control study methods are often criticized by behavioral scientists who seem not to appreciate how this tool can be used complementarily with other research strategies. However, case-control studies have a demonstrated value in public health emergencies such as the EMS outbreak and have also helped to propel etiologic research on Alzheimer's disease and other nonemergency public health problems in a manner that has facilitated the design of subsequent prospective and longitudinal investigations. For a fraction of the cost of other investigations, the epidemiological case-control study can yield invaluable information needed to plan prospective and longitudinal studies (e.g., estimates for the effect sizes needed to determine sample size values in prospective studies, or empirical evidence on plausible but possibly unimportant confounding variables). Under these circumstances, it often is imprudent to initiate fieldwork in expensive longitudinal or prospective studies until after epidemiologic case-control research has been completed.

Three emerging lines of case-control research on mental and behavioral disturbances deserve special note because they are likely to appear with increasing frequency in the research literature of interest to clinicians in the professions of psychology and psychiatry. First, in essence, studies of discordant twin pairs are sometimes special cases of case-control research: in these instances, a twin who has been affected by a disorder is found, and subsequently there is an effort to determine whether the index twin's co-twin has also been affected. These twin studies hold constant inherited characteristics (in the case of monozygotic twins) or allow them to vary (in the case of dizygotic twins) in order to identify aspects of the environment that might modify inherited vulnerabilities or that might affect occurrence of mental and behavioral disturbances independent of inheritance. The same logic applies to studies of discordant sibling sets in general. What makes them epidemiological (if they are at all) is that there is a deliberate search beyond

treated cases to assess who has become sick and who remains well, and to determine what might be causing this variation in the occurrence of sickness. What makes them examples of the case-control study design is that they sample on the outcome by starting with a sample of affected twins.

There now are many such twin studies of mental disorders and a growing number of discordant sibling studies. For many years, twin researchers were preoccupied with establishing whether inheritance had anything to do with the etiology of mental and behavioral disturbances, and the focus of attention was on a question of genes vs. environment or on estimation of the heritability of a disorder or a trait. More recently, the questions have looked beyond the more basic issue of whether inheritance matters. Instead, the hypotheses are framed in terms of which genes might account for observed inheritance of mental and behavioral disturbances or traits, and how genes and environment might combine in dynamic interplay to influence the development of these disturbances and traits. Simply by way of illustration, the twin studies of Breitner et al. (1995) provide a starting point in relation to the epidemiology of Alzheimer's disease. The work of Pickens, Svikis, McGue, Lykken, and Heston (1991) and Johnson, van den Bree, Uhl, and Pickens (1996) illustrate the case-control variety of twin study research as applied to alcohol and other drug dependence.

In a second line of case-control research, inter-related with twin studies, are recent tests of etiologic hypotheses about specific genetic polymorphisms. These experiments are especially well-served by the epidemiological case-control study design because the nature of the suspected causal associations has a temporal ordering that finesses some of the interpretive challenges sometimes faced in case-control studies. Namely, it is hard to confuse the temporal sequence that leads from genes and gene expression toward mental and behavioral disturbances, even when the underlying theory specifies complicated interactions between genes and environment. Once they have been used to establish a well-replicated linkage between specifically mapped genes or gene markers and subsequent mental or behavioral disturbances, adaptations of the case-control design can be used to test targeted hypotheses about how environmental conditions or processes might modify or interact with these genetic influences. This can be accomplished in the same way that case-control methods had to be adapted to evaluate which manufacturing processes might have changed the risk of EMS and caused that epidemic to occur.

Again, providing a starting point to understand this line of research, the first studies to investigate polymorphisms associated with the dopamine D2 receptor gene and their suspected causal linkage with severe alcoholism and cocaine dependence represent applications of the epidemiological case-control study design (Blum et al., 1990; Uhl et al., 1993). The continuing debate over these studies mainly has to do with the controversial selection of their noncase series ("controls"). Applying epidemiological strategies within the framework of family-genetic studies, and via case-control analyses of the resulting study data, Strittmatter et al. (1993) at Duke University and Henderson et al. (1995) at the Australian National University in Canberra have made advances in our understanding of polymorphisms linked to a gene for apolipoprotein epsilon-4 and their suspected causal or protective associations with Alzheimer's disease.

In a third line of research with importance for clinical psychology and psychiatry, the epidemiological case-control study design has been used with increasing frequency to analyze the evidence from large-sample epidemiological studies in a manner that holds constant shared aspects of local neighborhood environment in order to test hypotheses about how personal and individual characteristics might account for excess risk of mental and behavioral disturbances. The opportunity to use the case-control study design in this context emerges from a feature of the multistage area probability sampling plans that are used to draw these study samples. That is, these plans first sample from lists of primary sampling units (PSUs) which sometimes are as large as states or large metropolitan areas. From within the PSUs, there is another stage of probability sampling from blocks, block groups, census tracts, or neighborhoods within which there are multiple households or dwelling units; these "neighborhood"-level aggregations then are sampled. From within the sampled "neighborhoods" there is an additional stage of probability sampling of listed households or dwelling units, and then from within the households, there is a listing of all eligible study participants, and a random sample of these is taken in order to designate a participant for the study. Once sampling and recruitment has occurred, the study's assessment procedures are undertaken, either cross-sectionally or prospectively, and when mental disorders are under study, it is possible to identify those who have become cases as well as those who have not. Followed prospectively over time, and reassessed, those who have not become cases at the time of the baseline assessment are at risk of becoming incident cases.

Typically, over the span of a standard follow-up interval in these prospective studies, very few candidates become incident cases during the follow-up interval. In consequence, within any given sampled neighborhood, it is possible to compare and contrast the candidates who remain noncases with those who become incident cases. Thus, by using the conditional form of the logistic regression model or other numerical methods that treat the neighborhood-level variable as a matched characteristic, an investigator is allowed to hold constant shared aspects of living in a particular neighborhood (e.g., social disorganization, disadvantage, street-level availability of drugs, police presence). At the same time, it is possible to estimate the risk of becoming an incident case in relation to a suspected causal factor that has been measured at the personal or behavioral level.

This form of epidemiological case-control study design has been used to test a variety of hypotheses about suspected causal factors for mental disorders and behavioral disturbances:

(i) Whether an individual's use of cocaine might increase risk of panic attacks (Anthony et al., 1989), hallucinatory or delusion-like experiences (Tiens & Anthony, 1990), and obsessive-compulsive behavior (Crum & Anthony, 1993).

(ii) Whether failing to achieve social expectations for education and schooling increase the risk of developing *DSM-III* alcohol dependence or alcohol abuse in adulthood (Crum, Bucholz, Helzer, & Anthony, 1992), or the risk of developing major depression in middle age or later life (Gallo, Royall, & Anthony, 1993).

(iii) Whether jobs with high levels of physical or psychological demands and low levels of personal control and autonomy might increase risk of drug dependence in adulthood (Muntaner, Anthony, Crum, & Eaton, 1995).

Even the data from a cross-sectional survey can be organized within the framework of a case-control study and used to investigate questions about causation if an investigator will take care to gather the data carefully and analyze the results with attention to possible distorting influences. For example, one of the most informative recent studies on the issue of social causation of mental disorders was completed, in essence, as a cross-sectional study, but with case-control analyses of the resulting study data. The hypotheses to be tested in this study have long occupied the attention of epidemiologists, who were able to notice, even in mental hospital statistics, that individuals with mental disorders often seemed more socioeconomically disadvantaged than one might expect (e.g., see Faris & Dunham, 1939). A challenging question has been whether it is the disadvantaged social

conditions that increase the risk of mental disorders or whether it is more that the mental disorders cause individuals to be held back or even to lose social status. For example, lifelong social disadvantage would tend to hold people back from acquiring prestigious social status (e.g., as reflected in years of schooling, advanced degrees, and educational attainment in general). Accordingly, any socially disadvantaged person who managed to achieve high social status such as a good education might be an especially healthy and adaptive person, with life chances relatively unencumbered by mental disorders. Contrariwise, any socially advantaged person who failed to achieve high social status might more likely be one whose life chances had been encumbered by mental disorders. Evaluated at each point along a gradient of social status (e.g., educational attainment), social causation might result in higher risk of mental disorders among persons with lifelong social disadvantage due to discrimination based on race or ethnicity when compared to persons without such disadvantage. Social selection might result in higher risk of mental disorders among persons with lifelong social advantage when compared to persons without such disadvantage (Dohrenwend et al., 1992).

Presenting this analysis of the selection-causation issue, Dohrenwend et al. (1992) studied Jews living in Israel, cross-classifying them in relation to their membership in groups whose African family heritage, physical appearance, and ethnicity might place them at a lifelong social disadvantage. In addition to making standardized assessments of their lifetime histories of mental disorders, the research team evaluated each study participant's social status in relation to educational attainment, and also measured other suspected influences on mental life and behavior (e.g., age, sex, marital status). In a careful analysis of occurrence of different types of mental disorders in groups cross-classified by disadvantage attributed to ethnicity and socioeconomic status, these investigators found patterns of association consistent with a more prominent influence of social selection in relation to disorders such as schizophrenia. However, the evidence was consistent with a more prominent influence of social causation in relation to disorders such as the substance use disorders. That is, the evidence on schizophrenia and related disorders indicated social selection mechanisms at work: socially disadvantaged persons with high educational attainment actually were less likely to have developed schizophrenia than socially advantaged persons with high educational attainment. In contrast, all along the gradient of educational attainment, the estimated risk of having developed substance

use disorders was higher than expected for the socially disadvantaged when compared to socially advantaged persons. Case-control analyses based on the multiple logistic regression model were completed in order to adjust for other suspected distorting influences such as marital status, but the results from such analyses did not alter these inferences from the study (Dohrenwend et al., 1992). This important investigation serves as a good illustration of how a cross-sectional epidemiological study can do more than estimate how many people are affected, and where within the population the affected cases are more likely to be found. With careful reasoning, assessment, and analyses, this study shed new light on an important enduring issue about the causes of mental disorders, and identified some important new issues that can be probed more thoroughly in future prospective and longitudinal studies.

An often overlooked feature of the case-control study design is its capacity to make efficient and rapid use of study resources. In concrete terms, many prospective studies of mental disorders have involved samples of literally thousands of participants. Upon follow up, no more than a small fraction of these participants has developed into an incident case of any specific mental disorder. In the resulting analyses of all the study data, there are hundreds of noncase control participants for every incident case of mental disorder. This can represent an unnecessary and inefficient allocation of study resources in that statistical power in case-control hypothesis testing reaches near-maximum levels once the ratio of noncases to cases is 10:1 (Schlesselman, 1982).

Rather than computerizing and analyzing the study data on the thousands of participants, it would be justifiable and efficient to apply case-control reasoning, namely to identify the incident cases plus 10 noncases for comparison with each incident case, and to computerize and analyze the study data on each set of 11 participants. If useful, the noncases could be matched to the cases on possible distorting variables such as age, sex, or neighborhood, in order to hold these characteristics constant and focus attention on suspected causal factors at the personal or behavioral level.

The utility of this form of "nested" case-control analysis becomes especially clear in targeted research when the hypotheses involve laboratory work for genetic studies, biomarkers, or other bioassays, or when each participant's record includes data that must be reviewed in detail and coded. For example, in large-sample epidemiological research, it has become quite common to ask each of the thousands of participants for permission to draw a blood

sample or to take a buccal swab sample of tissue, which can be frozen and archived, and from which sufficient DNA can be extracted to test for homozygosity or heterozygosity with respect to specific genes or genetic polymorphisms. Although less common, there also are epidemiological studies with large samples in which videotapes of interviews or family interaction patterns are made and archived, in an effort to characterize facial expressions of each participant, or to evaluate dynamic interactional processes such as coercive interactions between family members. Along the lines of case-control reasoning, a case-control study can be nested within these large-sample studies. When cases from these studies are compared with noncases, it is possible to obtain efficient and unbiased study estimates by drawing case material from the stored archives (e.g., the frozen blood sample, or the archived videotape), along with material on 3–10 noncases for each case, and to conduct the detailed and intensive assays only on this subset of the thousands of participants. In studies of bioassays or videotape coding of facial expression or family interaction, this might mean no more than 100–500 assays, vs. the thousands that otherwise would be required in order to obtain the assay data on all the participants. The “nested” case-control design and associated statistical issues have been described by Checkoway and Demers (1994).

As implied throughout this section, we have concentrated on the epidemiological case-control study design because its advantages for etiological research are often overlooked by investigators whose own graduate research training has led them to focus on either cross-sectional surveys, prospective and longitudinal research, or experimental design. Nonetheless, as shown in examples of cross-sectional survey research, what distinguishes an epidemiological investigation that answers questions about causation is not to be found in the investigator’s choice of a specific design, but rather in the combination of research design with numerical methods used to analyze study data and to interpret the resulting evidence. In an investigation oriented toward the first two rubrics of epidemiology—quantity and location—there is no need for detailed attention to the issue of whether the study subgroups, or the cases and noncases, might be balanced with respect to potentially distorting influences. There is no reason to master or apply techniques of randomization, stratification, matching, exclusion, or statistical adjustment that can be used to bring these influences into balance. What characterizes epidemiological research on the question of causation is attention to this issue of balance, and to the appropriate use of techni-

ques that can bring distorting influences into balance, so that we might gain a clearer view of what accounts for some individuals becoming cases while other individuals are spared.

#### **1.12.3.4 Rubric 4: How? A Question of Mechanisms**

Epidemiologists turn to the wishy-washy concept of a risk factor whenever there is a constraint on our understanding about the causes and causal mechanisms that influence the risk of ill-health or disease. For example, on occasion, the available evidence highlights locations in space or time, or within subgroups of populations, where cases are more likely to be found. Depending upon the focus on prevalence or incidence, the observed associations will be described in terms of prevalence or incidence correlates. With these circumstances, nothing more than a correlation is implied; causation clearly is not established.

As this evidence is worked up, it sometimes becomes possible to point toward characteristics, conditions, or processes that seem to have some influence on the risk of becoming a case, but uncertainty remains. There is an unwillingness to finger a specific cause or causal mechanism, but the accumulated evidence implies an association that has some causal significance, at least in theory. Under these circumstances of still limited understanding, epidemiologists would like to say that a cause or a causal factor has been identified, but instead they will pull the punch and say that they have identified a “risk factor.”

The late lamented Abraham Lilienfeld, who served at Johns Hopkins as Professor and Chair of Epidemiology for many years, and later as Professor and Chair Pro Tem of the Mental Hygiene Department, introduced one of the present authors to this idea that the term “risk factor” reflects a limited understanding of causation and causal mechanisms. When asked about the origins of the term, his memory led him back to the 1950s and the Framingham Heart Study team’s original observation that the risk of heart disease was found to increase with increasing age as that study’s participants were followed forward in time. At the time, he said, the investigators did not wish to claim that age caused heart disease, as if heart disease were an inevitable consequence of growing old and there was nothing to do about it. At the same time, they inferred that increasing age was an important marker of age-related causal characteristics, processes, or events that might be altered in order to reduce the risk of heart disease, and they did not wish to throw away the

idea that some modifiable aspect of the aging process might become an appropriate target for successful intervention and prevention. In this respect, the term risk factor represents a felicitous choice of words because, by etymology, a "factor" can be taken to represent a force that might turn out to make a difference: it is possible that it has causal significance, either as an independent cause, a contributing cause or cofactor that works conjointly with other causes, or as an inert marker that runs along with some other important causal factor in a complex causal chain.

For present purposes, the origins and etymology of risk factor are less important than the array of distinctions to be drawn between prevalence correlates and incidence correlates on the one hand, and between risk factor and "cause" or "causal mechanism" on the other. The distinction between prevalence correlate and incidence correlate refers to variation in the epidemiological indicator of disease frequency or occurrence. If the observed association involves something that is associated with being a case or having become a case, then it involves a prevalence correlate. If the observed association involves something that is associated with becoming a case, then it involves an incidence correlate. Some prevalence correlates might also prove to be incidence correlates, but many prevalence correlates are neither risk factors nor incidence correlates. Rather, they are consequences of having become a case, or they are associated with the duration of remaining a case. For example, receiving clinical services from a mental health specialist is associated with being a case of a mental disorder: active cases are more likely to receive specialty services than noncases (Marino et al., 1995). If we are to make sense of the terms incidence correlate and risk factor then attention to the issue of temporal sequencing is required: the risk factor appears first, and becoming a case comes later.

Within epidemiology, the "incubation period" or "induction period" that precedes occurrence of a case serves as a useful heuristic and reminder of the temporal sequencing between the risk-promoting characteristic, event, or process, and the later occurrence of clinically apparent signs and symptoms. The domains of clinical psychology and psychiatry hold some direct analogies for the incubation period of communicable diseases. For example, with respect to post-traumatic stress disorder (PTSD), the signs and symptoms of the disorder can be observed shortly after exposure to the traumatic event, or the induction period for the clinical syndrome might last for a long period, as observed among wartime veterans in relation to "delayed-onset" PTSD occurring months or

even years after exposure to combat-related trauma. With respect to dependence upon various drugs, there are measurable induction periods from the initial "exposure opportunity" until the first use of a drug, for instance, as measured in terms of the first time a youth has a real opportunity to smoke a marijuana cigarette until the very first smoking experience (Anthony & Helzer, 1995; Van Etten, Neumark, & Anthony, 1997). Afterwards, there is a measurable induction period from the time of first drug use until the appearance of the initial drug problem (e.g., see Anthony & Petronis, 1995) or until the appearance of a fully developed clinical syndrome of drug dependence.

Within the set of "incidence correlates" we have "risk factors" and we have "causes" and "causal mechanisms." The distinction between "risk factor" and "cause" or "causal mechanism" refers to an investigator's judgment about whether the available evidence is sufficient to warrant to say more than "it seems like this might be making a difference in who becomes a case and who does not." When an investigator is willing to say no more than this, then the term "risk factor" is warranted. When the evidence is sufficient to allow investigators to make a causal inference, to point a finger at a specific characteristic, condition, event, or process, and to name it as a "cause," then there is no need to appeal to the wishy-washy concept of a "risk factor"; they might as well call it a "cause" or a "causal factor." When they have marshaled suitable evidence to allow them to point out specific sequences or linkages of states and processes that influence who becomes and remains a case and who does not, then they have clarified our understanding of at least some aspect of "causal mechanism," which remains one of the highest and most esteemed goals of the scientific enterprise of public health work.

Epidemiology's history of conducting empirical investigations that probe causal mechanisms dates back at least to the 1840s, when Peter Ludwig Panum, a Danish pathologist, studied an outbreak of measles on the Faroe Islands of the North Atlantic. Though the successive eras of bacteriology and virology had not yet begun, and no one had any idea that measles was caused by a virus, Panum recorded the details of how measles swept through the Faroe Islands, recording notes about the spread from person to person, from place to place, and over time. Coupled with his close observations and careful reasoning, what allowed Panum to shed new light on the epidemic spread of measles was the fact that this was essentially a "virgin" population, in which some 65 years had passed since the last recorded measles epidemic and which therefore had no immunity to speak of. In

contrast to prior observers, who had watched measles epidemics where many population members already had acquired immunity from past infection and therefore did not become cases, Panum was able to see that more than 70% of a population would contract measles when periods as long as 65 years passed between outbreaks. He appreciated that measles was spread by person-to-person contact, but also hypothesized a mechanism that now is considered to be uncommon but not impossible: namely, measles is spread when noncases come into contact with clothing worn by a case (Panum, 1846, translated by Hatcher & Dimont, 1940).

Of course, later in the nineteenth century, the scientific contributions of Louis Pasteur and Robert Koch created a bacteriological revolution that helped to clarify the causal mechanisms by which measles and other communicable diseases might be spread, from person to person or by other means. Even so, many years passed from the time that specific infectious agents were identified until it became possible to use antibody tests in order to clarify which individuals had come into effective contact with an infective agent (sufficient to produce an antibody response), but had not developed manifestations of disease and were clinically inapparent cases. Analogously, Mendel's advances in genetics were not widely appreciated until after the chromatids had been identified as specific mechanisms of genetic inheritance late in the nineteenth century; once this aspect of inheritance was illuminated, additional work in genetics led to increasing advances (McKusick, 1996), but also to efforts at social control as reflected in the eugenics movement.

As described early in this chapter, it was before the bacteriological revolution and later discovery of filterable viruses that John Snow and other epidemiologists were able to begin to distinguish diseases whose spread was primarily person-to-person, such as smallpox, from those emanating from a common source. The prototypical common source was the infamous Broad Street well and pump in London, which John Snow identified as a source of cholera during the London epidemic of August 1854. Snow learned that nearly all of the deaths from cholera in south London had occurred among residents living within a short distance of that shallow well, and he had linked drinking of the well's water to cholera mortality as far away as Hampstead, where a former resident had bottled water from the pump conveyed to her each day. Although the handle of the pump was removed in early September, the epidemic had already started to die down, and the pump was back in operation soon after the epidemic subsided—

perhaps because no one had a clear understanding of what was contaminating the well water and how it had become contaminated. This aspect of causal mechanism was clarified later by one of Snow's colleagues, who learned that the cholera epidemic had started soon after the death of a local baby from diarrheal disease. Helped by the residents, he located a cesspool less than three feet from the well, into which the baby's discharges had been thrown. Upon inspection, the cesspool and well were discovered to be constructed so poorly as to allow the cesspool's contents to flow through the well's faulty brickwork into the water supply. This prompted repair of the well, but it was not until the threat of a new cholera epidemic in 1866 that the Broad Street pump and shallow well were finally disabled (Chave, 1958).

Attempts to identify the more microscopic aspects of the causal mechanism of cholera started around 1849, when Snow first articulated his theory of waterborne infection. These efforts included early experiments in which rodents were fed fecal matter and other material from cholera victims. Nonetheless, this early "animal model" of cholera failed, and it was not until 1876 that Koch isolated *Cholera vibrio* and identified it as the specific microscopic causal agent for cholera. Of course, even after this success, aspects of the causal mechanism eluded Koch, including knowledge of what might account for some individuals being exposed to *C. vibrio* without ever developing cholera, as well as the information that oral rehydration therapy is sufficient to prevent many of the deaths from cholera once it occurs.

Several of the tools used by early epidemiologists to distinguish between common source epidemics and those spread by person-to-person contact can be traced directly back to the numerical methods of data analysis and careful study of clinical cases that had been taught to William Farr and others by Pierre Charles Alexandre Louis. One of these tools involved plotting the epidemic curve in relation to the day-to-day and hour-to-hour occurrence of new cases; that is, a frequency distribution of cases by the time of onset. As Farr discovered, diseases such as smallpox that are mainly spread by either direct or indirect person-to-person contact have an epidemic curve that grows slowly toward a peak and then gradually subsides, in accord with equations that could be replicated from epidemic period to epidemic period (Farr, 1885). In contrast, outbreaks emanating from a common vehicle or source, without any elaboration via person-to-person spread, typically rise quickly to a peak and then drop off quickly as well because there are no secondary cases following exposure to each primary case. The epidemic



curve of eosinophilia-myalgia syndrome followed this kind of trace, consistent with a common source in the contaminated dietary tryptophan products which were quickly removed from the market, although not showing the more explosive rise and fall of the epidemic curve that can be seen when there is common-vehicle spread of disease after a single exposure at one point in time. Within the domain of clinical psychology and psychiatry, post-traumatic stress syndromes after a mass disaster such as an earthquake often follow this "common source" type of epidemic curve, although some of the occurrence of these syndromes is delayed in onset, which tends to extend the peak of the epidemic curve.

Epidemiologists soon learned of complications to be faced when attempting to understand causal mechanisms of disease spread by studying epidemic curves. In some instances, these complications now can be seen as manifestations of immunity and a mechanism of antibody response that had been carried over from exposure to an infective agent during a previous outbreak or epidemic. In the case of measles, for example, mechanisms of acquired immunity prevent individuals with past exposure from developing measles, and this can lead to a lower peak and more rapid decline of the epidemic curve when the between-outbreak interval was short; the susceptible individuals either were surrounded by persons with acquired immunity who were not exposing them to the virus, or were becoming cases rapidly. As evidence that the epidemic had "burned out" all the susceptibles in a community's households, the epidemic would end.

In other instances, the interpretation of epidemic curves can be complicated when the outbreak starts from a common vector, but subsequently there is person-to-person spread. The result is often an epidemic curve that rises to a peak but then falls slowly in conjunction with the elaboration of cases secondary to person-to-person spread. For example, salmonella might be introduced into an institutional setting via food (e.g., bulk eggs or poultry, unpasteurized milk products), and then spread among patients or inmates via person-to-person contact and sometimes by fomites that have come into contact with an infected person and convey the bacteria to another.

Shortly after the turn of the twentieth century, epidemiologists like Henry Value Chapin developed new tools for studying the mechanisms that account for outbreaks and epidemics of disease. Chapin recognized that it was important to take the temporal sequencing of incident cases into account when studying the spread of diseases within families or other

groups of people. His observations led to specification of an epidemiological tool known as the "secondary attack rate," which has been extremely useful in epidemiological studies aimed at identifying familial and other group-wise aggregations of disease, and at investigating mechanisms that might account for the observed aggregation.

In operational terms, investigating the familial secondary attack rate requires an attempt to identify the first or "index" case that surfaces within each family, and to identify all of the remaining susceptible individuals in the family in order to determine which of them becomes a subsequent or "secondary" case (after the index case). The timing of the occurrence of the secondary cases relative to the index case and relative to other secondary cases is also studied. Among those who do not become cases, it is necessary to attempt a distinction between individuals who have a previous history of being affected by the condition (e.g., on the basis of past history of symptoms and signs) and those who have no apparent history but remain susceptible to future occurrences. Once these details have been investigated, the secondary attack rate for each family can be estimated as a conditional probability by dividing the number of cases who are secondary to the index case in a given family by the total number of susceptible persons in that family, setting aside the experience of the index case and of all persons with a past history of the condition. If necessary, the time of onset of each case can be used as a stratifying variable in order to express the probability of becoming a case in relation to units of time. Thus, the secondary attack rate is related to the conditional probability of becoming a case during some specified time span, given that an index case has occurred and that there is no prior history of the condition. Especially when an infection leaves few inapparent cases (i.e., all infected persons develop some form of clinical expression of the infection), the secondary attack rate can be especially useful. The magnitude of a secondary attack rate helps to clarify whether familial aggregation is occurring at all, and also can be used to narrow the search for specific causal mechanisms and agents of disease.

As might be surmised, years of accumulated experience with various diseases have left epidemiologists in a position to recognize the specific "signatures" of various diseases that are disclosed in their epidemic curves and in indicators such as incubation periods and secondary attack rates. These signatures allow epidemiologists to start narrowing the search for causal agents and mechanisms, even before it is possible to complete laboratory studies that

might be used to isolate factors such as bacteria, viruses, and contaminants of diet or poisons. Part of the signature consists of the clinical features in the form of symptoms and signs that can be observed without aid of laboratory tests, but in addition to these features observed by studying individual patients, the signature includes what can be learned or inferred about incubation periods from the time of suspected exposure to the appearance of the first clinical manifestations, the distribution of these incubation periods as summarized for a group of cases, the shape of the epidemic curve, and the size of secondary attack rates. As graduate students of epidemiology learn in their first laboratory exercises on outbreaks of foodborne illnesses, these signatures are sometimes essential for the discovery of causal mechanisms and can help public health officials decide, for instance, whether an outbreak of gastrointestinal illness might be the result of eating egg salad vs. tuna salad. When laboratory evidence on specific agents cannot be obtained, as often is the case, the study of these clinical and epidemiological details often provides the only evidence available for determining what might be causing the outbreak, and what are the indications for reducing or preventing the occurrence of future cases.

As for the application of these principles of epidemiology to disturbances of mental life and behavior, we have already mentioned PTSD and drug dependence. In addition, casual mechanisms for outbreaks of somatoform illness and epidemic hysteria can be studied using the concepts and principles of epidemiology (e.g., E. L. Goldberg, 1973). The epidemiological concepts of exposure opportunity, effective contact, inapparent cases, incubation periods, and secondary attack rates can also be used when studying normal and abnormal responses to events such as death by suicide of a classmate or co-worker, and when studying possibly neurotoxic and psychotoxic responses to industrial accidents or wartime exposures (e.g., the destruction of nerve gases or other biological weapons during the Persian Gulf War). In these instances, it is often useful to adapt the concepts and methods of epidemiology without forcing them to apply where the adaptation makes no sense. For example, in lieu of a focus on familial aggregation when estimating secondary attack rates, it is possible to substitute a focus on a classroom, a school's student body, a military platoon, or the workers in each shift of factory work.

The application of family study research methods in epidemiology has occurred in parallel with developments in other fields of inquiry. A "genealogical random test method,"

criticized by Fremming in his report on the Bornholm Island study, evolved into a variety of family-genetic research designs which work from an identified index case, with a backward look toward the history of mental disorders in deceased forebears and other older relatives, and a concurrent or even prospective assessment of mental disorders in surviving relatives, spouses, and progeny (e.g., see Merikangas, Risch, & Weissman, 1994; Weissman, 1993). Studies of especially informative pedigrees, and family study designs to detect founder effects, fit into this tradition of family-oriented epidemiological research, as do the nonconcurrent and concurrent prospective designs of twin and sibling research (Risch & Zhang, 1996).

Nonconcurrent twin studies involve recruitment and assessment of the past history of mental disorders in twin pairs, both monozygotic and dizygotic, most often reared together but sometimes separated at birth or in later years, and reared apart. The resulting study data can be analyzed within the case-control framework, but it is often more sensible to analyze the data as if it had been gathered prospectively, treating each twin pair as a homogeneous risk set observed from conception or birth until the time of follow-up. When the twins have been separated at birth or later, there are particular opportunities to tease apart the independent and combined effects of genes and the shared and nonshared aspects of their environments and experiences (e.g., see Breitner et al., 1993; DiLalla, Carey, Gottesman, & Bouchard, 1996; Kaprio, Koskenvuo, & Langinvainio, 1984; Kendler, Eaves, Walters, Neale, Heath, & Kessler, 1996; Kendler, Neale, Heath, Kessler, & Eaves, 1994; Langinvainio, Kaprio, Koskenvuo, & Lonnqvist, 1984; Lyons, 1996; Lyons et al., 1996; Lyons et al., 1995; Scherrer et al., 1996).

There is an increasing number of concurrent prospective studies of twin pairs who are recruited during fetal life or soon after birth, and followed prospectively to observe their disturbances of mental life and behavior. Closely related epidemiological research designs for siblings in general have been developed, one of the most interesting of which is based on a late twentieth-century upturn in the frequency of sequential monogamy and an associated increase in prevalence of half-sibs in the general population. That is, epidemiologists now have a greatly increased opportunity to recruit families in which there has been a change in spouse or partner during the reproductive years. As described by Olsen et al. (1997):

Like traditional square dances, modern life offers a large selection of new partners, only some of whom remain with one dance partner for more than a

limited period of time; a large number of individuals, therefore, have half-brothers or half-sisters, thus providing an opportunity to study the effect of a change in partner on reproductive outcome.

Olsen and colleagues coin the term “computerized square dance design” for epidemiological research in which computerized population registries are used to identify couples who change partners or social conditions within the reproductive time period, and to use the resulting risk sets for research on genetic and environmental contributions to adverse health outcomes. Despite possible distortions from developmentally important influences such as genomic imprinting, a variety of concurrent prospective designs with twins and half-siblings can be used to clarify genetic and environmental contributions to the occurrence of disease, and to study the interplay of these influences (Hall, 1992, Plomin, 1990). In their own application of this research design, Olsen et al. (1997) have studied congenital abnormalities, most recently focusing upon a cohort of men who fathered a child with a facial cleft defect and who had at least one additional child during the study period. As with the parallel concurrent prospective studies of twins reared together and apart, these studies of half-sibs can help tease apart genetic and environmental aspects of disease risk, with the advantage of prospective assessments from the fetal period or birth.

Notwithstanding their limitations, such as incomplete control over theoretically important distorting influences such as genomic imprinting (Hall, 1992), these “natural experiments” are among our most powerful opportunities for epidemiological research into the mechanisms underlying the risk of mental and behavioral disturbances. Harnessed with gene-mapping and biomarking tools of molecular biology and genetics, these concurrent and prospective research designs open new doors for epidemiological studies of these disorders. These research designs will help to shift the focus of attention away from the less fruitful study of the transmission of complex “inherited traits” to the more productive study of the mechanisms by which genes mapped on chromosomes are transmitted from parent to offspring, the phenotypic effects of these genes, and the influence of environment on patterns of gene expression during different developmental periods of human life (e.g., see Hulka & Margolin, 1992; Hyman & Nestler, 1996; Kosofsky, Genova, & Hyman, 1994).

In addition to these applications of family study research designs and longitudinal research designs in the study of causal mechanisms, there are some new developments in

epidemiological research on inherited traits. As we seek to discover causal mechanisms that lead toward disease, including mental and behavioral disorders, it is possible to think of large-scale epidemiological studies in which cases of mental disorders and samples of noncases are asked to provide biological samples and allow bioassays to clarify how their genes might respond to specific environmental challenges.

In a direction opposite to that of the microscopic study of genes responding to environmental challenges, epidemiology is also benefiting from an increased capacity to examine how characteristics of the household, neighborhood, and community at large can have an influence on associations observed at the level of the individual organism. For example, using an adaptation of the generalized estimating equations developed by Scott Zeger and K. Y. Liang, Katz et al. (1993) have been able to study how individual-level, household-level, and village-level characteristics work together to influence rates of diarrheal disease in developing countries. The statistical methodology has been refined to allow estimation of the strength of a possibly causal association between the hand-washing behavior of individuals and their risk of diarrheal disease, and then to tease apart how much of that association might be influenced by household-level characteristics (e.g., whether there is running water in the household), and how much might be influenced by village-level characteristics (e.g., whether the village practice is to fertilize crops with night soil, that is, human fecal matter). Together with related developments in hierarchical modeling (e.g., Bryk & Raudenbush, 1992), these innovations can soon become part of epidemiological research designs for inspecting causal mechanisms at various levels of organization: from the nation-state down to the community, family, and individual levels, and onward to the level of tissues and cells (e.g., see Sampson, Raudenbush, & Earls, 1997). As described by some observers (e.g., Susser & Susser, 1996a, 1996b), the results of these innovations will amount to a change in orientation for epidemiology, characterized (we think) by an increasing focus upon mechanisms that cause human suffering and the mechanisms for preventing human suffering, as distinct from “risk factors” that might or might not have causal significance.

#### **1.12.3.5 Rubric 5: What Can Be Done? A Question of Prevention and Intervention**

The theme of epidemiology in the service of prevention and intervention runs through this entire chapter and its stories of cholera and

pellagra. Indeed, sustained by both predisposition and training, epidemiologists aspire to make a difference in the world by discovering or devising effective ways to prevent human disease and suffering. In epidemiology, this practical goal generally is given a value even greater than those of discovering causes of disease, causal mechanisms, or effective treatment of active cases.

The degree of commitment of epidemiologists to the goal of prevention can sometimes be measured in relation to personal risk, suffering and even death. For instance, soon after the Spanish–American War, Major Jesse W. Lazear joined Walter Reed’s Yellow Fever Board in Cuba, which first sought to identify the specific cause of yellow fever by testing the idea that yellow fever was caused by the hog-cholera *Bacillus icteroides*. Unable to find evidence in support of the hypothesis about bacteria or any other specific causal agent, the Board decided that it might be better to investigate alternative mechanisms by which yellow fever was spread.

They turned to a “fomite” hypothesis, according to which yellow fever was spread by contact with clothing and bedding soiled by the excrement of yellow fever sufferers, and also to physician Carlos Juan Finlay’s theory about spread by mosquitoes. As the research team leader responsible for breeding and tending the experimental mosquitoes, Lazear came to be bitten by a mosquito, contracted yellow fever, and died. Others tested the fomite theory by sleeping on bed sheets and pillows that had been fouled by the blood and vomit of actively suffering yellow fever victims: none developed the disease. More than 20 volunteers participated in life-risking experiments on yellow fever in this manner before the Board accumulated sufficient evidence to confirm Finlay’s theory, and to guide Colonel William Crawford Gorgas toward an effective means of prevention. Namely, Gorgas saw that the mosquitoes generally remained near humans for a nutritional blood supply, and sought breeding places, to lay eggs, in nearby standing water. He organized teams to drain the standing water near homes; the density of mosquitoes near human habitations became much reduced, and the incidence of yellow fever declined as cases were prevented. (While yellow fever was being prevented by Gorgas’ methods, 30 years passed before Max Theiler identified a specific viral cause for yellow fever, from which an effective yellow fever vaccine was produced.)

The commitment of epidemiologists to prevention can also be seen in recent episodes of the history of smallpox, a communicable disease characterized by rash and fever, and with a case fatality ratio of up to 40%. Smallpox was

recognized by the ancients, but is with us no more. The virus that causes smallpox was eradicated from all but research laboratories and eliminated as a scourge of humankind in 1977. As background, partially effective means of inoculation against smallpox were introduced to Great Britain before 1750; Edward Jenner discovered a more effective vaccination method in 1796. The specific orthopoxvirus was identified by the middle of the twentieth century, while the vaccine and methods for its cryopreservation required refinement even beyond that date. However, the last smallpox case in the United States was recorded in 1949. In more tropical and developing countries, smallpox epidemics continued for an additional two decades, despite widespread mass vaccination with a refined and stable vaccine. Finally, epidemiologists working through the World Health Organization (WHO) devised a two-pronged strategy, described by epidemiologist D. A. Henderson (1980) as follows:

- (1) a 2- to 3-year program of systematic vaccination to reach at least 80 percent of the population and so diminish transmission, and (2) the development during this period of a reporting network and surveillance system that would permit prompt detection of cases so that outbreaks could be contained and transmission finally disrupted.

This global epidemiological surveillance–containment strategy was initiated in 1967. By 1976, WHO had reduced the number of smallpox-affected countries to Ethiopia and Somalia. Mobilizing for a final phase of the surveillance–containment method, WHO teams contained the last smallpox epidemic in early 1977. The last known case developed smallpox rash on October 26, 1977 in Somalia (D. A. Henderson, 1980).

Complementing its attention to smallpox and other diseases of infectious origin, more than 30 years ago the American Public Health Association recognized discoveries and accomplishments in the prevention and control of mental disorders by publishing a handbook analogous to its compendium on communicable diseases (American Public Health Association [APHA], 1962, 1994). Entitled *Mental disorders: A guide to prevention and control*, this APHA handbook continues to serve as a remarkable statement of what we already know about how to prevent mental disorders. Now an out-of-print classic, it is required reading for any serious prevention researcher in the field of alcohol, drug, or mental disorders, or epidemiologist interested in these disorders.

The introductory chapters of the APHA handbook convey the best ideas from one of the

most challenging prevention scientists of the twentieth century, Ernest M. Gruenberg, who has already been mentioned in this chapter for his key article *The failures of success* (Gruenberg, 1977). In the handbook, Gruenberg and his APHA colleagues first focus attention on the prevention of a "social breakdown syndrome," which is a breakdown of social functioning concurrent with anger, hostility and sometimes social withdrawal, often observed when mental disorders are accompanied by changes in affected individuals' social relationships after the onset of mental disorders—and often aggravated when these patients remain institutionalized for overlong periods of time.

Tackling the problem of how to prevent this type of syndrome, Gruenberg and his colleagues sought to address what is still a pressing question for primary care practitioners and specialists: namely, how can we reorganize the delivery of health and mental health services so as to reduce the suffering and secondary disabilities faced by victims of mental disorders? Seeking to answer this question, and to reduce the secondary adverse consequences of exposure to the "total institution" of a mental hospital, Gruenberg and his colleagues devised a population-oriented and patient-centered system of linkages between family, primary care provider, mental hospital, and other specialty sector resources. Focusing upon the social breakdown syndrome, his team used their system in an effort to prevent these unintended adverse consequences of hospitalization. It is important to acknowledge that Gruenberg's own attempt to evaluate his system of organizing mental health services was promising but inconclusive (Gruenberg, 1970). Nonetheless, the ideas retain pertinence, especially in the current era of clinical care for mentally disordered patients, during which concern about the mental disorders and secondary disabilities of sometimes angry, hostile, and sometimes socially withdrawn homeless persons has replaced a previous concern about the adverse consequences of excessively prolonged institutionalization (Gruenberg, 1977).

After introducing concepts and tasks for prevention in relation to the social breakdown syndrome, the APHA handbook turns to the task of cataloging all of the now recognized mental disorders and the techniques to prevent these disorders, much as the APHA communicable diseases handbook does for infectious diseases. With effectively compiled tables and narrative text, the handbook reviews the causes of specific mental and behavioral disturbances as they were known in 1962, as well as the means of prevention when the causes and causal mechanisms remained unclear. Thus, for ex-

ample, there are detailed tables of physical agents that can give rise to mental and behavioral disturbances, including the intoxication and delirium secondary to preventable exposures to chemicals such as mercury, carbon disulfide, and cocaine, as well as description of the types of effects and the antidotes, if known. There are also tables and text covering the infections that can produce mental disorders, such as St. Louis encephalitis, rubella (measles), and toxoplasmosis, which remain active as causes of mental disorders to this day. We are not aware of any more comprehensive catalog of the currently known techniques for preventing mental and behavioral disturbances.

A more recent attempt to summarize the means of prevention of mental and behavioral disturbances was published by the United States Institute of Medicine (IOM) in 1994, but this effort has much less focus upon the more firmly established means of prevention, for instance, increasing rubella immunization to reduce measles-related neuropsychiatric diseases (IOM report; Mrazek & Haggerty, 1994). Instead, the focus of the report is upon conditions for which the means of prevention are still quite uncertain (e.g., schizophrenia, major depression, conduct disorders).

The IOM report is particularly valuable for its description of a cyclical prevention research process by which emerging evidence on the suspected risk factors and causal mechanisms is translated into prevention program evaluations. To the present authors' knowledge, this is the most thorough review and summary of evidence from recent experimental field trials concerned with the prevention or reduced occurrence of mental and behavioral disturbances or with the promotion of mental health.

One useful illustration in the IOM report involves prevention of conduct problems or misbehavior, such as breaking rules about underage tobacco use and drinking. Some prevention scientists have approached these problems by means of comprehensive community-based programming efforts, and have conducted evaluations by comparing communities with and without a broad profile of political, school, and family-based interventions (e.g., Pentz et al., 1989; Perry et al., 1996). Others have oriented their prevention efforts to more focused health education initiatives within schools and classrooms, relying more heavily upon what can be accomplished within classrooms than upon a more broadly based approach (e.g., Botvin, Baker, Filazzola, & Botvin, 1990; Ellickson & Bell, 1990).

A different point of departure can be seen in the work of research teams formed by J. David Hawkins and by Sheppard G. Kellam. Orienting

themselves with regard to a long tradition of clinical observations about how drug users seen in clinical practice seemed to have an excess of childhood conduct problems, Hawkins and Kellam also noted Lee Robins' observations about the continuity of childhood conduct problems in relation to adult antisocial behavior. This was exemplified in a classic nonconcurrent prospective study over a period of more than 25 years, during which childhood rule-breaking and conduct problems were found to account for adult drug problems (Robins, 1966). Kellam and his Woodlawn, Chicago research team (Kellam, Brown, Rubin, & Ensminger, 1983) extended this line of research in their own prospective study of youths, who were first recruited and measured at school entry in 1966 and then followed up at age 16–17 in the mid-1970s. The Woodlawn research group found that first-graders who had been rated by teachers as rule breakers were about three times more likely to have become frequent tobacco, alcohol, and marijuana users by their teen years. The longitudinal association was particularly strong among boys, less so among girls.

Both Hawkins and Kellam were able to perceive that inferences about the causal significance of these observed associations depended upon completion of comparative trials. That is, the available evidence was not sufficient to sort out competing explanations for the observed data. On the one hand, it might be said that prevention of early misbehavior could reduce the risk of later drug involvement. On the other hand, it could be said that the apparent predictive linkage from early misbehavior to later drug involvement was no more than a spurious artifact of some unmeasured or unknown causal determinant of both early conduct problems and later drug use. The evidence to unravel this complex research issue would remain incomplete without preventive trials to study the impact of early childhood interventions. That is, within the framework of a preventive trial, there could be an attempt to alter early childhood misbehavior. If the early intervention proved to be effective, and if there is a causal link between early misbehavior and later drug involvement, then the children exposed to the intervention program should be less likely to initiate drug use.

The trials to test these causal and preventive hypotheses were initiated in the 1980s, with recruitment of children as they entered early elementary school. Early results are promising, both in the Seattle trial organized by Hawkins' research group and in the Baltimore trial organized by Kellam's research group (Hawkins, Von Cleve, & Catalano, 1991; Kellam & Anthony, in press; Kellam, Rebok, Ialongo, &

Mayer, 1994; Kellam et al., 1991; O'Donnell, Hawkins, Catalano, Abbott, & Day, 1995). Nonetheless, even if these trials end with inconclusive results, they clarify a new direction for prevention research in relation to mental and behavioral disturbances. Namely, analogous to the communicable disease epidemiologists who sought either to identify specific causes or to identify causal mechanisms, we have psychologically and psychiatrically oriented epidemiologists who seek to identify specific causes or causal mechanisms for mental and behavioral disturbances. As these investigations yield evidence of suspected associations of causal significance, the observed findings can be translated into research questions for prevention researchers, who must take the next step of using experimental evaluation methods to organize field trials that can probe more deeply into the suspected causal significance of the observed associations (Mrazek & Haggerty, 1994).

Several additional preventive studies conducted by teams led by or including epidemiologists deserve mention in this chapter. One of the most interesting of these involved an analysis of data that was gathered over years for the National Institute on Drug Abuse's *Monitoring the future* survey of high school seniors. Literally thousands of these high school seniors have been surveyed each year since 1976 in order to produce a trace of epidemiological trends in the prevalence of drug use. O'Malley and Wagenaar (1991) took advantage of these survey data to evaluate the impact of state-level restrictions on underage alcohol consumption and the occurrence of fatal car crashes. This evaluation of state-level policy making was possible because over a relatively short span of years the high school senior survey covered multiple states with different drinking regulations, some allowing legal alcohol consumption at age 18, and some prohibiting it until age 21. This is, of course, an important public health and public policy question, and one that would be difficult to test using an experimental design at the state level or at the local level. Notwithstanding some difficulties in study implementation and interpretation (e.g., relating to underage drinkers' capacities to cross state lines in order to become legal drinkers), the authors' analyses provided confirmatory evidence of modest but tangible beneficial effects of state laws to postpone legal drinking until age 21. Exploratory subanalyses indicated that the beneficial effects might be conveyed by factors such as reducing the amount of time that underage drinkers spend in bars and taverns.

Another pair of epidemiological studies investigated how handgun regulations might be influencing the rates of homicides and

suicides in North America, a topic of considerable public health and political interest. These investigations were made possible by making a direct comparison between the homicide and suicide rates of Seattle, Washington, and Vancouver, British Columbia. These two metropolitan areas of the Pacific Northwest are comparable in many respects, but they have substantially different handgun regulations. In Seattle, the handgun regulations are more lax, whereas in Vancouver, they are quite restrictive. Studying homicide rates, and making comparisons via analyses that were able to adjust for some of the apparent differences between Seattle and Vancouver, the research team found evidence of substantially lower homicide rates in Vancouver, and drew the inference that these lower rates could be attributed to the greater restrictions on handguns. Though an important epidemiological investigation, the study lacked a capacity to hold constant some of the potentially distorting influences on homicide rates, such as variation in the street drug trade in each city, Seattle's more prominent contrasts between wealthy and poor neighborhoods in close proximity, and the existence of a more comprehensive social "safety net" in Vancouver, which might reduce feelings of frustration and despair, with a resulting impact on aggressive behavior (Sloan et al., 1988).

The investigation on the possible preventive impact of handgun regulations in relation to suicide mortality rates, in general, was negative. Using the same methodology used in the homicide study, the research team compared Seattle and Vancouver, and found insufficient evidence to support the claim that more restrictive handgun laws can help reduce suicide death rates. Nonetheless, in exploratory subgroup analyses, the research team found some evidence that certain subgroups of the population might have lower suicide mortality rates due to restrictive handgun legislation. Namely, there was a tendency for the suicide rates to be lower for Native American young people living in Vancouver when compared to counterparts in Seattle (Sloan et al., 1990). As noted with respect to the study of homicides, interpretation of these comparative data is complicated by some between-city imbalances in possibly distorting variables, but this takes nothing away from an important epidemiological study of a significant issue for prevention.

One of the recent innovations that can help in the process of translating new scientific information on causal mechanisms into prevention efforts is the Cochrane Collaboration, which is an international network of individuals and groups who prepare, maintain, and disseminate systematic reviews of the effects of health care

(Bero & Rennie, 1992; Sheldon & Chalmers, 1994) Clive Adams in Great Britain serves as first editor of a Cochrane Collaborative Review Group concerned with mental health, with a focus on therapeutic interventions. The development of a collaborative review group concerned with prevention of alcohol, drug, and mental disorders was the subject of planning meetings started by C. Hendricks Brown, Pat Mrazek, and James C. Anthony in 1996. Publications based on the work on this review group should begin to appear in 1998–2000.

There are also some distinctively epidemiological research designs that have been brought into service for the evaluation of prevention programs, but which have not yet been used in the domains of psychology and psychiatry. For example, Comstock (1994) has described how the epidemiologic case-control design has been harnessed to evaluate the effectiveness of new vaccines.

There are some clear advantages to using case-control methods to evaluate prevention efforts, as well as some unusual challenges that can block proper inference. Nonetheless, the case-control study method may be especially helpful in understanding variation in response to preventive or therapeutic efforts. Unless a program evaluation has been pre-planned for measurement of all pertinent response-affecting variables, there might prove to be no alternative to case-control methods when we seek to account for why some individuals responded to the intervention while others did not.

#### 1.12.4 CONCLUSION

Epidemiological research continues to make exciting discoveries about the prevention of human disease and suffering, not only in relation to the long-time scourges for epidemiologists such as cholera, for which a new and more effective vaccine has just been developed, but also in relation to mental and behavioral disturbances (Mrazek & Haggerty, 1994). Nonetheless, there still remain important areas for new collaborations between epidemiologists, clinical psychologists, and psychiatrists. Future progress depends upon a narrowing of the gap between epidemiological psychology and psychiatry on one side and psychiatric or psychological epidemiology on the other.

As described in earlier sections of this chapter, clinical psychologists and psychiatrists have already harnessed tools of epidemiology when studying causal hypotheses and program evaluation issues. The works of Dohrenwend et al. (1992) and Kellam already have been cited (Kellam et al., 1983), but there are many other clinicians who have worked in this arena. The

cumulative epidemiological studies undertaken by Michael Rutter's research group in Great Britain serve as an especially important example of how to build a research program to answer questions of central importance in public mental health (e.g., see Rutter, 1989; Rutter, Tizard, Yule, Graham, & Whitmore 1976).

In the United States, Lloyd Johnston's and Jerrold Bachman's *Monitoring the future* surveys, published each year since the mid-1970s, serve as an annual reminder of the power of epidemiological methods in answering questions such as "How many high school seniors are taking drugs?" The field studies of Bentler, Newcomb, Brook, Muthen, and others have drawn heavily upon epidemiological methods and strategies while remaining psychological in focus (e.g., see Bentler & Stein, 1992; Brook & Newcomb, 1995; Muthen, 1995, 1996; Newcomb & Bentler, 1988). These investigations represent important progress at the interface of epidemiology, psychology, and psychometrics. Nonetheless, there still are some areas of differences between epidemiology and the clinical sciences of psychology and psychiatry that can be addressed in greater detail.

Epidemiology, originating with the study of a discrete categorical event such as death or frank disease, tends to emphasize the study of manifest indicators of human suffering. In addition, epidemiology's strong links to clinical medicine reinforce this tradition. In terms of measurement, this often means a focus on discrete categories and transitions between categories. With regard to the study of causal hypotheses, this emphasis often implies a reluctance to depend heavily upon statistical modeling when basic associations cannot be perceived without the assumptions of these models. From psychology, we often have the opposite tendencies, with a much heavier focus upon latent variables, and with considerable reliance upon structural equation modeling, which is sometimes so complicated that the basic relationships cannot be perceived except from within the framework of these models. As epidemiologists collaborate with clinical psychologists and psychiatrists, there is a need for greater dialog and truly interdisciplinary interaction on important matters such as these. Through dialog and interaction, we can hope for continuing progress under each of the rubrics of epidemiology as they relate to the disciplines of clinical psychology and psychiatry.

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# 1.13

## Learning Theory

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### 1.13.1 INTRODUCTION

Ever since the first controlled experiments were systematically carried out on learning and

conditioning in the early part of the twentieth century, the study and understanding of the processes of learning have contributed significantly to all aspects of clinical psychology. The

classical conditioning paradigm established by Pavlov very soon led to the investigation of psychopathology in terms of learning processes, with Pavlov's own studies of experimental neuroses in animals leading to a conceptualization of many aspects of psychopathology not in terms of dysfunction but in terms of the animal's experience of controlled replicable environmental contingencies. This process began the demystification of psychopathology, and enabled clinicians to remove much of the stigma that had been associated with psychopathology—psychological disorders were not so much the result of dysfunctional psychological systems, but the result of learning experiences generated by perfectly normal learning mechanisms.

Pavlov's work on classical conditioning led rather naturally to the use of that conditioning paradigm in the explanation of a variety of acquired psychological disorders. The most famous of these was the use of the classical conditioning paradigm in the explanation of specific phobias by J. B. Watson and his co-worker Rayner in the early 1920s. Their study of the classical conditioning of a specific phobia in eight-month old "Little Albert" (Watson & Rayner, 1920) has entered psychological folklore as the prototypical conditioning study of acquired fears. It is still quoted in nearly all clinical psychology textbooks that deal with anxiety, even though most texts criticize the ability of this study to explain many of the important features of acquired fears (see Sections 1.13.3.1.1 and 1.13.4.1.1). Even though it has had a significant impact on contemporary models of acquired fears (Davey, 1992a), the original Watson and Rayner study is poorly reported and frequently misdescribed in secondary texts (Harris, 1979). Nevertheless, this should not underestimate the importance of the conditioning paradigm in contributing to the understanding of a range of acquired fears, including specific phobias, panic disorder and, more recently post-traumatic stress disorder (PTSD).

While the studies of classical conditioning processes were providing useful insights into the etiology of anxiety-based disorders, it became clear that if conditioning theory could provide an explanation of these problems, then they should also provide principles relevant to the development of successful therapeutic procedures. This was exactly the thinking of Joseph Wolpe and Hans Eysenck in the 1950s. Disillusionment with the failure of psychodynamic therapies to fit comfortably into emerging objective and scientific conceptualizations of psychological processes led to the development of behavior therapies. These therapies

were based on objectively derived principles of conditioning and were consistent with the kinds of processes that conditioning theories assumed were responsible for the generation of anxiety and emotion-based disorders. Put in its simplest form, if anxiety-based disorders were manifestations of inappropriately learned conditioned responses, then those conditioned responses could be eliminated by adopting procedures which would extinguish those responses. This, as we shall see later, was the rationale for the development of a variety of highly successful behavior therapies, including flooding, counter-conditioning, and systematic desensitization (see Section 1.13.4.1.5).

Apart from the use of classical conditioning procedures in both the explanation and amelioration of psychological disorders, the development of operant conditioning paradigms offered completely new and different applications to clinical psychology. While there were clearly some psychological problems whose etiology could be explained by reference to the nature of the consequences of behavior (e.g., through conducting a functional analysis of the contingencies which were maintaining the problem), what operant conditioning also offered was a valuable tool for changing behavior. This has generated a wide range of behavior therapy and behavior modification programs that not only help to maintain psychologically healthy and desirable behaviors (e.g., *The token economy*; Ayllon & Azrin, 1968), but also enable the therapist to develop new behavior repertoires where none existed before (e.g., in establishing basic self-help and communication behaviors in individuals with learning disabilities). What is so valuable about the paradigm offered by operant conditioning is that it enables the therapist to examine the functional relationships between problem behavior and environmental contingencies, and in many cases allows the therapist to provide the client with a treatment program that the client can establish and execute on his or her own. Such behavioral self-control programs have become a valuable addition to many therapeutic programs (e.g., diet control, treatment of addictions, etc., see Section 1.13.4.2.4).

This chapter will provide a structured overview of the contribution of learning theory to clinical psychology and will begin with a description and explanation of the basic processes of conditioning. This will be followed by an assessment of the seminal historical contributions from learning theorists. The chapter will conclude with an overview of the contribution of contemporary conditioning theory to the etiology and treatment of psychological disorders.

### 1.13.2 BASIC DEFINITIONS, CONCEPTS, AND PROCESSES OF CONDITIONING

#### 1.13.2.1 Classical Conditioning

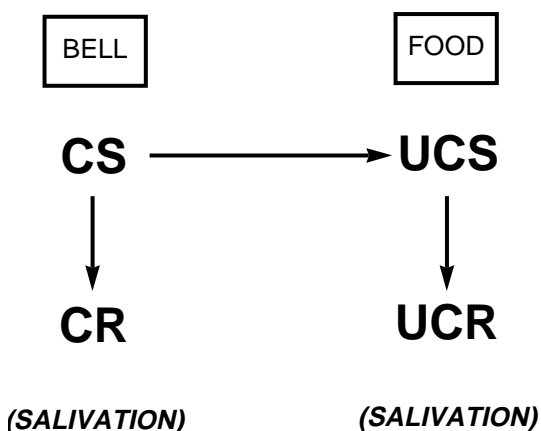
##### 1.13.2.1.1 Pavlov's prototypical conditioning procedure

In classical conditioning a conditioned stimulus (CS) is paired in close temporal contiguity with an unconditioned stimulus (UCS). After a number of CS-UCS pairings, the CS comes to elicit a conditioned response (CR) relevant in some way to the UCS. This process is displayed schematically in Figure 1. The UCS is usually some biologically important event which itself evokes an unlearned, reflexive reaction known as the unconditioned response (UCR). In Pavlov's prototypical conditioning experiment, a hungry dog was presented with pairings of a bell or metronome (the CS) and a bowl of food (the UCS), after a number of pairings of CS and UCS, the dog began to salivate to the CS (the CR).

UCSs used to generate conditioned responding are typically either appetitive or aversive. Appetitive UCSs are such things as food or water to a hungry or thirsty animal. Aversive UCSs are stimuli which the animal finds either painful or distressing (such as a brief mild electric shock or a loud noise).

##### 1.13.2.1.2 The nature of the conditioned response

In most traditional classical conditioning studies (such as Pavlov's traditional salivary



**Figure 1** Schematic representation of the typical classical conditioning procedure. Pairing a conditioned stimulus, CS (e.g., a bell), with an unconditioned stimulus, UCS (e.g., food), eventually evokes the conditioned response, CR, to the CS (in this case salivation).

conditioning procedure), the CR is often what looks like a faithful facsimile of the UCR. In Pavlov's study the UCR is salivation and the learned CR to the CS is also salivation. However, the learned CR can often be rather different from the type of response elicited by the UCS. For example, in rats an aversive UCS such as electric shock will elicit a UCR which consists of heart-rate increases, jumping, and squealing, whereas the CS which is paired with electric shock often results in the acquisition of "emotional" anticipatory responses in the presence of the CS—such responses include heart-rate decreases, freezing defecating, etc. (Black, 1971; Borgealt, Donahoe, & Weinstein, 1972). Some traditional explanations of classical conditioning have argued that what occurs during classical conditioning is a form of "reflex transfer" in which the CS becomes a "substitute" for the UCS and thus elicits the same responses as the UCS (cf. Davey, 1989b). This is clearly not the whole story. The CS does elicit a range of responses which are relevant to the UCS, but they are not necessarily the responses that are elicited by the UCS. This has important implications for the acquisition of specific fears in humans. The dental phobic does not wince and cry out in pain while sitting in the dentist's waiting room; what is elicited by the dentist's waiting room is an anticipatory fear rather than responses that would normally be reactions to the dentist's drill. What the reader should be aware of is that classical conditioning is not necessarily the nonconscious, reflexive form of learning that is portrayed in traditional textbooks. It is a highly cognitive form of associative learning which permits a wide range of conscious and nonconscious reactions to be emitted to the CS as a result of learning about the relationship between the CS and UCS (Davey, 1992a; see Sections 1.13.2.1.4 (vi) and 1.13.4.1.2).

##### 1.13.2.1.3 Control procedures

The important experimental manipulation in classical conditioning is the paired relationship between the CS and the UCS. Therefore, in order to conclude that classical conditioning has occurred, it is important to be able to determine that any responses which are elicited by the CS are the result of the CS being paired with the UCS. Clearly, there are other learning processes, such as habituation and sensitization, that could produce spurious behavior changes in a classical conditioning procedure. For example, a study by Grether (1938) first made monkeys fearful by presenting a number of powder flashes. Following this, the monkeys were given a series of presentations of a bell, all

of which elicited reliable fright reactions which they had not done prior to the powder flashes. In this example, the powder flash was a UCS and the bell a CS, yet a fearful response to the CS had been established without any pairing between CS and UCS.

Because the important manipulation in classical conditioning is the arrangement of the CS and UCS in a consistent temporal or predictive relationship, any classical conditioning experiment should possess controls which retain all the features of the conditioning procedure *except* the predictive relationship between CS and UCS. The most appropriate control procedure is known as the truly random control (Rescorla, 1967). In this control procedure, subjects are given a similar number of CS and UCS presentations to experimental subjects, but CS and UCS are programmed independently of each other. Thus, control subjects receive exactly the same number of CS and UCS presentations, but not the predictive relationship between them. This distinction is important particularly in relation to the acquisition of fearful or anxious responding, because such responding can be acquired through nonassociative processes such as sensitization as demonstrated in the Grether experiment (above). Models of psychopathology based on classical conditioning emphasize the importance of the learning of an *association* between the CS and UCS (e.g., Davey, 1992a, 1997), and it is this association that generates many of the features of psychopathological responding (see Section 1.13.4.1).

#### 1.13.2.1.4 *Basic phenomena of classical conditioning*

##### (i) *Predictive value of the CS*

Traditionally it has been assumed that the important feature of classical conditioning which generated the CR was the simple pairing of CS and UCS. That is, it was assumed that the number of pairings of the CS with a temporally contiguous UCS was a prime determinant of the strength of the CR (e.g., Kimble, 1961). Nevertheless, while temporal contiguity is an important contributor to learning, it is not necessarily the number of CS–UCS pairings that is important, but the predictive significance of the CS as measured by the correlation between the CS and UCS. When we come to discuss the features of classical conditioning in humans, it will become clear that it is not the number of pairings between CS and UCS that determines either the speed of acquisition of the CR or the strength of the CR. Reliable conditioning can occur after a single pairing of CS and UCS as

long as the subject was immediately aware of the predictive significance of the CS (cf. Davey, 1992a; Dawson & Schell, 1987;). Indeed, if human subjects are pre-experimentally informed of the predictive relationship between the CS and the UCS, a full-blown CR can be exhibited on the first presentation of the CS before *any* CS–UCS pairings have been experienced (Dawson & Grings, 1968; Deane, 1969; Wilson, 1968).

##### (ii) *Extinction*

If, after conditioning has been acquired, a CS is subsequently presented alone, the strength of the CR to that CS subsequently declines over trials until the CS no longer elicits a CR. This is known as extinction. This process has been used extensively in the application of conditioning models to psychopathology, largely in the development of behavior treatments for emotional disorders (e.g., flooding, systematic desensitization). These therapies attempt to ensure that the emotion-eliciting stimulus (the CS) is not presented in conjunction with an aversive UCS (such as fear), and thus the learned relationship between the CS and trauma is extinguished. There is some doubt about whether extinction represents the unlearning of previously acquired CS–UCS associations, because presenting a novel stimulus or an additional single CS–UCS pairing can often fully reinstate a previously extinguished CR (Pavlov, 1927). This suggests that extinction may represent either the inhibition of a learned CR or the learning of new CS–UCS associations which override the original ones (cf. Davey, 1989c). In either case, extinguished CRs do not have to be totally relearned in order to be reinstated and their spontaneous recovery may explain at least some instances of relapse following treatment of emotional disorders by therapies based on extinction principles.

##### (iii) *Latent inhibition*

When a CS is presented alone on a number of occasions prior to being paired with a UCS, it subsequently takes significantly longer to generate a CR to that CS than when no pre-exposure has taken place. This is known as latent inhibition and has been widely demonstrated in a variety of classical conditioning preparations in both animal and human subjects (Lubow, 1973; Siddle & Remington, 1987). This feature of classical conditioning is potentially important in a number of conditioning theories of psychopathology because it predicts that if a previously neutral CS (e.g., a car) is paired with an aversive UCS (e.g., a traumatic car accident), then whether the individual acquires a



conditioned fear response to cars will be inversely proportional to the amount of prior nonaversive experience they have had with cars. This principle, for example, can potentially help to explain why some people acquire a phobia following pairing of a stimulus or event with a traumatic UCS and some do not (cf. Davey, 1989a).

(iv) *Blocking*

If a UCS is already reliably predicted by a CS (call it A), then subsequently signaling that UCS with a compound CS consisting of the original CS, A, plus a new component (call it B), results in little or no learning about the new component B (Kamin 1968, 1969). This is known as blocking, and it is a robust phenomenon in animal conditioning (cf. Mackintosh, 1983). Blocking is normally considered to be an associative phenomenon in that organisms do not appear to learn about the new component B because the UCS is already reliably signaled by component A (Dickinson & Mackintosh, 1979; Rescorla & Wagner, 1975). Although this is an important phenomenon for theories of associative learning in animals (Pearce & Hall, 1980; Rescorla & Wagner, 1972), it has rarely been applied in the context of conditioning models of psychopathology. This may be because it has been difficult to experimentally isolate and demonstrate examples of blocking in human subjects (Davey, 1992a; Davey & Singh, 1988).

(v) *Higher-order conditioning*

Once a CS has been associated with a UCS and is capable of eliciting a reliable CR, that CS can then be used to reinforce other potential CSs. For instance, second-order conditioning can be demonstrated using the following procedure: a CS1 (e.g., a light) is paired with a UCS (e.g., food); then CS2 (e.g., a tone) is paired with CS1 (the light). This will usually result in a CR relevant to the original UCS (food) being evoked by CS2, even though CS2 has never been directly paired with food (e.g., Rescorla, 1980; Rizley & Rescorla, 1972). This phenomenon also has potential importance for conditioning models of psychopathology because it implies that emotional reactions can be acquired through higher-order conditioning in which the potentially phobic CS has never been paired directly with a traumatic UCS.

(vi) *What is learnt during classical conditioning?*

Although we know that pairing a CS with a UCS will generate a CR to the CS, we cannot easily explain how this happens. Most probably

the organism has learnt to associate certain contiguous or predictive events together and it is these learned associations which mediate the CR. However, the theoretical problem is that we cannot directly observe what associations have been formed but have to infer them from the experimental manipulations that are carried out. We can ask very generally what kinds of things the organism might have learnt during classical conditioning which led it to emit a CR during the CS. For example, Pavlov's dog may have learned that the bell (CS) predicts food; that is, after some pairings of the bell (CS) with food (UCS), the bell comes to elicit a memory or internal representation of the food that is to follow, and it is this evoked representation that stimulates salivation. Alternatively, the dog may be much less "cognitive" in its learning. Instead of becoming associated with food, the bell may have become associated with the act of salivation which is also associated with food. Thus, a direct link may have been formed between centers representing the bell and the reflex arc which controls salivation. In order to differentiate between these two possibilities, animal learning theorists have developed inferential techniques which enable them to ascertain exactly what kinds of learned associations are acquired during classical conditioning.

One simple technique is known as postconditioning stimulus revaluation (Dickinson, 1980; Rescorla, 1980). This procedure involves attempting to revalue the UCS for the subject, and normally consists of three stages: (a) subjects are given pairings of CS and UCS until a CR is established (b) subjects are then given off-the-baseline training with the UCS alone in which the UCS is revalued (e.g., a food UCS may be paired with gastric poisoning to make it less attractive and less palatable); and (c) the animal is then given test presentations of the CS. The logic here is that, if the CR is mediated by a CS–UCS association, then revaluation of the UCS will also affect the CR because the CR is mediated via the UCS representation. If, however, the CR is mediated by a more reflexive association between the CS and the UCR reflex, then the CR will not be affected by changes in the evaluation of the UCS. Most studies which have used this technique have demonstrated that first-order classical conditioning (both appetitive and aversive) appears to be mediated by CS–UCS associations (cf. Davey, 1989c; Dickinson, 1980; Rescorla, 1980) and not by reflexive associations formed between the CS and the response to the UCS. These findings have a number of important implications for our conceptions of the classical conditioning process, and for the application of classical conditioning to

psychopathological phenomena. First, it implies that classical conditioning is not a simple, mechanistic or reflexive form of learning. Instead, it involves (a) the learning of associations between contiguous and predictive events, and (b) mediation of the CR by representations of the UCS and the knowledge of the UCS contained in that representation. Thus, classical conditioning is a complex cognitive process which involves the formation of representations of events as well as the establishment of associations. Second, since CRs generally appear to be mediated by information about the UCS contained in a UCS representation, then the nature or strength of the CR can be affected by changes in the information contained in the UCS representation. This has extremely important implications for conditioning models of emotional disorders, and has been a central feature of contemporary conditioning models of phobias (Davey, 1992a, 1997; see Sections 1.13.4.1.2 and 1.13.4.1.3).

#### ***1.13.2.1.5 Special features of human classical conditioning***

##### *(i) Similarities between animal and human conditioning*

Davey (1992a) has argued that while there are some important differences in classical conditioning between human and nonhuman animals (see below), the basic conditioning phenomena tend to be common to both. For example, human subjects exhibit blocking (Dickinson, Shanks, & Evenden, 1984) and latent inhibition (Siddle & Remington, 1987; Siddle, Remington, & Churchill, 1985). Higher-order conditioning can be demonstrated in humans (Davey & Arulampalam, 1981, 1982; Davey & McKenna, 1983), and CR strength in humans can be shown to be a function of the predictive significance of the CS as measured by the correlation between the CS and UCS (Alloy & Tabachnik, 1984; Prokasy & Kumpfer, 1969; Prokasy & Williams, 1979). In terms of the associations that are learned during classical conditioning, human subjects also appear to learn to associate CS and UCS so that the CR is mediated by the subject's internal representation of the UCS (Davey & McKenna, 1983; White & Davey, 1989).

##### *(ii) Conscious awareness of contingencies*

A large number of studies have suggested that human subjects only exhibit a differential CR when they are able to verbalize the CS–UCS contingency (see Dawson & Schell, 1987 for a review). Only subjects who can verbalize the

correct CS–UCS relationship in postexperimental interviews exhibit differential CRs (Baer & Fuhrer, 1968, 1970); when awareness of contingencies is measured on a trial-by-trial basis, differential conditioning appears only after the appearance of contingency awareness (Dawson & Biferno, 1973; Dawson, Schell, & Tweddle-Banis, 1986), and subjects regularly fail to exhibit differential conditioning in studies which deliberately attempt to “mask” the relationship between CS and UCS by employing distracter tasks (Dawson, 1973; Dawson, Catania, Schell, & Grings, 1979). These findings have led some theorists to suggest that the classical conditioning of autonomic responses (such as those concerned with emotional responding) is only possible when individuals are consciously aware of the CS–UCS relationship, and that this can only occur as a result of strategic processing of the contingencies (e.g., Dawson & Schell, 1987; Dawson, Catania, Schell, & Grings, 1979). However, there are other forms of human classical conditioning that appear to occur in the absence of awareness of the CS–UCS contingency. There are (a) the evaluative conditioning procedure, where a subjectively neutral stimulus (CS) is paired with a liked or disliked stimulus (UCS) which results in the CS acquiring positive or negative valence depending on the valency of the UCS (Baeyens, Eelen, Van den Bergh, & Crombez, 1989, 1990), and (b) human aversive conditioning studies in which the CS is a picture of a fear-relevant stimulus (e.g., a snake) which is presented subliminally (Esteves, Dimberg, Parra, & Öhman, 1994; Öhman, 1993). Nevertheless, there is still much theoretical argument about whether these latter two paradigms represent conditioning without awareness (Davey, 1994, 1995), and a definitive answer to the question of whether conscious processing of contingencies is necessary for classical conditioning in humans is still awaited.

##### *(iii) Re-evaluation of the UCS*

Studies using the postconditioning stimulus reevaluation procedure (see Section 1.13.2.1.4 (vi)) have shown that by independently manipulating the animal's evaluation of the UCS, the strength or nature of the CR can also be changed. This implies that there is a nonassociative process that can affect the strength of the CR which is independent of the strength of the association between the CS and UCS. For example, in aversive conditioning, if the animal's evaluation of the UCS is changed so that it now perceives the UCS as being less aversive than it originally was, then the next presentation of the CS will evoke a significantly weaker CR.

Davey (1989c, 1992a) has argued that the process of UCS reevaluation is significantly more important in modulating the strength of the CR in humans than it is in nonhuman animals, and that because humans are social animals that have developed sophisticated communication skills, there will be many ways in which experiences and communicated information can alter an individual's perception and evaluation of a UCS. Thus, because learnt CRs are mediated via a representation of the UCS, then arguably the most important contributor to the strength of a CR in human classical conditioning is not necessarily the strength of the association between CS and UCS, but how the individual perceives and evaluates the UCS. This is critically important when it comes to considering conditioning models of psychopathology, and will be discussed more fully in Section 1.13.4.1.3.

However, it is worth listing the kinds of processes that can contribute to changes in evaluation of the UCS in human subjects.

(a) *Experiences with the UCS alone.* Some postconditioning experiences with the UCS in the absence of the CS can lead the individual to revalue the UCS. For example, an individual may reassess a UCS more favorably if they experience a number of UCS-alone trials which allow their fear to habituate (Davey & McKenna, 1983). Similarly, the perceived aversiveness of the UCS may be inflated by experiences with a similar UCS of greater intensity (e.g., White & Davey, 1989). Both of these processes will have a subsequent effect on the strength of the CR when the CS is next presented.

(b) *Socially/verbally transmitted information about the UCS.* In a laboratory conditioning experiment, subjects can simply be told that on future presentations the UCS will be more or less intense than before. If the subject believes this information, then the evaluation of the UCS is changed, and this affects the strength of the CR to subsequent CS presentations (Davey & McKenna, 1983).

(c) *Interpretation of interoceptive cues.* Many anxious people attend to their own bodily sensations and use these stimuli as a means of assessing the aversive nature of potentially threatening consequences (cf. Davey, 1988; Parkinson, 1985). As a result, the individual's reaction to either the CS or UCS can act as an important source of information for evaluating the UCS. For example, if they believe they are emitting a strong fear response to a CS (even though this may not be the case physiologically), then they will often attribute this to being fearful of the UCS, and this inflation of the aversive evaluation of the UCS in turn does produce an actual increase in the strength of the

CR (Davey & Matchett, 1996; Russell & Davey, 1991).

(d) *Cognitive rehearsal of the UCS.* It is clear that individuals who suffer anxiety disorders have a tendency to focus on and rehearse the possible aversive outcomes of phobic encounters (Marks, 1987), and this ruminative tendency may act to inflate their aversive evaluation of the potential aversive outcome (UCS). A laboratory conditioning study by Davey and Matchett (1994) showed this to be the case, but only in subjects who were already anxious. When asked to experimentally rehearse the UCS after conditioning had been completed, subsequent presentations of the CS elicited a greater magnitude fear CR than had occurred prior to rehearsal, but this effect was found only in subjects with high levels of trait anxiety or who had undergone a procedure which induced an acute anxious state. Davey and Hatchett argued that this increase in the strength of the CR following rehearsal of the UCS in anxious subjects was the result of rehearsal inflating the aversive evaluation of the UCS in these subjects.

(e) *Coping strategies which neutralize the UCS.* Many individuals who have aversive-conditioning experiences during their daily lives (e.g., traumatic car accidents, being bitten by a dog, painful or traumatic dental experiences) subsequently fail to develop a learned anxiety or fear reaction to these stimuli. One reason for this failure may be the ability of some individuals to devalue or neutralize the trauma immediately following the experience, and they may do this by adopting appraisal strategies which allow them to effectively devalue the stressful meaning of the trauma. In a recent study Davey, McDonald et al. (1997) identified seven factorially independent reappraisal strategies all of which contribute to trauma or UCS devaluation. These are downward comparison (e.g., "other people are worse off than me") (Wills, 1981), positive reappraisal (e.g., "in every problem there is something good") (Davey, 1993), cognitive disengagement (e.g., "the problems involved in this situation simply aren't important enough to get upset about"), optimism (e.g., "everything will work itself out in the end") (Scheier & Carver, 1992), faith in social support (e.g., "I have others who can help me through this"), denial (e.g., "I refuse to believe this is happening") (Breznitz, 1983), and life perspective (e.g., "I can put up with these problems as long as everything else in my life is okay"). Davey et al. (1997) found that use of these strategies (except for denial) was positively correlated with measures of psychological health and inversely correlated with a variety of measures of psychopathology. In addition,

Davey, Burgess, and Rashes (1995) found that both simple phobics and panic disorder patients differed from normal controls by reporting reduced use of appraisal strategies which devalue the stressful meaning of a trauma. Such coping strategies may therefore play an important role in insulating individuals from the acquisition of phobic responding following traumatic conditioning experiences.

#### (iv) UCS expectancy biases

It was mentioned in Section 1.13.2.1.4 (i) that human subjects will emit a fear CR to the first presentation of a CS if they have previously been informed that the CS is to be followed by an aversive UCS. Thus, a UCS “expectancy” can be generated simply through verbal transmission of the contingencies. Life consists of regular discussion and transmission of contingencies relevant to daily living, so it is not unreasonable to suppose that individuals will develop beliefs and expectancies about what will happen when they encounter a conditioning episode. As a result, when they enter a conditioning episode, individuals are not *tabula rasa* but will hold beliefs and expectancies about what is likely to happen.

An example of the associative biases that influence the formation of CS–UCS associations can be found in covariation assessment studies. Studies of covariation have pointed out that assessing whether two stimuli covary appears to be influenced by both situational information (i.e., current information about the contingency) and prior expectations or beliefs about the covariation (e.g., Alloy & Tabachnik, 1984; Crocker, 1981). There are a variety of circumstances in which the combination of situational information (e.g., the experienced CS–UCS contingency) and prior expectancies (e.g., the belief that a CS is to be followed by a particular kind of UCS) give rise to what is called a covariation bias which generates a distorted perception of the covariation—usually in the direction of the prior expectation (Alloy & Tabachnik, 1984).

Expectancy or covariation biases can be found quite frequently in human classical conditioning, especially in circumstances where subjects will have developed a belief that a particular type of CS may be followed by a particular type of UCS (even though that is not necessarily the case). For example, when pictures of fear-relevant stimuli (i.e., stimuli that are potentially dangerous such as a snake or a gun) are used as CSs in aversive conditioning studies, subjects acquire the CR more rapidly than if the CS were a picture of a fear-irrelevant stimulus (e.g., a flower). The CR in such

circumstances also shows a significant resistance to extinction (Hugdahl & Johnsen, 1989; Öhman, 1979). A series of experiments by Davey (1992b) showed that this tendency for fear-relevant stimuli to show stronger conditioning effects than fear-irrelevant stimuli was probably the result of a pre-experimental expectancy bias. In this study, Davey used a “threat” conditioning procedure (where subjects are warned that they might receive aversive UCSs following some CSs, but in fact receive none) and found that subjects began the experiment with a significantly higher expectancy of aversive UCSs following fear-relevant stimuli than fear-irrelevant stimuli. These studies indicate that individuals enter a conditioning episode believing that fear-relevant stimuli are more likely to have aversive consequences than fear-irrelevant stimuli. This speeds up the learning of associations between fear-relevant CSs and aversive UCSs, and also makes them more resistant to extinction. There has been considerable debate about how such expectancy biases are derived, with some arguing that they have been biologically determined as a result of evolutionary pressures (e.g., Cook, & Mineka 1990; Öhman, Dimberg, & Öst, 1985), and others that they are the result of culturally transmitted information about the stimulus (e.g., Davey, 1995; Merkelbach & de Jong, 1997). This discussion has particular relevance to the application of conditioning models to phobic responding, and we will return to it in Section 1.13.4.1.3.

### 1.13.2.2 Operant Conditioning

#### 1.13.2.2.1 Basic concepts in operant conditioning

Whereas classical conditioning involves a contingency relationship between two environmental stimuli (the CS and UCS), operant conditioning involves a contingency between some aspect of the organism’s behavior and some aspect of the environment. Thus, whereas classical conditioning involves *stimulus–stimulus contingencies*, operant conditioning involves *response–stimulus contingencies*. Operant conditioning acts to alter the frequency of the response involved in the contingency relationship in a predictable way, depending on the nature of the consequence of the response. Those consequences which increase the future probability of the response are known as *reinforcers*, and those that decrease the future probability of the response are called *punishers*. A common example of a reinforcer is food for a hungry animal, and a frequently used punisher in experimental studies of operant conditioning

is mild electric shock (cf. Davey, 1989b). Reinforcers and punishers can be either positive or negative, depending on the kind of change the response exerts on the environment. Positive means that the response adds something to the situation (e.g., it delivers a food pellet or electric shock), and negative means that the response removes or avoids some environmental consequence (e.g., it terminates or avoids an electric shock). Thus, reinforcer/punisher refers to the effect of the consequence on the frequency of the response, and the adjective positive/negative refers to the nature of the response consequence. This terminology is illustrated in Figure 2.

A further term that is used frequently in operant conditioning is *discriminative stimulus*. When a particular response is reinforced only in the presence of a specific stimulus (e.g., a light), the organism will come to make that response only in the presence of that stimulus and not when the stimulus is absent. In this case the light is known as a discriminative stimulus because it “sets the occasion” for the organism to respond and for the response to be reinforced. The process of learning to respond in the presence of a discriminative stimulus is called *discrimination learning*.

In an organism’s normal environment many responses are maintained by reinforcement contingencies. Analyzing an organism’s interactions with its environment in order to discover the nature and frequency of the reinforcement

contingencies that are maintaining behaviors is known as *functional analysis*, and functional analysis is a process that has been regularly utilized in clinical psychology to discover the environmental contingencies that might be maintaining problematic or dysfunctional behavior.

#### 1.13.2.2.2 Basic phenomena of operant conditioning

##### (i) Superstitious reinforcement

It is unclear what aspect of the relationship between response and reinforcer is important for the operant conditioning process. It could be that a causal relationship between response and reinforcer is necessary for operant reinforcement to occur; alternatively, it could merely be temporal contiguity between a response and a reinforcer that is necessary. Skinner (1948) argued that organisms are sensitive to contiguity between response and reinforcer in such a way that mere accidental correlation between response and reinforcer will result in the organism learning about the relationship and modifying its behavior accordingly. For example, winning money on a fruit machine may accidentally coincide with a sequence of behaviors emitted by the player (e.g., pressing buttons in a particular sequence with a particular speed, etc.). This sequence of

	REINFORCER	PUNISHER
POSITIVE	RECEIVING A FOOD PELLET FOR PRESSING A LEVER	RECEIVING AN ELECTRIC SHOCK FOR PRESSING A LEVER
NEGATIVE	PRESSING A LEVER SERVES TO AVOID OR ESCAPE ELECTRIC SHOCK	PRESSING A LEVER DELAYS OR PREVENTS THE DELIVERY OF FOOD

**Figure 2** Reinforcement and punishment as procedures in operant conditioning. Each cell contains an example of positive and negative reinforcement and punishment.

behaviors has no causal relationship to winning money, but may be perceived as doing so by the player. Hence the player emits these sequences more frequently, and they are then more likely to be accidentally associated with winning in the future, thus strengthening the ritual. This process is known as *superstitious reinforcement*, and is a common feature of operant conditioning. In clinical contexts, superstitious reinforcement may be responsible for a variety of phenomena, including inappropriate rituals and the accidental reinforcement of disruptive or dysfunctional behaviors.

### (ii) Response shaping

From the basic principles of operant conditioning, it is obvious that a particular behavior must first occur for some unspecified reason before it can be reinforced. When a behavior is commonplace in an organism's repertoire the problem of this initial occurrence is not an important one. However, when one wishes to reinforce a behavior pattern which occurs either very rarely or is particularly complex, the problem of the first occurrence of the behavior is a salient one. Instead of waiting indefinitely for the first occurrence of a rare behavior, the way around this is to reinforce *successive approximations* to the desired behavior. For instance, in order to speed up the acquisition of lever-pressing in a rat, approximations to this behavior can be successively reinforced. The experimenter may first choose to reinforce going to the side of the apparatus where the lever is situated (a response that is likely to occur quite frequently). When this is well-established, the response requirement is made stricter by reinforcing the animal only when it touches the lever, and so on. This process of response shaping is an important one in behavior modification programs based on operant conditioning, because it allows new and complex behavior repertoires to be constructed where none had existed before (e.g., in building up self-help and communication skills in individuals with learning disabilities) (see Section 1.13.4.2.3).

### (iii) Conditioned reinforcement

Some operant reinforcers are considered to have intrinsic and immediate biological importance (e.g., food to a hungry animal). However, effective reinforcers can be established which do not have this intrinsic biological value. *Conditioned reinforcers* are those which acquire their reinforcing properties by being paired with biologically important stimuli. If, for instance, a hungry rat is trained to press a lever to obtain

food, and food delivery is accompanied by a brief tone stimulus, that tone stimulus is likely to acquire reinforcing properties. This can be demonstrated by showing that rats which have received such training will continue to press the lever merely to deliver the tone (e.g., Pierrel & Sherman, 1963). Conditioned reinforcement is a valuable contributor to many behavior modification programs in that it enables clinicians to develop reinforcers which are convenient to deliver, will reinforce all members of a group, and which can be exchanged for a variety of biologically important stimuli. The most well-known of these procedures is the *Token Economy* (see Section 1.13.4.2.2).

### (iv) Schedules of reinforcement

One of the important features of operant conditioning is that not every instance of a response needs to be reinforced in order to increase the frequency of the response. Intermittent reinforcement of this kind can be programmed on what are known as *schedules of reinforcement*, with responses being reinforced on the basis of time (e.g., time since the last reinforcement) or number (e.g., every *n*th response is reinforced). Basic schedules of reinforcement generate characteristic patterns of behavior which it is not necessary to elaborate on here (see Davey, 1989b; Ferster & Skinner, 1957).

However, intermittent reinforcement of the kind used in schedules of reinforcement does have one important quality—it produces robust responding that is significantly more resistant to extinction than when continuous reinforcement is used (cf. Ferster & Skinner, 1957). This is an important consideration when behavior modifiers are attempting to develop patterns and repertoires of behavior that will survive beyond the therapeutic conditions in which they are initially reinforced.

#### 1.13.2.2.3 Special features of operant conditioning in humans

While it is clear that human subjects do learn about the relationship between their behavior and its consequences in simple operant conditioning procedures (e.g., Alloy & Tabachnik, 1984; Dickinson, Shanks, & Evenden, 1984), the variables that influence human operant performance appear to be more involved than those already implicated in the performance of nonhuman animals. For example, human operant performance is often rigid and insensitive to changes in the reinforcement contingencies. Some experimenters have encountered great difficulty in extinguishing a

simple learned response in human subjects under experimental conditions (Bijou & Baer, 1966; Buchwald, 1959). Others have reported that human responding on schedules of reinforcement frequently entails erratic rates often interspersed with long and unpredictable pauses (Davey, 1989b; Lowe, 1979; Weiner, 1969). Lowe (1983) has argued that these apparently maladaptive aspects of human operant performance can to a large extent be explained by the fact that, unlike nonhuman animal performance, much human operant performance becomes rule-governed rather than contingency-governed. That is, when faced with any learning situation, human subjects will attempt to formulate hypotheses about the relationships between behavior and reinforcement. Very often, these initial hypotheses are wrong, especially in the artificial and simplistic environment of the operant conditioning laboratory. Lowe (1983) suggests that “it is the human subject’s capacity to formulate their own descriptions of reinforcement contingencies and to use these descriptions to formulate rules to govern their behavior which results in human operant behavior being so different from that of lower animals” (p. 77). The conclusion, therefore, is that while humans are clearly able to learn about the relationship between their behavior and its consequences, the factors which translate this learning into performance are complex, and frequently involve processes not obviously observed in nonhuman performance.

### 1.13.3 HISTORICAL ASPECTS OF LEARNING THEORY AND CLINICAL PHENOMENA

#### 1.13.3.1 Classical Conditioning

##### 1.13.3.1.1 *The “Little Albert” study and attempts at its replication*

Attempts to explain human psychopathology in terms of classical conditioning processes date back to the “Little Albert” study reported by Watson and Rayner (1920). Watson and Rayner attempted to condition in 11-month-old Albert a fear of his pet white rat. They did this by pairing the pet rat (the CS) with a loud noise produced by striking an iron bar (the UCS). After several pairings of CS and UCS, Little Albert would begin to cry when the rat was introduced into the room (the CR) (see Figure 3). In the subsequent 80 years this study has drifted into psychological folklore, being quoted in most psychology textbooks (frequently with inaccurate details) as the prototypical example of a learned fear (Harris, 1979).

Yet while it appeared to constitute a demonstration of a learned fear, Watson never attempted to formulate a theory of phobias based on classical conditioning (cf. Eysenck, 1979)—mainly because at the time there was nothing available to him that resembled a cohesive body of knowledge on classical conditioning itself. When critics subsequently came to analyse conditioning accounts of acquired fears, the Watson–Rayner approach could only be judged by evaluating the adequacy of the procedure, and this—not surprisingly—was nearly always found wanting.

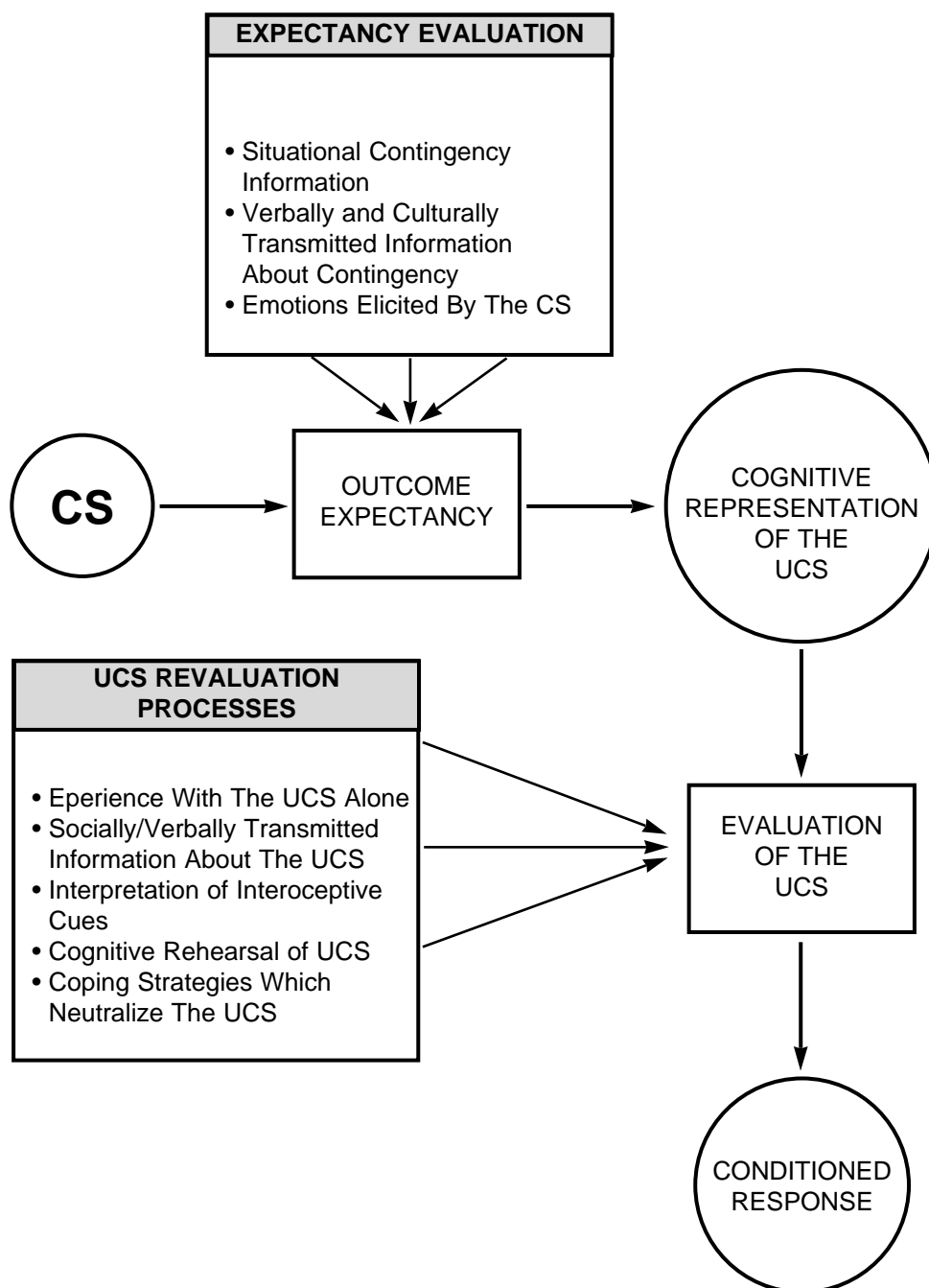
Less well publicized than the Little Albert study are the subsequent attempts to replicate it. These studies almost invariably failed to reproduce the straightforward learning reported by Watson and Rayner and often reported other curious side effects.

English (1929) reported attempting to condition a fear in a 14-month-old girl. A wooden toy duck was used as the CS which was paired with a hammer blow to a large metal bar (UCS). Even after 50 pairings, English reported no evidence of a CR to the wooden duck. However, these negative results might be attributed to a weak UCS: English found that the UCS itself failed to evoke fear in the girl. In another child—who was reported to be frightened of the noise UCS—English does report conditioned fear to a stuffed black cat.

In another study, Bregman, a student of Thorndike (1935), paired a variety of objects (wooden shapes and colored clothes) with an electric bell (UCS). None of the objects used as CSs became a focus of fear, although the electric bell UCS was genuinely frightening to the infants used as subjects.

During these attempts to replicate the original Watson and Rayner study, Valentine (1930) observed that it seemed to be easier to condition fears to furry or leathery objects rather than more common household objects. English had reported that the child in his original study—although not showing fear to a wooden duck—did subsequently learn through conditioning to fear a pair of patent leather boots she had never seen before.

More recently, writers have revived these attempted replications as evidence against simple contiguity-based conditioning accounts of specific fears (e.g., Emmelkamp, 1982; Eysenck, 1979; Marks, 1987). Even the original Little Albert study has not gone uncriticized. Some reports suggest that Little Albert only showed fear to the rat when his comforting thumb was pulled out of his mouth, while others imply that Watson and Rayner never reported a replication although they must undoubtedly have attempted one.



**Figure 3** A schematic representation of a contemporary model of human classical conditioning (see text for further explanation) (*Phobias: Handbood of theory, research and treatment*, by G. C. L. Davey, 1997, Chichester, UK: Wiley. Copyright 1997 by Wiley. Reprinted with permission).

Nevertheless, despite criticisms of the Watson and Rayner study, it was a ground-breaking attempt to apply principles derived from experimental psychology to psychopathology. Many of the failures to replicate this seminal study have themselves been criticized on methodological grounds (e.g., Delprato, 1980), and the Watson and Rayner study is still the influential forerunner of contemporary conditioning models of fears and phobias (cf. Davey, 1997).

#### 1.13.3.1.2 Pavlov and experimental neuroses

One of the most single important premises of the learning theory approach to psychopathology and therapy is that much of psychopathology can be considered as resulting from perfectly normal learning processes: that is, if an organism encounters a specific set of environmental contingencies then this will result in the learning of emotional reactions or dysfunctional re-



sponses. Thus, psychopathology was to be explained, not in terms of the dysfunction of any psychological or physiological process, but in the interaction between perfectly normally functioning learning processes and particular environmental contingencies.

Perhaps the oldest application of this view derives from Pavlov's own studies on experimental neuroses. In studies of discrimination learning he and his colleagues found that certain types of experimental procedures induced signs of anxiety and irritability in previously friendly and cooperative dogs (Pavlov, 1927, p. 29). In fact, the symptoms resembled those of behavior labeled as "neurotic" in humans. One particular procedure involved training the dog to salivate when a circle was presented on a screen. Following this, the subject was taught a discrimination between the circle (CS+) and an ellipse (CS-), with an initial ratio between the semi-axes of 2:1. The discrimination was learnt quite quickly and as training progressed the shape of the ellipse was changed until it was almost circle-like (a ratio between the semi-axes of 9:8). Kimble (1961) describes the subsequent change in the dog's behavior: "The hitherto quiet dog began to squeal in its stand, kept wriggling about, tore off with its teeth the apparatus for mechanical stimulation of the skin and bit through the tubes leading from the animal's room to the observer's . . . On being taken into the experimental room the dog now barked violently . . . In short, it presented symptoms of a condition which, in human beings, we would call neurosis" (p. 441).

Numerous other studies have described similar behavioral effects under conditions which require a very difficult discrimination (Masserman, 1943, 1950; Schneidman et al., 1971). The effects are usually quite predictable and the phenomenon extremely persistent (Anderson & Parmenter, 1941).

This type of experiment served as a forerunner of animal conditioning studies which attempted to discover the environmental conditions underlying psychopathological phenomena. Influential others included the approach-avoidance conflict paradigm reported by Masserman (1943), and the learned helplessness procedure reported by Seligman (1976). Both described the systematic nature of environmental contingencies which generated behavior resembling human psychopathology, and both were influential in determining theoretical conceptions of neurosis and depression.

#### 1.13.3.1.3 Two-factor theory of neuroses

The first attempt to construct a genuine conditioning theory of acquired fears was made

by Mowrer (1947). This was based on Mowrer's two-factor theory of learning, and attempted to combine both classical and operant conditioning into a single model. Mowrer hypothesized that on encountering pairings between a CS and an aversive UCS, fear or anxiety became conditioned to the CS. Subsequent escape from, or avoidance of, the CS acted to reduce this anxiety or fear, and this process of fear reduction acted to operantly reinforce the avoidance or escape response and maintain the phobia. This model was particularly attractive because it appealed to specific creditable learning principles, and also helped to explain what was known as the "neurotic paradox"—that is, why phobic avoidance was so persistent even in the absence of continued CS-UCS pairings.

While Mowrer's account was influential as the first integrated conditioning model of acquired fears (Eysenck & Rachman, 1965), it has generally failed to stand up to a number of theoretical and practical considerations. First, it does not explain why fear evoked by the CS does not extinguish rapidly with continued avoidance of the UCS. Second, there is little evidence for the hypothetical state of fear postulated to occur during the CS presentation and which is reduced as a result of avoidance. "Fear" does not appear to bear any simple relationship to its measures (McAllister & McAllister, 1971) and, indeed, both nonhuman animals and humans frequently show no physiological signs of fear during the presentation of an aversively conditioned CS (Brady, Kelly, & Plumlee, 1969; Davey & Arulampalam, 1982)—especially when an avoidance response is available (cf. Seligman, 1976). Nevertheless, attempts to understand phobic responding have still needed to explain what had been known as the "neurotic paradox," and more recent cognitive-based models of anxiety-based disorders have used Mowrer's two-factor theory as a basis for evolving more sophisticated models of both phobias (cf. Davey, 1997) and panic disorder (e.g., Salkovskis, 1991).

#### 1.13.3.1.4 Eysenck's theory of incubation

Over a number of years Eysenck has developed a conditioning model of neuroses designed specifically to account for the fact that phobic reactions are often observed to increase in intensity with nonreinforced presentations of the CS (Eysenck, 1976, 1979; Eysenck & Kelley, 1987). This phenomenon is known as *incubation*, and while it is a common clinical phenomenon, it is clearly inconsistent with the conditioning principle of extinction which predicts a reduction in the intensity of the CR

with successive nonreinforced presentations of the CS.

Eysenck attempted to resolve this paradox by suggesting that the CS comes to act as a partial substitute for the UCS. That is, once paired with an aversive UCS, the CS comes to evoke fear; on subsequent occasions when the CS is presented alone, the fear evoked by the CS can act as a potent reinforcer, thus generating an increasingly stronger CR over a series of CS-only presentations. Eventually, because of this process, the CR can become even stronger and more intense than the original UCR.

At the time, Eysenck's model was an attempt to explain one of the features of phobic responding which clearly did not fit comfortably into existing models of classical conditioning, and during the 1960s and 1970s represented a series of *ad hoc* models designed to explain individual anomalies in conditioning accounts of psychopathology. These individual *ad hoc* accounts have not proved to be enduring, but have been superseded by integrated conditioning models which account for a whole range of traditional criticisms leveled at conditioning models (cf. Davey, 1992, 1997; see Section 1.13.4.1.2).

#### 1.13.3.1.5 The origins of behavior therapy

Behavior therapies based on conditioning principles have been shown to provide some of the most effective forms of treatment for a whole range of psychological problems (cf. Rachman & Wilson, 1980), but this begs the question of why conditioning principles were chosen to form the basis of an approach to applied human psychology. In this respect, it is instructive to trace the origins of behavior therapy and to pinpoint some of the events from which it developed. Krasner (1971) has outlined a number of these historical factors:

(i) the early conditioning studies of J. B. Watson and his subsequent influence on experimental psychology;

(ii) the field of operant conditioning, and in particular a seminal report by Lindsley, Skinner, and Solomon (1953) on conditioned bar-pulling in psychotics;

(iii) Wolpe's development of reciprocal inhibition techniques based on the classical conditioning research of Pavlov and Hull;

(iv) the work of Eysenck and colleagues at the Maudsley Hospital, UK, working within a Hullian framework;

(v) Dollard and Miller's (1950) attempts to interpret psychoanalysis into learning theory terms;

(vi) the development of behavioral and social learning alternatives to the traditional medical model of psychopathology; and

(vii) the failure of psychodynamic and psychoanalytic psychotherapies as indicated by internal dissatisfaction and widespread external critique.

In effect, these historical influences can be distilled to form just three good reasons why learning theory was to provide a basis for new psychotherapies. First, the writings of behaviorists J. B. Watson and later B. F. Skinner (1953) were to stress a belief in the continuity of psychological mechanisms between nonhuman animals and humans. If conditioning principles had been established with animals then—since humans were only quantitatively rather than qualitatively different from animals in learning abilities—these principles should also apply to humans. Second, there was a growing dissatisfaction in the late 1940s and 1950s with the medical or disease model of psychopathology. This was gradually being replaced by a “faulty learning” account which stressed the need to treat symptoms as *bona fide* behavioral problems rather than mere symptoms of underlying causes. Third, the growing belief in the greater efficiency and usefulness of concepts derived from experimental psychology. In particular, that branch of experimental psychology which rested on the rubric of behaviorism was most favored, primarily because the principles of behavior derived from behavioristic analyses readily suggested therapeutic action and had greater predictive power than their contemporary psychodynamic counterparts.

### 1.13.3.2 Operant Conditioning

#### 1.13.3.2.1 Principles of the experimental analysis of behavior and their application to psychopathology

From its beginnings as the study of learning processes in animals (Ferster & Skinner, 1957; Skinner, 1938), operant conditioning developed as the basis for an entire philosophy of the understanding of human behavior. Pioneered by B.F. Skinner, operant reinforcement principles provided the basis for an entire analysis of human behavior, covering aspects of human behavior such as social behavior and human achievement, problem-solving, language development (Skinner, 1953, 1957, 1969, 1974), and eventually being touted as a form of behavioral technology which could shape and determine the nature of whole cultures (Skinner, 1971). This form of behaviorism has come to be known alternatively as radical behaviorism, the experimental analysis of behavior or simply behavior

analysis, and the principles surrounding this approach to the explanation of human behavior are clearly ones which would have relevance to understanding and treating human psychopathology.

The radical behaviorist approach to psychopathology which gave rise to the treatments called behavior modification techniques (see Section 1.13.4.2) is based on a number of interrelated assumptions:

(i) that human behavior is governed by the principles of learning which are embodied in operant and classical conditioning principles;

(ii) many human behaviors (including many of those which are diagnosable as psychological disorders) are acquired, maintained, and modified by these principles of learning;

(iii) since this approach emphasizes that the causes of behavior are located in the environment (in the form of reinforcement contingencies) and not "within" the individual (in the form of "motives," "intentions," etc.), these causes can be readily accessed and manipulated;

(iv) if the causes of behavior can be manipulated, then the behavior of the individual can also be manipulated in a controlled and predictable fashion.

In these assumptions were the beginnings of a technology of behavior, and this technology was first applied in the late 1950s and early 1960s to behavioral problems in the clinical setting (e.g., Ayllon & Azrin, 1968). From these beginnings developed the broader field of behavior modification and techniques then evolved from single-client therapies based on classical conditioning principles to group therapy and management procedures based on operant conditioning. The introduction of operant principles into the field of therapy was an important step because operant psychology offered a wider range of therapeutic possibilities. First, it provided a method for changing complex and integrated behavior patterns in a way that classical conditioning procedures could not. Second, it allowed the introduction of group management procedures where therapy could be conducted in large groups and in settings which resembled those found in everyday life (e.g., Token Economy procedures, Kazdin, 1975, 1981; see Section 1.13.4.2.2). Third, it provided a framework in which those variables controlling behavior could be analyzed, and as a consequence of this analysis, more appropriate therapy could be devised. Sandler and Davidson (1973) emphasize the importance of a functional analysis of this kind: "The views expressed by Skinner and other operant theorists suggest that a better understanding of pathological conditioning can be accomplished by analysing the interactions

between (a) the variables involved in an individual's behavior history, and (b) those determinants currently impinging upon the organism. With the knowledge of the former we can better predict how the latter will influence behavior" (Sandler & Davidson, 1973, pp. 63–64).

Since behavior analysts assume that psychological problems are acquired via normal learning processes, there is thus nothing pathological about them, and the removal of the problematic behavior thus removes the problem. This aspect of the traditional behaviorist approach has been controversial since it assumes that hypothetical underlying causes either do not exist or are irrelevant to successful therapy. Opponents to this view have argued that simply dealing with behavioral symptoms without addressing the basic underlying cause of the problem will result in "symptom substitution" (i.e., that the successfully treated symptom will simply be replaced by another). However, the evidence on this is equivocal. Some studies have found little evidence for symptom substitution following therapies based on learning principles (e.g., Kazdin, 1975; Rachman & Wilson, 1980), while some others have (e.g., Willems, 1974).

#### ***1.13.3.2.2 Operant reinforcement and psychopathology***

Of all the applications of learning principles to psychopathology, perhaps the most obvious one is to suggest that some features of psychopathological behavior are developed and maintained because they have reinforcing consequences. They may either reduce anxiety (as in phobic avoidance), or they may help the individual to acquire certain things which are valuable or important to them (such as attention or approval).

The way in which attention, for example, can act as a reinforcer is well illustrated in a study reported by Williams (1959). This study reported the modification of tantrum behavior in a 21-month-old infant. The child had been seriously ill in very early life and one parent had always spent time at the child's bedside waiting for him to fall asleep. When a parent was not present the child cried. This crying behavior was virtually extinguished by allowing the child to cry without reinforcing it with attention. To underline the fact that this crying behavior was controlled by attentive consequences, Williams also reports that crying was reinstated by an aunt who unwittingly reinforced the behavior by re-entering the bedroom when the child cried.

A further study by Ayllon, Haughton, and Hughes (1965) compared explanations of bizarre psychotic behavior patterns given by learning and psychodynamic theories of psychopathology. They reinforced a female schizophrenic resident in a psychiatric hospital for carrying a broom. Whenever she was observed holding the broom a nurse would approach her, offer her a cigarette, or give her a token which could be exchanged for a cigarette. Eventually, when this behavior was established, it was transferred from a continuous to an intermittent reinforcement schedule until the patient was carrying the broom around for a considerable part of the day. It was at this point that Ayllon et al. called in two psychodynamic therapists (who were unaware of the reinforcement schedule) to give their opinions on the nature of this behavior. One of them gave the following reply: "Her constant and compulsive pacing, holding a broom in the manner she does, could be seen as a ritualistic procedure, a magical action . . . Her broom would be then: (1) a child that gives her love and she gives him in return her devotion, (2) a phallic symbol, (3) the sceptre of an omnipotent queen . . . this is a magical procedure in which the patient carries out her wishes, expressed in a way that is far beyond our solid, rational and conventional way of thinking and acting" (Ayllon et al., 1965, p. 3).

There are two points to be made here. First, the description given by the psychodynamic therapist may well represent what goes on in the patient's head—we do not know for sure—but it does not in any way reflect the process by which the behavior was acquired. Second, although Ayllon et al. have systematized the acquisition process in this example, it seems reasonable to suppose that contingencies of this kind could be unwittingly set up quite frequently. Although this study does caricature the process of acquisition, it certainly implies that operant reinforcement can play a powerful role in establishing maladaptive, inappropriate, or bizarre behavior patterns.

#### **1.13.4 CURRENT APPLICATIONS OF LEARNING THEORY IN CLINICAL AND EXPERIMENTAL PSYCHOPATHOLOGY**

##### **1.13.4.1 Classical Conditioning**

###### ***1.13.4.1.1 Traditional criticisms of classical conditioning models of phobias***

The discussion of the Little Albert experiment of Watson and Rayner (1920) described in Section 1.13.3.1.1 provides a basic framework for a possible model of fears and phobias based on classical conditioning processes. Watson and

Rayner never explicitly articulated the principles of this conditioning model, although the implication was that it was a model based on findings from animal learning studies, and in particular the acquisition of conditioned fear to a CS based on the contiguous pairing of that CS with an aversive UCS. In the subsequent 60 years there have been many criticisms of the classical conditioning model of phobias, and they are ones which primarily address a contiguity-based model of classical conditioning (e.g., Emmelkamp, 1982; Rachman, 1977). It is worth detailing these criticisms, so that they can be assessed in the context of more recently developed contemporary conditioning models of fears and phobias.

First, many phobics appear unable to recall any trauma or aversive conditioning experiences at the time of the onset of their phobia (Emmelkamp, 1982; Rachman, 1977). This appears to be particularly true of some animal phobics such as snake or spider phobias (Davey, 1992c; Murray & Foote, 1979), and also height phobias and water phobias (Menzies & Clarke, 1993a, 1993b).

Second, not all people who experience pain or trauma (UCS) paired with a situation (CS) develop a phobia. For example, not everyone who has a traumatic experience undergoing dental treatment acquires a dental phobia (Lautch, 1971), not everyone who experiences a violent thunderstorm acquires a thunderstorm phobia (Liddell & Lyons, 1978), and not all fliers who experience a traumatic flying accident express a subsequent anxiety of flying (Aitken, Lister, & Main, 1981; Goorney, 1970). A simple contiguity-based conditioning model does not appear to have the power to predict when an individual will acquire a phobia and when they will not.

Third, a simple incremental-decremental model of conditioning (where pairing a CS with an aversive UCS produces an increment in fear to the CS, and an unreinforced presentation of the CS produces a decrement in fear) does not appear to account for the common clinical phenomenon of incubation of fear (see Section 1.13.3.1.4). While incubation is a common clinical phenomenon (Eysenck, 1979), it is rarely found in laboratory analogue studies (cf. Richards & Martin, 1990). Incremental-decremental models of phobias would predict a successive decrease in fear with successive nonreinforced CS presentations (extinction), not an increase in fear as found with incubation.

Fourth, simple conditioning models treat all stimuli as equally likely to enter into association with aversive consequences, yet fears and phobias are not evenly distributed across stimuli and experiences. People appear to develop

phobias of animals (snakes, spiders), heights, water, death, thunder, and fire more readily than fear of hammers, electricity outlets, knives, guns, and so on, even though the latter group of stimuli seem to have a high likelihood of being associated with trauma (Agras, Sylvester, & Oliveau, 1969; Kirkpatrick, 1984; Seligman, 1971). This uneven distribution of fears appears to violate the Pavlovian principle of equipotentiality, which states that all stimuli should be equally capable of entering into associative relationships with a consequential UCS.

Fifth, many surveys suggest that a substantial percentage of phobics appear to acquire their fear through observational learning rather than direct experience with trauma (Menzies & Clarke, 1993a, 1993b; Öst & Hugdahl, 1981; Rachman, 1977). This has generally been conceived of as a route to phobias that is an alternative to direct conditioning.

The next section argues that while these criticisms are valid in terms of traditional contiguity-based classical conditioning models, they are significantly less damaging for more recently developed contemporary cognitive conditioning models of phobias.

#### **1.13.4.1.2 Contemporary cognitive models of human classical conditioning**

Contemporary models of human classical conditioning differ from their contiguity-based predecessors in a number of important ways.

First, unlike their behaviorist forerunners, modern learning theories are happy to incorporate cognitive conceptualizations of the conditioning process. Examples of such kinds of incorporations include the willingness to attempt to discover the exact nature of the associations that are formed during conditioning—even though they cannot be directly observed (see Section 1.13.2.1.4 (vi)).

Second, it is now well accepted that many factors other than the experienced pairing of the CS and UCS can affect the strength of the association between the CS and UCS. In the case of humans, these include verbally and culturally transmitted information about the CS–UCS contingency (e.g., Dawson & Grings, 1968; Wilson, 1968), existing beliefs and expectancies about the possible consequences associated with a particular CS (Davey, 1992; Honeybourne, Matchett, & Davey, 1993), and emotional reactions currently associated with the CS (e.g., Davey & Dixon, 1996) (see Section 1.13.2.1.5 (iv)).

Third, and arguably of most importance in this context, is the finding that the strength of a CR can be radically influenced, not just by the strength of the CS–UCS association, but also by

the way in which the individual evaluates the UCS (Davey, 1989c, 1992a). In humans, there are a variety of processes that can influence the evaluation of the UCS and some of these have been described in Section 1.13.2.1.5 (iii).

Figure 3 provides a schematic representation of a contemporary cognitive model of human classical conditioning. This illustrates the kinds of factors that may influence the strength of an association between CS and UCS (Expectancy Evaluations), and also how the UCS representation's evocation of a fear CR will be influenced by how the UCS has been evaluated or revalued (UCS Revaluation Processes).

#### **1.13.4.1.3 How contemporary conditioning models of phobias account for the traditional criticisms**

##### *(i) Inability to recall trauma at the onset of the phobia*

Many phobics appear unable to recall any trauma at the time of the first appearance of their phobia. Nevertheless, the contemporary conditioning view conceives of acquisition of the CS–UCS association and modulation of the aversiveness of the UCS as relatively independent processes (Figure 3). Thus, it is quite conceivable for an individual to learn an association between a CS and UCS when the UCS is relatively nonaversive, and then subsequently (through the process of UCS inflation—see Section 1.13.2.1.5 (iii)) to have the aversiveness of the UCS inflated. This process would eventually generate a fear response to the CS without that CS ever having been explicitly paired with an *aversive* UCS.

White and Davey (1989) demonstrated this process in a laboratory analogue, and then described a putative “real-world” analogue of the following kind. An individual may witness an unknown person die of a heart attack on a bus or a train; on future occasions, riding on public transport may evoke memories of this incident, but no anxiety. Subsequently, however, that individual may be present when a close friend or relative dies of a heart attack, thus inflating the aversive properties of heart attacks. This may then give rise to acute anxiety when riding on public transport. Davey, De Jong, and Tallis (1993) report a number of actual case histories in which this type of scenario fits the etiology of a variety of anxiety disorders, including phobias, panic disorder, and obsessive-compulsive disorder. What these findings suggest is that a conditioning account of anxiety disorders is no longer bound by the need to discover contiguous stimulus-trauma experiences in the etiologies of individual cases.

(ii) *Not all people who experience pain or trauma develop a phobia*

There are a number of processes by which contemporary conditioning theory would predict that encounters with traumatic stimuli would not generate acquired fears.

First, the process of latent inhibition (see Section 1.13.2.1.4 (iii)) allows for the failure to develop a CR following pairings of a CS and UCS. Davey (1989a) tested this possibility by investigating the role of latent inhibition in the acquisition of dental phobias. He found that individuals who reported having a painful dental experience but did not acquire a dental phobia reported having their first painful experience significantly later in the dental treatment careers than individuals who did acquire a dental phobia. Thus, consistent with the operation of latent inhibition, individuals who did not acquire a phobia had the equivalent of a series of CS alone trials prior to the first pairing of the CS with a traumatic UCS.

Second, the process of UCS revaluation permits the aversiveness of any experienced UCS to be devalued following an aversive conditioning episode. There are a number of processes that have been identified as contributing to UCS reevaluation (see Section 1.13.2.1.5 (iii)), and effective devaluation of the UCS will result in the failure of the CS to evoke a substantial fear CR. Some empirical support is lent to this hypothesis by the fact that individuals with clinically diagnosed specific phobias and panic disorder are significantly less likely to report using trauma devaluation strategies (see Section 1.13.2.1.5 (iii)) than a sample of normal control subjects (Davey, Burgess, & Rashes, 1995).

(iii) *Conditioning models do not appear to account for the clinical phenomenon of incubation*

When a CS is presented alone on several occasions following conditioning, extinction should occur (see Section 1.13.2.1.4 (ii)). However, in many phobic circumstances, presentation of the CS alone leads paradoxically to incubation of the fear (Eysenck, 1979). Nevertheless, contemporary conditioning theories are able to describe some circumstances in which CS-alone presentations following conditioning may lead to increases rather than decreases in the strength of the CR. This has again been linked to the operation of UCS revaluation processes (Davey, 1997; Davey & Matchett, 1993). Davey and Matchett found that when subjects in a conditioning experiment were asked to cognitively rehearse the aversive UCS for a period following acquisition trials

and prior to a series of extinction CS-alone trials, subjects who were either high in trait anxiety levels or who had an induced anxious mood subsequently showed *increased* fear CRs to CS presentations during extinction. Davey and Matchett argued that this effect was the result of UCS rehearsal in anxious individuals inflating the aversive evaluation of the UCS, and thus causing an increased strength CR on future CS presentations. This process is analogous to the rumination about the consequences of contact with phobic stimuli that is often reported in individuals with anxiety disorders, and may provide at least a partial explanation of clinical incubation effects.

(iv) *Fears and phobias are not evenly distributed across stimuli and experiences*

There appears to be an uneven distribution of fears, with some stimuli being very common foci for phobias (e.g., snakes, spiders, heights, water), and other—equally potentially dangerous stimuli—only rarely being reported as phobic stimuli (e.g., guns, hammers, electricity outlets).

The traditional explanation of this uneven distribution of fears has been a variant of conditioning theory called *preparedness theory* (Seligman, 1971). This account hypothesizes that stimuli which tend to become the focus for fears and phobias are those which have been hazardous for our pretechnological ancestors (e.g., poisonous snakes and spiders, dangerous heights, etc.). This has resulted in the evolution of phylogenetically-based predispositions to associate biologically “prepared” stimuli with aversive outcomes.

Support for the preparedness hypothesis has come from:

(a) Laboratory conditioning studies which have paired “prepared” CSs (such as pictures of snakes and spiders) with aversive UCSs such as electric shock (e.g., Öhman, Dimberg, & Öst, 1985; Öhman, Frederickson, & Hugdahl, 1978). These studies have demonstrated a greater resistance to extinction in “prepared” stimuli than “unprepared” stimuli (McNally, 1987).

(b) Studies of the observational learning of fear of snakes in primates. These studies have indicated that monkeys who are not initially afraid of snakes will rapidly acquire an intense fear when they have watched a wild-reared monkey behaving fearfully in response to a toy snake, but will regularly fail to acquire fear towards artificial flowers or a toy rabbit using the same paradigm (Cook & Mineka, 1987, 1989, 1990; Cook, Mineka, Wolkenstein, & Laitsch, 1985; Mineka, Davidson, Cook, & Weir, 1984).

However, Davey (1995) has critically reviewed much of the evidence that is often used to support the biological preparedness hypothesis. Among the problems identified with this evidence are:

(a) that the evidence from clinical studies largely fails to support predictions from preparedness theory (de Silva, 1988; de Silva, Rachman, & Seligman, 1977); clinical phobias identified as “prepared” rarely exhibit the resistance to extinction expected of such fears,

(b) the failure of the primate studies of Mineka and colleagues to use a balanced design; as a result these studies are open to explanations other than biological preparedness, and

(c) the finding that ontogenetic fear-relevant stimuli (such as guns, electricity outlets, etc.) often display similar conditioning characteristics to so-called “biologically prepared” stimuli (such as snakes and spiders) (Davey & Dixon, 1996; Honeybourne, Matchett, & Davey, 1993; McNally & Heatherton, 1993).

As an alternative to biological preparedness, Davey (1992b, 1995) has argued that in laboratory preparedness experiments, human subjects begin the conditioning procedure with an inflated estimate of the probability of fear-relevant stimuli being followed by aversive UCSs (Davey, 1992b). Details of some of the evidence supporting this view are presented in Section 1.13.2.1.5 (iv). Subsequent studies have identified some of the factors that give rise to this UCS expectancy bias. These include estimates of the “dangerousness” of the CS (Davey & Craigie, 1997; Davey & Dixon, 1996), judgements about the semiotic similarity of the CS to the potential aversive UCS (Davey & Dixon, 1996; Hamm, Viatl, & Lang, 1989), and existing prior fear to the CS (Diamond, Matchett, & Davey, 1995; Davey, & Dixon 1996). Interestingly, nearly all of these studies have failed to find any significant differences between phylogenetic and ontogenetic fear-relevant stimuli in the determinants of UCS expectancy bias. This suggests that the same underlying process may mediate expectancy bias to both types of stimuli.

To summarize, there are at least two contrasting conditioning-based theories which attempt to explain the uneven distribution of fears. Preparedness theory is essentially an evolutionary-based account which argues that many phylogenetic fear-relevant stimuli are biologically “prepared” to enter into associations with aversive outcomes (Seligman, 1971). Alternatively, the UCS expectancy bias account says that the selective conditioning of certain stimuli results from biases in the way that individuals process information concerning fear-relevant stimuli; these biases create a

heightened expectancy of aversive outcomes following such stimuli, and hence such stimuli readily enter into association with aversive outcomes (Davey, 1995).

#### (v) *Indirect associative routes to phobias*

Some writers have suggested that, rather than being caused by specific direct conditioning experiences, the majority of phobias are caused by vicarious learning in the form of either observational learning (watching someone else being frightened in the presence of the phobic stimulus) or verbally transmitted information about the phobic stimulus and its potentially threatening consequences (Öst & Hugdahl, 1981; cf. Rachman, 1977). However, contemporary conditioning models make it clear that associations can be formed between a stimulus (CS) and its outcome (UCS) in a variety of ways—not just by direct conditioning experiences (see Section 1.13.2.1.5 (iv)). An implication of this is that an association between a CS and UCS can be learned as a result of information about the contingency, or through observing someone else experiencing the contingency. In terms of contemporary conditioning models, this is still conditioning. Admittedly, there is very little information at present on the strength and persistence of associations that are learned in these vicarious manners. They may be weaker and less resistant to extinction than associations acquired through direct experience, but they do appear to be mediated by the same associative mechanism that mediates conditioning through direct experience (Mineka & Cook, 1993). However, evidence that is available does suggest that direct conditioning experiences are more memorable than vicarious learning experiences, and that a majority of strong fears are attributed by individuals to direct conditioning rather than vicarious learning (Merckelbach, de Ruiter, van den Hout, & Hoekstra, 1989; Withers & Deane, 1995).

#### (vi) *Summary*

Section 1.13.4.1.3 has described how contemporary models of conditioning have contributed to our understanding of anxiety-based clinical disorders. Contemporary conditioning models differ from their more traditional counterparts in that they explicitly contain both an associative element and an element which allows the anxiety or fear CR to be modulated through revaluation of the UCS. Such conditioning models provide a systematic framework for the understanding of many features of anxiety-based responding.

#### 1.13.4.1.4 *Further applications of classical conditioning to anxiety-based disorders*

While classical conditioning models have primarily been applied to the understanding of phobic responding, conditioning models are increasingly being applied to an understanding of the etiology of other anxiety disorders, and, indeed, are being integrated into broader cognitive accounts of psychological disorders. At a specific level, classical conditioning models have been postulated to account for some of the important features of panic disorder (Goldstein & Chambers, 1978; Wolpe & Rowan, 1988) and post-traumatic stress disorder (PTSD) (Fairbank & Brown, 1987; Keane, Zimering, & Caddell, 1985; Kilpatrick, Veronen, & Best, 1985). However, aspects of associative learning are becoming increasingly integrated into broader cognitive models of psychopathology—especially those models that postulate the existence of associative networks underlying the expression and maintenance of fear and anxiety (e.g., Brewin, 1988, 1989; Foa & Kozak, 1986), and the revaluation of information related to threat (e.g., Brewin, Dalgleish, & Joseph, 1996). Thus, while conditioning models continue to be valuable at the level of specific phenomena, the processes contained in contemporary models of conditioning are themselves having increasing influence on the conceptualizations of broader-based cognitive models of psychopathology.

#### 1.13.4.1.5 *Classical conditioning in behavior therapy*

Ever since Wolpe (1958) published what was arguably the seminal text on behavior therapy, a variety of therapeutic techniques have been developed which have their origins in classical conditioning principles. Many of these methods have since been modified and supplemented, but almost all owe their origins to the Pavlovian principle of extinction. The assumption was that if emotional disorders were learned through a process of classical conditioning, then those disordered responses could be eliminated through applying procedures which effectively disrupted the learned CS–UCS association and extinguished the learned emotional response. The most famous of such techniques are flooding, counterconditioning, and systematic desensitization. Wolpe's main contribution was to introduce the notion of *reciprocal inhibition*, by which the emotional response is eliminated not just by extinction, but also by attaching a response to the emotion-eliciting stimulus which is incompatible with the pathological emotion.

There has been a tendency to assume that such techniques are applicable only to the treatment of anxiety and phobic disorders (e.g., Bandura, 1969; Paul & Bernstein, 1973), but they have in fact been applied successfully to a wider range of disorders including sexual dysfunction (e.g., Kockott, Dittmar, & Nusselt, 1975; Mathews et al., 1976), marital conflict (e.g., Jacobson & Weiss, 1978), and addictive disorders (e.g., Lichtenstein & Danaher, 1976; O'Leary & Wilson, 1975).

Such techniques have, over the years, been subjected to rigorous tests of their internal and external validity (cf. Rachman & Wilson, 1980), with results convincingly demonstrating that such techniques are successful because of the principles on which they are based, and superior in outcome to many other differing types of psychotherapy.

##### (i) *Flooding*

Flooding is an extinction-based therapy procedure involving therapist-directed prolonged exposure to the anxiety-eliciting stimulus or situation. Flooding can be conducted *in vivo* or *in vitro*, the latter by asking the client to imagine extended contact with their phobic stimulus. It is not necessary for high levels of fear to be elicited during the flooding experience, and mere exposure appears to be the sufficient condition (Hafner & Marks, 1976; Mathews, 1978). This would be consistent with the extinction rationale on which the method is based, and inconsistent with more psychoanalytically-oriented versions of exposure therapy (such as implosion therapy) which demand that high levels of fear are elicited during exposure in order for treatment to be effective.

Studies have demonstrated that flooding is significantly more effective than placebo treatments, indicating that its success is not simply the result of client expectations (Gelder et al., 1973).

##### (ii) *Counterconditioning*

Counterconditioning is also a procedure based on Pavlovian extinction principles, but as well as extinguishing the emotional CR it develops an acceptable alternative CR. The procedure is illustrated well in an animal analogue study by Klein (1969). Initially rats were trained on a discriminated avoidance task to run between two compartments in a shuttle-box. This response was subsequently extinguished, but during the extinction procedure the rats were divided into three groups: one group was confined to the compartment where shock



had been delivered and was also given food in this compartment (the counterconditioning group); animals in the second group were simply confined in the compartment without food (flooding); and members of the third group were not confined at all (extinction). The results suggested that the counterconditioning procedure was the most effective in eliminating the conditioned response.

A variation of the counterconditioning procedure is known as systematic desensitization by reciprocal inhibition (Wolpe, 1958).

### (iii) *Systematic desensitization*

In this variant of counterconditioning, the therapist constructs a ranked list of events or stimuli to which the client reacts with increasing fear or anxiety (the fear hierarchy). The client is then trained to relax while at the same time being exposed to the stimulus condition at the bottom of the fear hierarchy. When the client feels quite relaxed in this situation, they then progress on to the next most fear-inducing event on the hierarchy (Wolpe & Lazarus, 1966). This technique involves extinction of the anxiety response through graduated exposure to the anxiety-eliciting stimuli, and counterconditioning of a response incompatible with anxiety (relaxation) to these stimuli (the principle of reciprocal inhibition).

Systematic desensitization has arguably been one of the most durable and successful of all the behavior therapy procedures. Studies have shown that there is no methodologically acceptable evidence to prove that the therapeutic benefits derived from systematic desensitization can be attributed to nonspecific treatment effects such as placebo influences and expectations of therapeutic improvement (Gelder et al., 1973; Kazdin & Wilcoxon, 1976; Wilson, 1973), and in comparative outcome studies systematic desensitization has generally been shown to be more effective than both no treatment conditions and almost every other psychotherapy variant with which it has been compared (cf. Leitenberg, 1976; Rachman & Wilson, 1980).

### (iv) *Aversion therapy*

A rather different use of classical conditioning principles is found in aversion therapy. This is where the conditioning paradigm is used to condition an aversive response to a formally attractive stimulus. Aversion therapy is most widely used in the treatment of addictive behaviors such as alcoholism, and aversive UCSs that have been used include electric shock and drugs (such as emetine) that induce unpleasant physiological reactions (cf.

Rachman & Teasdale, 1969; Wilson, 1978). Some original studies by Voegtlin and Lemere (1942) and Lemere and Voegtlin (1950) serve as examples of this method with alcoholics. In their procedure alcoholic patients were given injections of emetine or apomorphine, which quickly elicit both nausea and vomiting (UCS). Immediately prior to vomiting the patient is given a drink of their favorite alcoholic beverage (CS). The success of this procedure depends on whether the CS (alcohol) comes to elicit a CR of nausea. Some early studies of this type of conditioning therapy report relatively successful treatment of addictive disorders such as alcoholism. For example, Wiens, Montague, Manaugh, and English (1976) reported that 12 months after receiving aversion therapy, 63% of 261 treated patients were still abstaining. Nevertheless, treatment programs such as aversion therapy do appear to rely for their long-term success on related support programs such as community reinforcement (e.g., Azrin, 1976), and there has been little success in demonstrating that aversion therapy using electric shock UCSs is any more effective than placebo control conditions (Hedberg & Campbell, 1974; Wilson, 1978; Wilson, Leaf, & Nathan, 1975). However, certain types of aversion therapy do have a role in the treatment of specific disorders, especially addictive disorders, but it is a role that is best embedded in a multifaceted approach to therapy.

### (v) *Recent developments in the treatment of anxiety-based disorders*

While classical conditioning principles have contributed substantially to the development of exposure-based therapies, the last 10–15 years have seen rapid and important changes in the nature of therapies for anxiety-based disorders. Arguably the two most important aspects of change have been (a) the emergence of cognitive therapies, and (b) the development of treatment packages addressed to specific disorders. These changes have meant that while incorporating some elements of traditional conditioning principles in their procedures, contemporary treatment packages are considerably more multifaceted than their behavior therapy predecessors, and their primary objective is to change aspects of the cognitions maintaining the disorder (e.g., Beck, 1976; Öst, 1997).

Nevertheless, many of these contemporary procedures retain exposure as the central element in the treatment (e.g., Booth & Rachman, 1992; Öst, 1989), although what may have changed is the rationale for retaining exposure. Traditional behavior therapies such as flooding and systematic desensitization

included exposure because of its role in facilitating extinction of any associative link between the phobic stimulus (CS) and fearful outcomes (UCS). However, the perceived role of exposure appears to have changed somewhat to one that either disconfirms dysfunctional beliefs about the phobic stimulus (e.g., Öst, 1997; Salkovskis, 1981) or facilitates the acquisition of behaviors designed to help the individual cope with contact with the phobic stimulus or situation (e.g., “guided mastery,” Williams, Turner, & Peer, 1985).

Thus, while traditional classical conditioning principles can be seen as the seminal influence in the development of the basic behavior therapy treatments for emotional disorders, their direct influence in contemporary therapies is limited. However, what has yet to be exploited is the therapeutic value of contemporary conditioning models (see Section 1.13.4.1.2). These recent models have cognitive components (such as UCS revaluation processes) which are compatible with modern cognitive conceptions of therapy and which seem highly suited to therapeutic application; however, this potential has yet to be explored.

#### 1.13.4.2 Operant Conditioning

Operant conditioning has contributed extensively to modern-day clinical psychology, both at the level of behavioral assessment and specific techniques. This section covers some of the more influential of these applications.

##### 1.13.4.2.1 Functional analysis

One of the important by-products of the study of operant conditioning was the discovery that behavior could be understood in terms of the consistent relationships between behavior and the environment. For example, rats in a Skinner box would learn to press a lever because that act had a consistent relationship with the consequential delivery of food. This identification of consistent relationships between behavior and environmental events led to the development of the behavioral assessment technique known as functional analysis. This is currently an assessment technique that is widely used in clinical psychology to understand aberrant behavior and also to facilitate effective therapy.

While functional analysis has its roots in operant conditioning, it has now developed into an assessment technique that is considered to be theoretically neutral (Owens & Ashcroft, 1982). As a result there is often dispute about how functional analysis should be defined. A

theoretically neutral definition would be one that emphasizes the functional relationships between behavior and environment. Haynes and O'Brien (1990), for instance, define functional analysis as “The identification of important, controllable, causal functional relationships applicable to a specified set of target behaviors for an individual client” (p. 654). This definition emphasizes that the relationships identified should be controllable—otherwise they cannot be manipulated during the course of treatment, and that functional analysis is idiographic (addressing causal relationships for behavior problems of individual clients) rather than nomothetic (addressing causal relationships for a behavior problem across clients).

Functional analysis has two specific roles in the treatment process. First, it allows the therapist to identify reinforcers that may be maintaining aberrant behavior. As a result the therapist may then be able to manipulate those reinforcement contingencies in such a way that will eradicate the aberrant behavior. For example, self-injurious behaviors or challenging behaviors may be maintained by any number of reinforcing consequences such as seeking attention, being left alone, or sensory stimulation. Identifying exactly the type of consequence that reinforces such behavior allows the therapist to disrupt the contingency and reduce the frequency of the behavior through extinction (e.g., Mazaleski, Iwata, Vollmer, Zarcone, & Smith, 1993; Wacker et al., 1990), or even through presenting reinforcers noncontingently (e.g., Hagopian, Fisher, & Legacy, 1994; Vollmer, Iwata, Zarcone, Smith, & Mazaleski, 1993). Second, a functional analysis is important because it improves the therapist's ability to develop an effective behavioral intervention. For example, attempting to eradicate aberrant behaviors by reinforcing appropriate ones is less likely to be successful if the reinforcement contingencies maintaining the aberrant behaviors have not been identified and are still operating (Vollmer & Smith, 1996). This effectively means that existing reinforcement contingencies are competing with the reinforcement contingencies used to develop the appropriate new responses.

Functional analysis has been adopted across a range of clinical settings as an aid to effective treatment. These applications include aggressive/challenging behavior (O'Reilly, 1995; Samson & McDonnell, 1990), tantrums (Darby et al., 1992), pica (Mace & Knight, 1986), stereotypy (Mace, Browder, & Lyn, 1987), attention deficit hyperactivity disorder (Northrup, Broussard, Jones, George, Vollmer, & Herring, 1995), depression (Ferster, 1985), anorexia nervosa

(Slade, 1982), and self-injurious behavior (Iwata, Dorsey, Slifer, Bauman, & Richman, 1985).

#### **1.13.4.2.2 Token economy schemes**

One great advantage of operant conditioning principles is that they are more readily adaptable to group therapy and group management situations than principles derived from classical conditioning, and perhaps the first group management procedure of this kind was the token economy (Ayllon & Azrin, 1968). In this type of program the participants receive tokens (a generalized reinforcer) when they have engaged in appropriate behavior and they can, at some later time, exchange these tokens for a variety of desired items. In the psychiatric setting, these might include access to the hospital grounds, preferred consumables, etc. The token acts as a generalized conditioned reinforcer and can be delivered with the minimum of delay after the required response is emitted. Its primary use in clinical psychology is as a therapeutic program with psychiatric patients (usually inpatients, but it has increasingly been used in community programs, cf. Corrigan, 1991). Behaviors normally fostered in these programs are prosocial or self-care ones (e.g., combing hair, bathing, brushing teeth, etc.) (Ayllon & Azrin, 1965). With more withdrawn patients, tokens can be administered simply to encourage socialization. Such programs help to increase the sociability of institutionalized individuals, strengthen behaviors that are likely to be needed in life outside the institution, and help to keep the individual receptive to other, more specialized, therapies. As a technique for behavior modification, the token economy has a number of advantages: (i) it does not necessarily need professionally trained therapists to appropriately dispense tokens; (ii) such programs prevent the deterioration of normal social and self-care behaviors on admission to an institution such as a psychiatric hospital (Dickerson, Ringel, & Parente, 1994; Kazdin & Bootzin, 1972; Lippman & Motta, 1993); and (iii) they can be used in any group situation, either as a therapeutic or a management program (Kazdin, 1975, 1981).

A number of studies have demonstrated that token economies can have significant therapeutic gains. For example, Gripp and Magro (1971) showed that schizophrenic patients in a token economy ward improved significantly more than patients in a traditional ward. Herson, Eisler, Alford, and Agras (1973) found that depressed patients exhibited less depressive behavior while participating in a token economy. Gershon, Errickson, Mitchell

and Paulson (1977) found that patients in a token economy scheme were better groomed, spent more time in activities and less time in bed, and made fewer disturbing comments than patients on a traditional ward. Patients on token economy schemes also earn discharge significantly sooner than patients who are not on such a scheme (Hofmeister, Scheckenbach, & Clayton, 1979). A large-scale study by Paul and Lentz (1977) compared token economy programs with milieu therapy and traditional custodial care approaches. The results of this comprehensive four-and-a-half year study showed that participation in both the token economy and milieu therapy groups was followed by major improvements at the end of treatment. However, the token economy group produced significantly greater reductions in bizarre behavior and increases in adaptive behavior such as self-care and interpersonal social skills than the milieu therapy. Assessment of overall functioning on a number of standardized scales showed that the token economy program resulted in significantly greater improvement than the milieu therapy at every six-month evaluation.

Nevertheless, despite these positive findings, recent surveys indicate that the use of token economies in clinical settings is in serious decline (Boudewyns, Fry, & Nightengale, 1986; Corrigan, 1995; Hall & Baker, 1973). The reasons for this are not entirely clear given the demonstrated efficacy of the technique as a therapeutic process. However, a number of reasons have been put forward for this decline.

First, there are legal and ethical issues which need to be considered. This is especially so when decisions have to be made about who will participate in token economies, for how long, and what will be made available as positive reinforcers. Legislation over the past 25 years has sought to protect patients' rights, and treatment staff are severely constrained with regard to the use of more basic items as reinforcers (Glynn, 1990)—especially when patients now have a legal right to their own personal property, humane treatment including comfortable bed, chair, bedside table, nutritious meals, cheerful furnishings, and so on.

Second, one of the major challenges for token economies has been maintenance and generalization of therapeutic effects. To the extent that patients can obtain reinforcers outside the program and avoid punishment by exiting from the program, the therapeutic benefit of token economies becomes less useful (Glynn, 1990). It is true that some studies have shown that behaviors targeted for improvement in a token economy scheme return to low baseline levels outside of the program (e.g., Ayllon & Azrin,

1968; Walker & Buckley, 1968). However, there are other studies that have shown positive effects of maintenance and generalization (Banzett, Liberman, & Moore, 1984). Nevertheless, it should be pointed out that generalization is not a passive process, and clinicians must actively build into the program strategies that transfer positive effects to settings outside the treatment scheme (Stokes & Baer, 1977; Stokes & Osnes, 1988).

Third, some other proponents of the token economy have argued that its decline has been the result of unfounded misconceptions about the nature and efficacy of such programs (e.g., Corrigan, 1995). These include such misconceptions as token economies not being therapeutically effective, their benefits do not generalize, they do not provide individualized treatment, they are abusive and coercive, and they are not practical to implement in the context of present-day attempts to treat patients in the community. Corrigan (1995) argues that these are all unfounded, and that the token economy remains an important and valuable tool for the management of patients and staff in treatment settings.

#### 1.13.4.2.3 Response shaping

As noted in Section 1.13.2.2.2 (ii), response shaping is a useful procedure for strengthening rarely-occurring behaviors or building up complex response repertoires, and this method is utilized regularly in behavior modification programs. An early study by Isaacs, Thomas, and Goldiamond (1960) serves to illustrate this method. They attempted to reinstate verbal behavior in a psychiatric inpatient who had been mute for over 19 years. In this example, the target behavior occurs relatively infrequently, and thus has to be approached via the reinforcement of successive approximations to the behavior. They discovered that although the patient was withdrawn, he did appear to respond to chewing gum, which they considered would act as an effective reinforcer. They then broke down the target behavior so that it could be reached by reinforcing a series of approximations to verbal behavior. The first responses to be reinforced were fairly simple, discrete responses whose baseline levels were high enough for them to occur spontaneously within a training session. The shaping program went as follows.

(i) When the patient moved his eyes towards the chewing gum, he was reinforced by being given the gum; after 2 weeks the probability of this response was relatively high.

(ii) The experimenters then only gave the patient gum when he moved his mouth and lips;

by the end of week 3, these behaviors were relatively frequent.

(iii) The experimenters then withheld gum until the patient made vocalizations of some sort; by the end of the fourth week the patient was moving his eyes and lips and making audible “croaking” noises.

(iv) During weeks 4 and 5, the experimenter asked the patient to “say gum,” repeating this each time the patient vocalized. At the end of week 6, the patient spontaneously said “gum please.”

(v) In later sessions the patient verbally responded to questions from the experimenters, but only in the therapeutic situation.

(vi) To enable verbal behavior to generalize beyond the experimental setting, the patient was placed back on the ward and the nursing staff were asked to attend to his needs—but only if he verbalized them.

This example demonstrates a number of features of the response shaping procedure in clinical settings. First, it provides an example of how response shaping can be a powerful and effective means of establishing complex response repertoires relatively quickly. Second, it also illustrates the distinction between “arbitrary” and “natural” reinforcers in behavior modification. In this case, chewing gum was an effective reinforcer for the behaviors being shaped—but it is an “arbitrary” one in that it is not a normal reinforcer for verbal behavior. Thus, while chewing gum may have acted as an effective reinforcer during the shaping process, in order to be maintained in any way, verbalizations need to be transferred to a more “natural” reinforcer for those behaviors. This was the aim of stage (vi) in the study where the patient’s needs were met only if he verbalized them.

Finally, there was no apparent follow-up analysis of the gains achieved in this study, and one suspects that, once back in the unstructured setting of the ward, the patient in the Isaacs et al. (1960) study would have reverted to his previous mute state. However, this study does still emphasize two things. First, as is the case with token economy studies, behavior change has to be subsequently supported by stable and structured changes to the individual’s environment which will maintain the therapeutic gains achieved in the behavior modification program. Second, whether the patient in this study did revert to a mute state still does not deny the usefulness of response shaping procedures in swiftly developing relatively complex behavior repertoires—the problem of response maintenance, however, usually requires other considerations (cf. Glynn, 1990; Stokes & Baer, 1977; Stokes & Osnes, 1988).

#### 1.13.4.2.4 Multifaceted behavioral self-control programs

Operant conditioning offers a wide range of principles that the clinician can utilize for therapeutic purposes, but so far we have discussed those that center on individual principles and which are administered in relatively traditional therapist-client settings. However, operant conditioning principles can be used as the basis for devising behavior change programs that individuals can apply themselves in a way which structures and controls the interactions between their behavior and their environment. These were traditionally known as behavioral self-control programs (e.g., Thorlesen & Mahoney, 1974), but have since been adapted into multifaceted behavioral programs to deal with a variety of personal problems which include addictions, habits, obsessions, and other behavioral problems (e.g., Lutzker & Martin, 1981; Stuart & Davies, 1972). The resulting program is one that the individual can manage and assess without the need for constant, external therapeutic supervision.

Operant principles that have been incorporated into behavioral self-control programs include stimulus control (environmental planning), reinforcement/punishment, the principle of response-reinforcement contiguity, response shaping and response discrimination (response feedback), among others.

##### (i) Stimulus control (environmental planning)

This essentially involves fading out or extinguishing undesirable responses by decreasing the number of stimuli which will elicit them. For example, in the treatment of obesity, eating responses will occur in the presence of very many environmental cues that all gain control over the response. To regulate overeating, therefore, it is important to begin by reducing the number of these cues. This can be achieved by eating only in specific environments (such as a kitchen), or even eating from one particular, distinctively patterned plate, and by never engaging in reinforcing activities while eating (for example, watching TV or reading an interesting magazine) (Ferster, Nurnberger, & Levitt, 1962; Stuart, 1967).

##### (ii) Reinforcement/punishment

In contrast to controlling the stimuli that elicit target behaviors, the individual can also control the consequences of these behaviors by ensuring that desirable behaviors are reinforced and undesirable behaviors go unreinforced or are punished. The control of the consequences

of behavior in this way has frequently been used in weight loss programs. Typical reinforcers include refunding of portions of a deposit or fee for meeting behavioral goals (e.g., Harris & Bruner, 1971; Harris & Hallbauer, 1973), and the return or loss of prewritten cheques (perhaps sent to some organization that the individual particularly dislikes) or valuables (Mann, 1972). While such contingencies have proven relatively effective in helping clients to meet behavioral targets, they are often inadequate in maintaining any therapeutic gains. One solution to this is to facilitate generalization of gains by using contingency contracting procedures which foster mutual reinforcement of desirable behaviors between family members (e.g., J. R. Lutzker & S. Z. Lutzker, 1977).

##### (iii) Response shaping and the setting of attainable therapeutic targets

All behavioral programs set attainment targets of some kind, and it is extremely important that any subgoals in the programs are attainable. For example, in weight-loss programs it is critical that weekly targets are attainable: if they are not met because they are overambitious, then this is tantamount to punishing the effort that was expended in attempting to meet the goal. The reinforcement of modest gains on a regular basis is advisable, and is consistent with the principles inherent in response shaping (see Section 1.13.4.2.3).

##### (iv) Response discrimination/feedback

Many habitual or addictive behaviors are characterized by the fact that the individual is not always aware of their occurrence, has poor recall of the frequency of the behavior, or, has little structured knowledge of the circumstances in which the behavior is emitted. This being the case, it is perhaps not surprising that such individuals have difficulty controlling these behaviors. One way in which this can be overcome is by including in the program a period of self-observation, where the individual records or charts information relevant to controlling the response.

This may take the form of a diary in which the person notes the time of occurrence of the target behavior, the circumstances in which the behavior is emitted, and any potentially reinforcing or punishing consequences of the behavior (Mahoney, 1974; Stuart, 1971). While this form of self-observation may be beneficial in enabling the individual to identify the frequency of target behaviors and to make readjustments to existing contingencies (i.e. it is a form of functional analysis, see Section

1.13.4.2.1), it is normally the case that self-monitoring alone is not sufficient to maintain any therapeutic gains, but it is best utilized in the context of more multifaceted programs (Bellack, Schwartz, & Rosensky, 1974; Mahoney, 1974).

(v) *Response-reinforcer contiguity/contingency*

While many people claim to be aware of the principle of operant reinforcement, most rarely apply it consistently. For instance, a student may decide to spend two hours in the library writing a paper and then reward oneself for this effort by going and having a coffee and a chat with friends. However, after two hours the student finds that he or she is working so well that he or she continues writing until concentration and motivation begins to wane, and then goes for coffee. With all the good intentions in the world, what has happened is that the student has inadvertently reinforced behaviors consistent with falling levels of concentration and motivation rather than the two hours focused work that preceded this. Thus, the essence of behavioral programming in self-control procedures is to ensure that the correct behaviors are reinforced, and that sloppy management of contingencies does not inadvertently reinforce inappropriate behaviors.

A good example of a multifaceted behavioral self-control program based on these rules is a behavioral control program aimed at weight loss devised by Stuart (1967). The main elements of this program were: (a) recording of time and quantity of food consumption (self-observation), (b) weighing in before each meal and before bedtime (aiding discrimination of the consequences of eating), (c) removal of food from all places in the house except the kitchen (environmental planning), (d) pairing eating with no other activity that might be reinforcing (such as watching TV) (behavioral programming), (e) setting a weight-loss goal of 1–2 pounds per week (setting attainable goals), (f) slowing down the pace of eating (defining appropriate responses), and (g) substituting other activities for between-meal eating (programming acceptable competing responses).

### 1.13.5 SUMMARY

This chapter has traced the history of the influence of learning theory on clinical theory and practice. It is clear that the principles of both classical and operant conditioning have played prominent roles in the development of therapeutic techniques and understanding the etiology of psychopathology. Originally, in an

attempt to put psychotherapy on a more scientific footing, it was learning theory that provided the basis for a new experimental analysis of psychopathology and a radical alternative to contemporary psychodynamic interpretations. Learning theory is still evolving—especially that area of learning theory that is concerned with human conditioning—and as a result, it still has much to offer clinical psychology in terms of its contribution to the understanding of psychopathology and to the development of new and effective therapies.

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# 1.14

## Psychodynamic Theory

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## 1.14.1 INTRODUCTION

### 1.14.1.1 The Basic Psychodynamic Model

This chapter provides a review of psychodynamic theories, including classical and contemporary structural theories, developments of ego psychological models, and British and US object relational approaches. The discussion of each of these psychoanalytical schools is organized in terms of illustrating the contributions each may be seen to provide to clinical psychology in terms of etiological treatment and empirical considerations.

Psychodynamic psychology, Freud's discovery and invention (Cooper, 1985), has enjoyed considerable success as an explanatory framework for understanding psychopathology and clinical work because its few basic assumptions and propositions are open to endless elaboration, revision, and refinement. Most specific psychodynamic propositions are data dependent, that is, they may be revised or discarded without fundamentally affecting the integrity of the psychoanalytic approach. There may be some assumptions which are essential to the theory (Sandler & Joffe, 1969). These include:

- (i) *psychic determinism*, namely that cognitive, emotional and behavioral aspects of pathology may be most conveniently studied in terms of psychological causes (rather than physical causality or random biological events);
- (ii) *the pleasure–unpleasure principle*, namely that behavior may be seen as an adaptive effort at minimizing psychic pain and maximizing psychic pleasure and a sense of intrapsychic safety;
- (iii) *the biological nature* of the organism drives its psychological adaptation;
- (iv) *the dynamic unconscious*, namely that mental forces contend for control over access to consciousness and to actions; and
- (v) *the genetic–developmental* proposition which states that behavior is explicable in terms of earlier (or even earliest infantile) events.

### 1.14.1.2 The Plurality of Psychodynamic Theories and the Homogeneity of Psychodynamic Frames of Reference

Psychodynamic theories are diverse in terms of focus but share a common frame of reference. Undoubtedly because of the flexibility of its assumptions, psychodynamic theory has been in

a constant state of change since its inception. Historically, it has evolved from an early concentration on the role of instincts (sexual and aggressive) in psychopathology via a focus on the functions of the ego, to the current interest in the early mother–infant dyad and its long-term effect upon interpersonal relationships and their internal representation (object relations). In the meantime a psychology of the self has become part of most psychoanalytic theories, and the move away from a physical science metaphor for psychopathology has resulted in a clinical theory where both pathology and its treatment are viewed in terms of mental representations.

Psychodynamic theory has changed literally out of all recognition over the past 30 years. Yet mental health scientists, if they think about psychodynamic theories at all, are still likely to think of these as some parody of the libidinal developmental phases of oral, anal, phallic, and genital sexuality: excessive indulgence of any of these pleasures, as well as their unreasonable frustration, causes fixations which psychoanalysts are supposed to regard as the roots of adverse developmental consequences.

Although now clearly outdated, there was a remarkably prescient aspect to Freud's (1905b) psychosexual theory. His general approach, seeing adult pathology as the outcome of deviations from normal developmental processes, set a developmental frame of reference for psychoanalytic thought which has remained important to the present day and currently links psychodynamic thought to the vibrant discipline of developmental psychopathology (Cicchetti & Cohen, 1995). The continuity of personality from childhood through adolescence to adulthood, and the role of childhood experience in setting these trajectories, lies at the heart of the psychodynamic approach and serves to unify it.

Early theories, however, continue to exist side-by-side with more recent formulations. Some writers even suggest that a number of frameworks are necessary to provide a comprehensive theory of psychopathology and therapy (Pine, 1985). In practice, each theory has its "range of convenience," particular clinical phenomena which it addresses in a particularly helpful way. For example, dreams are still best explained in the context of Freud's topographical frame of reference (Freud, 1900). By contrast, neurotic disorders are most easily

explained psychodynamically as part of a psychology of conflict (Brenner, 1982). Axis II disorders, particularly personality disorders in the Dramatic Cluster, are most productively explored in the context of theories which have evolved as repudiations of the conflict-based models (Kohut, 1971).

#### 1.14.1.3 Problems of Validity of Psychodynamic Theories

A major shortcoming of psychodynamic models lies in the fact that hypotheses about the nature of development, psychopathology, or treatment techniques are rarely based on direct evidence. To accept clinical accounts as validating psychodynamic hypotheses runs into justifiable opposition from philosophers of science who regard them as irretrievably confounded by suggestion (e.g., Grünbaum, 1984). Psychoanalytic metapsychology is at best loosely coupled to clinical observations (Fonagy, 1982; Holzman, 1985; G. S. Klein, 1976b; Ricoeur, 1977; Schafer, 1976) and clinical reports cannot therefore provide an independent confirmation for psychodynamic theory.

Their clinical roots explain why psychoanalytic ideas tend to reflect the population of patients which preoccupied particular theoreticians. For example, Winnicott (1965c) conceived of inauthenticity and the false self as a core problem and focused on its cause as the failure of good enough mothering and the inadequate provision of a holding environment. Kohut's (1971, 1977) central clinical puzzle was the question of how one develops an enfeebled self, and he oriented his interests towards the mother's capacity for empathic responsiveness. It is rarely clear whether each psychodynamic approach is associated with a particular category of clinical cases or, as is more likely the case, theoreticians reconstruct their patients in ways that fit the theory.

There is a further reason why psychodynamic ideas are hard to integrate into the network of behavioral science knowledge even at the level of clinical description. Psychodynamic diagnostic descriptions map poorly onto current psychiatric nomenclature (Jacobson & Cooper, 1993; Shapiro, 1989). Many psychodynamically oriented clinicians claim that the phenomenological approach of modern psychiatry is inherently alien to the etiological models of psychoanalysis (A. Freud, 1965), and the intention of the framers of the *Diagnostic and statistical manual of mental disorders*, 4th edition (*DSM-IV*), is to exclude all psychodynamic data and inference as basic to clinical assessment (Frances, Pincus, Widiger, Davis, &

First, 1990). The behavior-based, descriptive approach of *DSM* minimizes clinical inference, is rooted in logical positivism and thus favors behavioral and biological orientations (M. A. Schwartz, 1991) over dynamic and other potentially useful orientations to psychopathology (M. A. Schwartz & Wiggins, 1988).

Psychodynamic accounts have a further pervasive limitation: that of gender bias. Since the work of Freud (1900), masculine development has invariably been more coherently described than its feminine counterpart (Horney, 1939). In contrast, psychodynamic models far more often implicate the mother in pathological processes than they do the father, with remarkably few exceptions (e.g., Limentani, 1989).

#### 1.14.1.4 Psychic Reality, Physical Reality, Fantasy, and Reconstruction

A specific issue concerning the status of clinical evidence has emerged recently and acquired the status of a major pseudoscientific debate: that of the validity of reconstructions of childhood traumatic experiences in therapy (Allen, 1995; Sandler & Fonagy, 1997). The classical psychoanalytic view emphasized the intrapsychic experience of the individual and was relatively uninterested in the "real" world. There was a silent assumption that the maturational stages of drives were of greater importance than so-called "accidents" of the environment. In contrast many more recent theories, based on the study of adult pathology, posit that the actual behaviors of the caregivers towards the young child, as recovered in therapy, are of crucial significance to pathogenesis (e.g., Bowlby, 1958; Kohut, 1971; Sullivan, 1953; Winnicott, 1965a). Do such reconstructions have a truth value?

There is controversy in psychoanalysis (reflecting a culture-wide debate) concerning the "knowability" of early experience. Shengold (1989) links the controversy to the eighteenth-century debate initiated by George Berkeley concerning the knowability of reality beyond the mind and its ideas. Masson (1984) fueled the current debate by his oversimplified *Assault on truth* in which he chastised Freud for having defensively abandoned and deliberately withheld evidence supporting the seduction theory of neurosis. Fredrick Crews (1995), in a series of vicious attacks, laid the blame on false memories of childhood sexual abuse at the door of psychodynamic approaches.

Faced with the unpleasant choice between risks of fabrication and denial, many psychodynamic clinicians adopted a hermeneutic

approach to psychodynamic theory (e.g., Ricoeur, 1977; Spence, 1982). They repudiate a therapeutic search for the “real” past and embrace the criterion of internal coherence as the sole appropriate test of “truth.” For example, Spence (1987) insists that psychoanalysis cannot claim to have a privileged position with regard to what was (Freud’s archaeological past), and encountering the past in the therapeutic context is an act of creation of a “plausible” coherent narrative of our patient’s life. Spence (1982) gives a critical warning: “Once stated, it (the narrative truth) becomes partially true, as it is repeated and extended, it becomes familiar; and as its familiarity adds to its plausibility, it becomes completely true” (p. 177).

There is no adequate resolution to this debate. In most of the cases the quality of the patient’s recall leaves little room for doubt as to whether abuse actually happened. In others, there is room for doubt, and while the search for meaning is a ubiquitous aspect of human personality, the therapist must resist the temptation to give false meaning to current misery, anguish, and dejection by “discovering” a spurious historical account of early deprivation. There is no special expertise concerning the client’s past which the therapist has access to beyond that which emerges as part of the relationship that the client creates with him or her in the therapeutic situation. This experience is unique, but whether it is an enactment of an actual past experience or the product of a defensively distorted representation of a self–other relationship, fantasized or real, is, at the current state of knowledge, impossible to determine.

#### 1.14.1.5 Further Controversial Issues

The integration of psychodynamic theories into a coherent singular model has been undermined by a number of unresolved issues which include the following.

(i) The relative importance for pathogenesis of early childhood (infantile) experiences in contrast to later oedipal (age 4) and middle-childhood events (see Cooper, 1983).

(ii) The interpersonal vs. intrapsychic nature of human development: is the self an individual construction as conceived by Freud or is it an intersubjective unit derived from the interaction of mother and infant (Winnicott, 1956) or infant and his or her self-objects (Kohut, 1971)?

(iii) Is the basic constitution of human nature in conflict with its environment which must tame and inhibit it as Freud (1930) and Klein (1957) conceive, or is it potentially consonant

with the external world provided that the latter does not thwart its desire for self-actualization, through malevolence and unresponsiveness (Winnicott, 1965b)?

(iv) Can stage theory used to describe child development be extended to the adult, as for example Erikson (1950a) suggests, or is development better seen as the repetition of characteristic positions throughout an individual’s life course (Klein, 1952)?

(v) If development continues throughout life, is such development merely a shift of concerns (e.g., Tyson & Tyson, 1990), or does the structure of the mind remain flexible, open to drastic alteration and the generation of new structures, more or less, throughout the lifespan (e.g., Emde, 1985)?

### 1.14.2 FREUD’S MULTIPLE MODELS OF THE MIND

#### 1.14.2.1 The Affect-trauma and Topographic Frames of Reference

His work with hypnosis led Freud (Freud & Breuer, 1895) to believe that he had discovered the etiology of neurosis in the actual event of childhood seduction. In this conception the interpersonal event of the early trauma was represented in a distorted form in the neurotic symptom. For example, a child of eight with hysterical blindness may have achieved relative internal safety by “shutting his eyes” to the memory of having witnessed his mother’s rape. In this model he assumed little by way of a mental apparatus, simply the physical conversion of emotional energy generated and damned up by trauma. The therapeutic intervention was appropriately principally cathartic.

The discovery of free association and dream interpretation as clinical-research tools prompted Freud to seek a more elaborate psychological model of mental disorder. The three-layered topography of the mind—the systems unconscious, preconscious, and conscious—motivated by sexual (and later aggressive) instincts gave adequate accounts of the therapeutic value of the recovery of repressed, unconscious sexual fantasies of early childhood. The turning away from his seduction hypothesis in favor of his second model, however, discredited psychoanalytic theory as a social theory of psychopathology and therapy. It led Freud (1905b) to attempt to explain all actions in terms of the failure of the child’s mental apparatus to deal adequately with the pressures of a maturationally predetermined sequence of drive states. Adult psychopathologies, as well as dreaming, jokes, and parapraxes, were seen as the revisiting of unresolved

childhood conflicts over sexuality (Freud, 1900, 1901, 1905a). He now saw anxiety as arising from the failure of repression of unacceptable sexual wishes (Freud, 1905b, p. 224).

#### 1.14.2.2 The Structural Frame of Reference

The fundamental influence of the social environment again found a preeminent place with the third major shift in Freud's thinking (Freud, 1920, 1923, 1926). Freud's model now recognized three mental structures: the id (sexual and aggressive instincts), the ego (the self as agent) and the superego (a personalized source of internal and social moral injunction). This new structural theory was to survive long after Freud because of the compelling fit with clinical observational data. In his dual instinct theory, he describes the child's struggle with innate destructive and self-destructive forces as well as sexual conflicts (Freud, 1920). Freud (1926) also revised his theory of anxiety from one caused by inhibited biological drives, to a psychological state linked to the perception of internal (instinctual or moral) or external danger. The danger situation was specified as the fear of helplessness resulting from loss (loss of the mother, her esteem, loss of a body-part, or loss of self-regard).

This revision achieved two goals: (i) it restored adaptation to the external world as an essential part of the psychoanalytic account, and (ii) it recast the theory into cognitive terms, making way for a representational account of the mind (see below). Freud's theory of pathology was now of inadequately resolved conflict (Freud, 1938). The hypothesis that conflicts within the human mind are essentially of three kinds (wish vs. moral injunction, wish vs. reality, and internal reality vs. external reality) has had extraordinary explanatory power. In particular, the ego's capacity to create defenses which organize characterological and symptomatic constructions as part of an adaptive process, became the cornerstone of psychodynamic theorization and clinical work in the USA (Hartmann, Kris, & Loewenstein, 1946) and Britain (A. Freud, 1946) for 30–40 years.

#### 1.14.2.3 Some Limitations of Freud's Models

The limitations of Freud's models are manifold, and many of these were pointed out by Freud's contemporaries who moved away from organized psychoanalysis. For example, Jung's controversial rejection of libido theory drew attention away from the undoubted advances he made in the understanding of narcissism and his

development of a theory of the self throughout the life-cycle (Jung, 1916, 1923). Other important omissions were:

- (i) the cultural and social context, including interpersonal aspects of development;
- (ii) the significance of the experiences of infancy;
- (iii) the developmental significance of the real behavior of the real parents, including transactional processes;
- (iv) the role of attachment and safety in development alongside the role of instinctual drives;
- (v) the synthesizing function of the self;
- (vi) the importance of the nonconflictual aspects of development;
- (vii) the role of the experiences of adolescence and adulthood in shaping normal and pathological development.

### 1.14.3 THE STRUCTURAL APPROACH

#### 1.14.3.1 American Ego Psychology

##### 1.14.3.1.1 Hartmann's classical model

Freud's third, structural, model was refined and advanced in the ego psychology of Heinz Hartmann and his colleagues. Hartmann et al. (1946) postulated an initial undifferentiated matrix which contains the individual's endowment and from which both the id and the ego originate. They also introduced the concept of an "average expectable environment," which affirmed the importance of the parental contribution to development, and outlined a scheme for the phase-specific maturation of autonomous, conflict-free ego functions, accommodating both environmental and maturational influences upon personality development.

Development of the ego is driven by a maturational pull, whereby independently emerging components and functions come to be linked, forming a coherently functioning organization (the ego) which is more complex than the sum of its parts (Hartmann, 1958, 1964c). Stages of ego development represent nodal points at which "fixation" may occur and to which, under the stress of conflict, the individual may return. For example, obsessive-compulsive disorder is seen by structural theorists (Arlow & Brenner, 1964) as a regression to the phase of ego functioning characteristic of the two-year-old (magical phenomenalism, repetitive, ritualistic behaviors).

Hartmann (1958) accurately claimed that psychoanalysts frequently used the developmental point of view in a reductionist way. His concept of the "change of function" (Hartmann, 1958, p. 25) and secondary autonomy (Hartmann, 1964a) pointed to how the same behavior



in the adult may serve quite different functions and is likely to be functionally independent from the childhood wish from which it may have originated. The persistence of dependent behavior in adulthood cannot be treated as if it were a simple repetition of the individual's early relationship with the mother. Adult behaviors should be seen as having multiple functions (Brenner, 1979). The failure to recognize this has been termed the "genetic fallacy" (Hartmann, 1964b, p. 221). It is not uncommon for primitive modes of mental functioning (e.g., splitting or identity diffusion) in severe personality-disordered individuals to be treated as evidence for the persistence or regressive recurrence of early pathogenic developmental experiences. Yet, their reemergence in adult mental functioning may be linked to later or persistent trauma (see Tyson & Tyson, 1990).

#### *1.14.3.1.2 Modern structural theory*

Partly in response to the challenge of object relations theories (see below), there has been a revival of the structural theory in psychoanalysis. Modern structural theory (see, e.g., Boesky, 1988) retains the tripartite model of id, ego, and superego, but dispenses with concepts of psychic energy and other problematic notions. The theory takes as its central premise the ubiquitous nature of internal psychic conflict (see Brenner, 1982). Brenner suggests that all mental contents (thoughts, actions, plans, fantasies, and symptoms) are compromise formations which are best conceived of as multiply determined by components of conflict:

(i) a drive derivative, conceived of in this context as an intense, personal childhood wish for gratification;

(ii) unpleasure in the form of anxiety, or depressive affect, and their ideational contents of object loss, loss of love, or castration, associated with the drive derivative;

(iii) defense, which functions to minimize unpleasure; and

(iv) manifestations of superego functioning such as guilt, self-punishment, remorse, and atonement.

Self and object representations, in this scheme, are the result of compromise formation which in their turn effect further compromises between the tendencies above.

#### *1.14.3.1.3 Extensions of the ego psychology model*

##### *(i) Contribution of René Spitz*

Spitz (1959) was one of the first "empiricists" of the psychoanalytic tradition. He proposed

that major developmental shifts in psychological organization, marked by the emergence of new behaviors and new forms of affective expression (e.g., social smiling), occurred when functions are brought into new relation with one another and are linked into a coherent unit. He drew attention to the meaning of new forms of emotional expression ("psychic organizers") such as the smiling response (2–3 months), marking the initial differentiation of self and object, eight month anxiety which indicates differentiation amongst objects, especially of the "libidinal object proper" and the assertion of self in the "no" gesture between 10 and 18 months. The way in which these organizers herald dramatic changes in interpersonal interactions was elaborated in a highly influential series of papers by Robert Emde (1980a, 1980b, 1980c).

Spitz (1965) was also a pioneer in seeing the infant's and child's human partner as "quickening" the development of the child's innate abilities and mediating all perception, behavior, and knowledge. Spitz (1957) saw self-regulation as arising out of the mother–infant relationship. Psychoanalytic observational studies repeatedly showed the ways in which constitutional, early environmental, and interactional factors contribute to the structuring of the self-regulatory process leading to adaptation or maladaptation (Spitz, 1959). The mother's emotional expression at first serves a "soothing" or "containing" function which facilitates the restoration of homeostasis and emotional equilibrium. Later, the infant uses the mother's emotional response as a signaling device to indicate safety. Later still, the infant internalizes the affective response and uses his or her own emotional reaction as a signal of safety or danger (Emde, 1981). These concepts were to become a core part of attachment theory (see below).

##### *(ii) The work of Erikson*

An important extension to the classical model was proposed by Erik Erikson (1950b). His concern was the interaction of social norms and biological drives in generating self and identity. He described eight developmental stages spanning the whole of life (e.g., the "identity crisis" of adolescence) determined by biologically caused life events that were thought to disturb the equilibrium between drives and social adjustment. Personality would be arrested if the developmental challenge was not mastered through the evolution of new skills and attitudes, compromising later developmental stages. Erikson was remarkable among psychoanalysts for his attention to cultural and family

factors and his extension of the developmental model to the entire life-cycle. His theory introduced plasticity to the psychoanalytical model, as well as attributing critical importance to the need for a coherent self concept fulfilled in a coherent, supportive social milieu.

### *(iii) The work of Edith Jacobson*

Edith Jacobson (1964) advanced the idea that the infant acquires self and object images with good (libidinal) or bad (aggressive) valences, depending on experiences of gratification or frustration with the caretaker. In order to clarify and distinguish the concepts of ego, self, and self-representation, she used the term “self-representation” to stress the notion of the self and object as they were experienced, as distinguished from external objects. She stated that the ego was a structure in contrast to the self, which is the totality of the bodily and psychic person, and defined self-representation as “the unconscious, preconscious, conscious, intra psychic representation of the bodily and mental self in the system ego” (p. 19). She assumed that introjections and identificatory processes replaced the state of primitive fusion, and, through these, traits and actions of objects became internalized parts of self-images. She was particularly concerned with superego formation, which she saw as initially polarized between pleasure and unpleasure, then by issues of strength and weakness, and finally as the internalization of ethical considerations which regulated self-esteem as well as behavior.

#### ***1.14.3.1.4 The classical model of the neurosis***

The classical model of the neurosis is well known and will not be elaborated in detail here. Childhood sexual wishes are presumed to arouse conscious repugnance when experienced in adulthood because of their aggressive or incestuous content. They can reach awareness only when disguised. The neurotic compromise represents a disguised (id) derivative of childhood sexuality, the ego’s defense, and signal anxiety marking the ego’s experience of internal danger. It unifies the wish and the reaction against it in a part of the personality that is experienced as separate (ego dystonic). This is characterized by a subjective experience of punishment, suffering, and irritation which originates from, and is designed to placate, the superego. The sequence of events associated with neurotic reaction are: (i) frustration, (ii) regression, (iii) internal incompatibility, (iv) signal anxiety, (v) defense by regression, (vi) return of the repressed, (vii) compromise formation and symptomatic disorders.

Specific neurotic reactions are thought to reflect regression to particular developmental fixations and characteristic modes of compromise formation. Regression occurs because the compromise formations established to resolve the oedipal dilemma (the so-called ubiquitous infantile neurosis) was challenged by internal or external stressors. In conversion hysteria, the compromise achieves dramatic representation in somatic form and reflects an oral or phallic fixation. In obsessional neurosis, it is assumed that the ego binds anal sadistic and aggressive drive derivatives into forms of secondary process thinking (e.g., ruminations, obsessional doubts, etc.), but it is developmentally unable to neutralize these drive derivatives and therefore aggression and anal concerns will be transparent and arouse massive anxieties (see e.g., Fenichel 1945; Glover 1949). In phobias, the fear is externalized and is wholly psychological in its presentation, but in unconscious content, may reflect quite similar developmental concerns. The process remains largely an internal one; the neurotic compromise that results in obsessions is located in the thought processes themselves.

Psychoanalytic writers have noted problems with this concept of neurosis. As the focus shifted in the psychoanalytic literature to pre-oedipal development—which had been the primary focus for the Kleinian group—questions were raised about the obligatory link between adult neurosis and oedipal issues. Outside of psychoanalysis, the concept of neurosis has been assaulted with much greater ferocity. The concept of neurosis has been discredited as the trend toward descriptive clarity and reliability in diagnosis gains the upper hand, together with the push toward atheoretical, operational definitions of psychiatric disorders and biological explanations of pathogenesis. Derided as vague, unreliable, impossible to verify empirically, overinclusive, and tied to an obsolete theory, neurosis has been excluded from psychiatry’s official diagnostic classifications. Despite this slight, neurosis refused to disappear.

#### ***1.14.3.1.5 The classical model of severe psychopathology***

Whereas notions of neurotic pathology have, on the whole, evolved little since the structural theory of Freud, models of personality disorder have become “paradigmatic” of various psychoanalytic models. As subsequent sections will illustrate, extremes of personality types, now embodied in the psychiatric diagnostic schemes as the second axis of psychiatric diagnosis (American Psychiatric Association, 1994), are

formulated radically differently across different theoretical models.

Structural theory distinguishes those character disorders which resemble neurosis in terms of dynamic considerations and those which reflect a non-neurotic pattern based on structural deficit (see Waelder, 1960). The so-called character neurosis (a concept introduced by Alexander (1930)) of the former category is assumed to be dynamically similar to neurosis with the exception that compromise formations are not split off from the ego and thus the symptoms are not experienced as ego alien or ego dystonic. More severe personality disorders—for example, narcissistic personality disorder—are regarded as a consequence of a developmental arrest, deviation, or disharmony. The structural view of such cases tends to be in terms of faulty ego development (see Gitelson, 1955; Rangell, 1955). Important ego functions such as reality testing, anxiety tolerance, and stable defenses are impaired, while others appear to retain their integrity, thus giving the patient a semblance of normality.

*(i) Structural model of borderline personality disorder*

Knight (1953) was the first to propose a comprehensive model of personality disorder in terms of ego functions impaired by traumatic development. Among the ego functions he considered were: “integration, concept formation, judgment, realistic planning, and defending against eruption into conscious thinking of id impulses and their fantasy elaborations” (p. 6). Erikson (1959a, 1959b) in his epigenetic sequence of identity formation described the syndrome of identity diffusion, which he saw as reflecting deficiencies in a sustained sense of self-sameness, temporal continuity of self experience, and a feeling of affiliation with a social group of reference. Jacobson (1964) drew attention to how these individuals, at times, experience their mental functions and bodily organs not as belonging to them, but as objects which they wish to expel. They may also attach their mental and body self to external objects. She saw them as retaining an “adolescent fluidity of moods” (Jacobson, 1964, p. 159).

*(ii) Structural theory of antisocial personality disorder*

Aichhorn (1935) was the first psychoanalyst to seriously work with delinquent individuals. In his influential formulation he posited a failure of progression from the pleasure principle to the reality principle in conjunction with a malformation of the superego. Reich (1933)

suggested that the ego kept the superego at a distance, causing it to be isolated and therefore unable to prevent the individual from yielding to an impulse.

Johnson and Szurek (1952) suggested that superego lacunae (lack of superego in certain circumscribed areas) was the nature of the superego pathology. Such gaps in the superego (which is regarded as an internalization of parental morality) were thought to occur because of the parents’ unconscious wish to act out forbidden impulses; the child is unconsciously encouraged by the parents to act in amoral ways, but is consciously discouraged from doing this. Lampl-de-Groot (1949) suggested that the balance of the superego and the ego ideal explained why certain individuals became neurotically depressed, whilst others became antisocial. The former corresponds to a severe superego and strong ego ideal, whereas the latter is a consequence of a menacing superego and a weak ego ideal.

These ideas exemplify the strengths and weaknesses of structural theory. While the suggestions fit well with clinical observation and phenomenology, their explanatory value is sustained by the assumption of the *actuality* (concrete existence) of the psychic structures proposed by Freud, yet Freud was clear that his metapsychology was, in essence, metaphoric (Schafer, 1976).

### 1.14.3.2 The Structural Developmental Tradition

#### 1.14.3.2.1 Anna Freud

Anna Freud represented, and to some degree continues to represent, the structural psychodynamic model, although her work embodies a unique psychodynamic approach to both psychopathology and clinical work. Anna Freud (1965) was one of the first coherently to adopt a developmental perspective on psychopathology. She provided a comprehensive developmental theory using the metaphor of developmental lines to stress the continuity and cumulative character and transactional nature of childhood development. For example, aspects of the child’s relationship to the mother may be described as a line moving from “dependency to emotional self-reliance to adult object relationships,” “from irresponsibility to responsibility in body management.” Unevenness of development may be regarded as a risk factor for psychiatric disturbance, and thus developmental lines have etiological significance. A child’s problem may be understood in terms of an arrest or regression in terms of a particular line of development (A. Freud, 1965).

The clinical implication of her formulation is that in addressing disturbance the psychodynamic clinician should focus not only on the determinants of symptomatic aspects of the disorder, but also on offering “developmental help” to the child and restore him/her to the “path of normal development” (A. Freud, 1981).

*(i) Developmental approach to anxiety*

The developmental approach is helpful to distinguish emotional experiences at different levels of maturity. Yorke, Wiseberg, and Freeman (1989), following in Anna Freud’s tradition, differentiate a nameless terror, a principally somatic experience developmentally rooted within the undifferentiated mother–baby unit, psychic panic dating back to a time when the infant is capable of mental experience but not yet capable of affect regulation, pervasive anxiety where there is fear of helplessness but already some capacity to control affect and a signal anxiety which functions to circumvent over-arousal by activating defenses. While this theory is compelling, there is surprisingly little observational data to support the notion that early stages of development are associated with more pervasive and intense emotional experience (Harris, 1994; Stern, 1985).

*(ii) Anna Freud’s approach to severe psychopathology*

Anna Freud agrees with structural theorists in regarding severe personality disorders as reflecting structural deficits such as defects in reality testing, the dominance of primitive defenses, limited capacities for anxiety tolerance, poor superego development, etc. She explains these as developmental disturbances (deviations or disharmonies). For example, Yorke et al. (1989) suggested that the inadequate response by the mother to an infant’s instinctual needs creates dangers and external conflict. Such disharmony of need and external environment will be most intensely felt when structuralization is not yet ready to sustain the pressures caused by the internal and external stresses thus created. Ego development will suffer because the internalizing and identificatory processes will be specifically threatened. A constant representation of the other, for example, may not develop if the early relationship with the mother is disrupted by trauma. The failure to achieve structured compromise produces the labile character of borderline and other personality disturbances. Narcissistic character disorder is seen as rooted in early emotional deprivation which compromises the

process by which objects (representations of people) are invested with instinctual energy. The individual attempts to identify with the frustrating and disappointing object, providing a focus for libidinal cathexis that heightens narcissism and cathexis of the self (ego-centrism).

**1.14.3.2.2 Margaret Mahler**

Margaret Mahler (Mahler, 1968; Mahler, Pine, & Bergman, 1975), using a naturalistic observational strategy of infants and young children, evolved an influential psychodynamic model. Of particular relevance was her description of the separation–individuation process beginning at 4–5 months and ending in the third year of life. Overall, the process is one of moving away from a symbiotic unity with the caregiver to a consolidation of individuality. A critical subphase is the 6–10 month period commencing in the latter half of the second year of life when the child’s greater awareness of separateness intensifies separation anxiety at the same time as promoting a greater need to be separate from her. The caregiver’s handling of this subphase (“rapprochement”) is thought to be critical, as the mother must combine emotional availability with a gentle push towards independence. If the balance of availability and push toward independence is weighted too much on either side, the infant may become desperately dependent and clingy, experience great difficulty in investing his or her environment with sufficient interest, and his or her pleasure and confidence in his or her own functioning will be impaired (Settlage, 1977).

Masterson (1972, 1976) believes that borderline patients experience a deep conflict between the wish for independence and the threat of loss of love and thus search for a clinging tie with a mother substitute. Such a tie will temporarily ensure a feeling of safety, but any wish for self-assertiveness will present him with the terror of abandonment. A lifelong and vicious cycle of brief blissful unions, ruptures and emptiness and depression will ensue.

He discusses “abandonment depression” as the consequence of the borderline child’s quest for separation from the withdrawing or aggressive maternal object who in turn, for pathological reasons of her own, wishes to keep the child in a symbiotic relationship with her. The patient develops a fear that “his very existence is dependent ultimately upon the presence of need gratifying and life sustaining others” (R. Klein, 1989, p. 36). The withdrawing and rewarding object representations are kept rigidly separate to maintain the possibility of symbiotic union with the rewarding object and

to ward off abandonment depression. The borderline individual's dramatic response to actual separation is thus explained by his incomplete separation from his objects, with the psychological experience of separation becoming equivalent to a loss of a part of the self. Borderline patients' common vigorous pursuing of their therapists at home, in their holidays, or in other professional activity can be understood in this way.

There is strong evidence, particularly from retrospective studies, that borderline patients appear to have parents with mental illness, personality disorder, and drug abuse (e.g., Ogata, Silk, & Goodrich, 1990a) and frequently report having experienced family violence, physical and sexual abuse and other trauma (Ogata et al., 1990b; Paris, 1992; Paris & Zweig-Frank, 1992). Evidence is only indirectly supportive of Masterson's view because data are retrospective and because experiences of maltreatment usually pertain to teenage years rather than early development. More recent developmental evidence tends to disconfirm Mahler's assumption of an early self-object merger postulated by Mahler for the first half of the first year of life (see Gergely, 1991). Nevertheless, Mahler's emphasis on the second year of life has been helpful in identifying what may be a critical period for the emergence of awareness of mental states in both self and others, in other words an appreciation of human individuality (Dunn, 1996; Fonagy et al., 1995a).

#### ***1.14.3.2.3 Sandler's reframing of the structural model***

Bringing a unique blend of developmental and psychological sophistication to the classical structural model, Joseph Sandler can be said to have achieved the most coherent integration between object relations theory and the structural model. Of greatest clinical relevance is Sandler's meticulously systematic work to move psychoanalysis to the level of representations and affects (Sandler & Rosenblatt, 1962). Sandler reframed the psychodynamic theory of motivation, highlighting the role of affects rather than drive state (Sandler, 1985). A key concept is that of safety, an aspect of phenomenal experience which is intensely sought throughout development (Sandler, 1987). Past patterns of relationships and associated emotional states are thought to be actively recreated by the individual because of the associated sense of safety, even when these patterns appear maladaptive (Sandler & Sandler, 1987). An important aspect of this formulation is the distinction between past and present uncon-

scious (Sandler & Sandler, 1987). According to Sandler, the etiological and appropriate therapeutic focus of psychodynamic approaches is not early childhood experience and primitive fantasy, but rather the current derivatives of these experiences—the ways in which unconscious wishes and fantasies of the past create dynamic conflicts and elicit defensive maneuvers in the present.

The direct influence of one individual on another within Sandler's scheme (Sandler, 1976) is accounted for by the evocation of particular roles in the mind of the other person who is being influenced. The behavioral role of the influencing person is seen as crucial in eliciting a complementary response from the other participant. In this way, within the individual's present unconscious, infantile or childhood patterns of relationships may be enacted or actualized in adult interpersonal interaction.

#### **1.14.3.3 Strengths and Weaknesses of the Model**

The quasiphysiological character of the original model has been the subject of intense criticism (e.g., G. Klein, 1976a). Many of the classical explanations of psychopathology appear to reify the hypothetical constructs of ego, superego, and id in order to produce credible explanations. To talk of lacunae in the superego as a cause of antisocial behavior is only plausible if we assume that such a structure actually exists in the mind rather than conceiving of it as a metaphor evocatively summarizing a certain range of behaviors. Stripped of reification, many structural accounts are revealed as circular. For example, an absence of the ego function of anxiety tolerance, stripped of metapsychological jargon, means no more than such an individual gets very upset in situations that induce anxiety.

This is not to say that either the clinical or the metapsychological observations are without value. In reading many of these classical papers, even with the sophistication freely provided by hindsight, it is hard not to be impressed by clinical sensitivity and the remarkable integrative abilities of many of these pioneers who, in many instances, decades before other professionals had highlighted key characteristics of important disorders.

### **1.14.4 THE OBJECT RELATIONS APPROACH**

#### **1.14.4.1 Introduction to the Object Relations Approach**

Greenberg and Mitchell (1983), in their definitive review of theoretical work on object

relations theory to date, use the term to denote theories “concerned with exploring the relationship between real, external people and the internal images and residues of relations with them and the significance of these residues for psychic functioning” (p. 14).

The rise of object relations theories signaled a change of focus in psychodynamic theories. Intrapsychic conflict, particularly conflicts relating to the sexual and aggressive drives, and the central organization of oedipal compromises and the complementary influences of biological and experiential forces in development are no longer the cornerstones of psychodynamic theory, a change which has been regretted by some (see, e.g., Spruiell, 1988). Regardless of particular theoretical models, psychodynamic thinking seems to have moved in the 1980s towards a phenomenologically based perspective which emphasizes the individual’s experience of being with others and with the therapist during clinical work (see, e.g., Loewald, 1986). The clinical emphasis upon experience inevitably drives theory away from a structural mechanistic model towards what Mitchell (1988) broadly terms “relational theory.” Patients in treatment express themselves in terms of relationships (Modell, 1990), and the move towards object-relations-based psychodynamic theories may thus be seen as led by an increasing demand on clinicians to explore clinical phenomena from the point of view of the patient.

There are several assumptions which object relations theories share (see Fonagy, Target, Steele, & Gerber, 1995b). These include:

- (i) that severe pathology has pre-oedipal origins (i.e., the first three years of life);
- (ii) that the pattern of relationships with objects becomes increasingly complex with development;
- (iii) that the stages of this development represent a maturational sequence which exists across cultures but which nevertheless may be distorted by pathological personal experiences;
- (iv) that early patterns of object relations are repeated, are in some sense fixed and reproduced throughout life;
- (v) that the developmental continuum of these relationships is isomorphic with the continuum of pathology;
- (vi) that patients’ clinical reactions to their therapist provide a window for examining healthy and pathological aspects of early relationship patterns.

There exist, however, considerable differences between psychoanalytic theories in terms of the rigor with which the problem of object relationships is tackled. Friedman (1988) differentiates between hard and soft object relations

theories. Hard theorists, in which he includes Melanie Klein, Fairbairn, and Kernberg, see much hate, anger, and destruction, and dwell on obstacles, illness, and confrontation, whereas soft object relations theorists (Balint, Winnicott, and Kohut) deal with love, innocence, growth needs, fulfillment, and progressive unfolding.

#### 1.14.4.2 The Klein–Bion Model

Melanie Klein’s (1964, 1975a, 1975c) model combines the structural model with an interpersonal, object-relations model of development. It was with the contributions of Bion (1962) that the theory became a fully relational object relations model. Until 1935 Klein was basically working with the theoretical framework of Freud and Karl Abraham. The 1935 (1975a) and 1940 (1984) papers on the depressive position, the 1946 (1952) paper on the paranoid-schizoid position, and the 1957 book *Envy and gratitude* established her as the leader of an original psychoanalytic tradition.

##### 1.14.4.2.1 Some essential Kleinian concepts

###### (i) The two basic positions

In the Kleinian model the human psyche has two basic positions: the “paranoid-schizoid” and the “depressive” (Klein, 1952). In the paranoid-schizoid position, the relationship to the object (the caregiver) is to a part object, split into a persecutory and idealized relationship, and similarly the ego (the self) is split. In the depressive position, the relation is to an integrated image, both loved and hated, and the ego is more integrated. The paranoid-schizoid superego is split between the excessively idealized ego ideal characterized by the experience of narcissistic omnipotence and the extremely persecutory superego of paranoid states. In the depressive position the superego is a hurt love object with human features.

The paranoid-schizoid position is the infant’s earliest relationship with the external world and is dominated by innate internal representations (Klein, 1975c, p. 248). The infant’s initial attempt at organizing internal and external perceptions is dominated by splitting. In this way he attributes all goodness, love, and pleasure to an ideal object, and all pain, distress, and badness to a persecutory one. All good feelings of affection and desire are aimed at the idealized good object which the infant wishes to possess, take inside (introject) and experience as himself (identify with). Negative affect (hatred, disgust, etc.) is directed to the persecutory object and projected (externalized) onto it since

the infant wishes to rid himself of everything that is experienced as bad and disruptive. The infant's mental life is envisaged as extremely labile; good rapidly turns into bad, the bad gets worse, and the good gets increasingly idealized. Each external object has at least one good and one bad representation, but both representations are partial and not the whole person.

The depressive position is marked by the infant's capacity to perceive the mother as the whole person who accounts for both his good and bad experiences and is seen by Klein (1975a, p. 310) as the central achievement or process in the child's development. At this moment the infant becomes aware of his or her own capacity to love and hate the parent. The discovery of this ambivalence and the growing capacity to recognize absence and potential loss of the attacked object, opens the child to the experience of guilt over hostility to a loved object. This is what Klein calls "depressive anxiety" as distinct from the persecutory anxieties of the earlier paranoid-schizoid position. Working through the characteristic experiences of the depressive position brings with it reparative feelings. The psychic pain associated with the integration is so great that it can lead to defenses characteristic of this position, including manic or obsessional reparation, total denial of damage or contempt. Segal (1957) links the capacities for symbolization and sublimation to depressive reparation. Once the object is perceived as a mentally independent entity, it is seen as having desires, wishes, loyalties and attachments of his/her own, and oedipal concerns about the feeling about the "third" may arise (Britton, 1989).

### *(ii) Projective identification*

For Melanie Klein (1957), projective identification is an unconscious infantile phantasy by which the infant is able to relocate his persecutory experiences by separating (splitting) them from his self representation and making them part of his image of a particular object. Disowned unconscious feelings of rage or shame are firmly believed by the infant to exist within the mother. By acting in subtle but influential ways, he may achieve a confirming reaction of criticism or even persecution. The phantasy of a magical control over an object may be achieved in this way. Projective identification is not a truly internal process and involves the object who may experience it as manipulation, seduction or a myriad of other forms of psychic influence. Spillius (1992) suggests the use of the term "evocatory projective identification" to designate instances where the recipient of projective identification is put under pressure

to have the feelings appropriate to the projector's phantasy.

Bion (1959) pointed to the general necessity for projective identification in infancy, a time when the individual is ill-equipped to absorb its impressions of the world. By projecting these elements into another human mind (a container) that has the capability to accept, absorb and transform them into meanings, his mental survival was ensured. The absence of a suitable container makes projective identification a pathogenic process of evacuation. Bion (1962) discussed the significance of the mother's capacity mentally to "contain" the baby and respond to the infant emotionally and in terms of physical care in a manner that modulates unmanageable feelings. Capable caregivers are likely to experience and transform these feelings into a tolerable form which probably combines mirroring of intolerable affect in combination with emotional signals, indicating that the affect is "contained," that is, under control (Bion's "alpha function"). The infant can cope with, accept and re-internalize what was projected, thus creating a representation of these emotional moments of interaction with the caregiver which is tolerable, in place of his original experience which was not. In time he internalizes the function of transformation and will have the capacity to contain or regulate his own negative affective states. The nonverbal nature of this process implies that physical proximity of the caregiver is essential.

### *(iii) Envy and gratitude*

Klein (1957) suggests that early, primitive envy represents a particularly malignant form of innate aggression. This is because, unlike other forms which are turned against bad objects, already seen as persecutory, envy is hatred directed to the good object and arouses a premature expression of depressive anxiety about damage to the good object. The child may resent the inevitable limitations of maternal care, cannot tolerate the mother's control over it and would prefer to destroy it rather than experience the frustration. This interferes with the primal differentiation of "good" and "bad." Excessive envy is seen as interfering with working through the paranoid-schizoid position, and ultimately it becomes the developmental precursor of many forms of confusional states (Rosenfeld, 1950).

### *(iv) Primitive transferences*

In therapy, Kleinians prefer to work exclusively with interpretations, primary transference interpretations aimed at the patient's current

anxieties. They work psychodynamically with relatively severe disorders, and stress early interpretation of negative transferences derived from the paranoid-schizoid position. Kleinians have contributed enormously to our understanding and use of projective and introjective aspects of the countertransference (e.g., Racker, 1968, chap. 6.5). A pioneer in this respect was Bion (v1962), for whom transference and countertransference are about the transfer of intolerable mental pain by projective identification, originally from infant to mother, and in the treatment situation from patient to therapist.

#### **1.14.4.2.2 Kleinian approach to neurosis**

Psychological illness reflects the predominance of the paranoid-schizoid position whereas health implies the stabilization of the depressive framework, promoting development and maturity. In neurotic states, the transition between schizo-paranoid and depressive positions is perceived as partial; the superego contains both paranoid and depressive features, manifesting as persecutory guilt. The most common anxiety is the fear of guilt and the possibility of the loss of the loved object. If the depressive position is not approached, the anxiety will be about fragmentation, annihilation, and persecution, and the reality sense will be grossly distorted by projections. This picture is more fitting of patients with severe personality disorders, such as borderline or narcissistic personalities.

Bion (1962) outlined the processes that can lead to pathology in the paranoid-schizoid position. He names two factors: (i) deficiencies in the mother's capacity for receiving the child's communications through projective identification ("reverie") (see also the formulation of primary maternal preoccupation by Winnicott, 1962), and (ii) overwhelming envy in the infant.

#### **1.14.4.2.3 Rosenfeld's developmental model of narcissism**

Rosenfeld (1964, 1988) views narcissistic states as characterized by omnipotent object relations and defenses which deny the separateness and identity of the object. He stresses the destructiveness of the narcissist's relationship with others, his ruthless use of people, his denial of their value to him and yet need for them. To recognize the object would mean recognizing the object's control over "goodness" and his vulnerability to separation from it. By introjective identification the narcissistic individual lays claim to the good part of the object and in fantasy owns it. Projective identificatory processes help him deposit his own perceived

inadequacies in others whom he can then denigrate and devalue. To deal with their envy, narcissists devalue their objects (their therapists, their spouses). Denigration serves the function of avoiding recognizing goodness in others, which the person finds threatening to his or her own delusional state of self-idealization as in possession of the "good breast."

#### **1.14.4.2.4 Kleinian approach to severe psychopathology**

Klein's (1975b) formulations were vital to the understanding of borderline personality conditions. The paranoid-schizoid condition is the template for borderline personality functioning:

(i) In object relationships, splitting predominates over repression, and others are seen as either idealized or denigrated. There is no real knowledge of the other and the inner world is populated by parts (or caricatures) of the object.

(ii) Since the depressive position is avoided and all badness is pushed into the object, there can be no genuine sadness, mourning, or guilt.

(iii) Projective identification predominates; communication cannot be meaningful, and the object is manipulated by being forced to experience unacceptable aspects of the borderline individual's personality.

Splitting is both a cause and a consequence of the borderline person's difficulty in maintaining an ambivalent, balanced view of both self and object, which in Kleinian theory would require the acknowledgment of his experientially overwhelming destructive potential. With more than one object at their disposal, borderline individuals may externalize their incapacity to integrate good and bad objects, by polarizing people working with them and constantly attacking the links between them (Main, 1957).

#### **1.14.4.2.5 Strengths and weaknesses of the Klein-Bion model**

Critics of Kleinian formulations focused on the unwarranted assumption of the early development of higher-order cognitive and perceptual capacities (see Hayman, 1989; Yorke, 1971). For example, the mechanism of projection in the paranoid-schizoid position assumes a differentiated sense of self and other, since were there not yet a differentiation of inner and outer, self and other, it would seem impossible to displace the experienced source of negative affect from the self onto an external object. By now there is detailed documentation of remarkably abstract and complex cognitive capacities of the human infant. In particular there exists compelling evidence that the infant



differentiates between the self and other; for example, at five months a baby differentiates a video image of his or her own legs moving from those of another baby's legs (Bahrick & Watson, 1985).

Kleinian assumptions concerning infantile mental capacities extend beyond the representation of self and other and assume causal reasoning on the part of the infant. Studies of the perception of causality in infancy (Bower, 1989) and causal reasoning (Golinkoff, Hardig, Carlson, & Sexton, 1984) suggest that the human mind is innately predisposed to impose a causal structure on perceptual experience. If the Kleinian model of development is inaccurate, it is so because it under- rather than overestimates the child's cognitive capacities.

More pertinent current criticisms address the "fuzziness" (Greenberg & Mitchell, 1983, pp. 148–149) of Klein's descriptions of the development of mental structure. The emphasis upon "phantasy" as the building block of mental structure confounds the experiential and nonexperiential aspects of mental functioning (see Sandler & Joffe, 1969). Klein and post-Kleinians have moved mental structuralization into the experiential realm, which carries with it the advantage of experience-nearness for clinicians, and rids theory of much reified pseudoscientific terminology. However, it bypasses essential questions concerning the nature of the mechanisms underpinning the organizations of mental functions.

### 1.14.4.3 The Fairbairn–Winnicott Model

#### 1.14.4.3.1 *Major innovations of the "Independent" tradition*

The term "Independent" is not a misnomer; unlike other psychoanalytic schools, this tradition is the work of a number of analysts working independently, without a single leader or reference point. Not surprisingly, the approach lacks the internal coherence of a more unified group. Fairbairn (1954) was the systematic theory builder, but major contributions came from Winnicott (1958a) and Balint (1959, 1968).

The focus of the Independents on early development led them away from a libidinally driven structural model to develop a "self-object" theory. Parts of the self are seen as in dynamic interaction with each other and with complementary internal and external objects. Fairbairn (1954, 1963) envisages the self as a crucial agent of motivation; there is no emotion without the self and no self without emotion (Rayner, 1991). Fairbairn (1952) wrote "The libido is primarily object-seeking (rather than pleasure seeking as in classical theory)" (p. 82).

Winnicott (1958a) described the powerful desire to develop a sense of self and an equally strong impulse to hide or falsify it. The loss of optimal intimacy with the primary object will give rise to "splitting" in the self (the ego). Conflicting multiple ego-object systems are seen as the developmental roots of psychopathology.

Winnicott (1965b) saw the child as evolving from a unity of infant and mother. The mother holds the infant, both actually and figuratively, and so gives cohesion to his or her sensorimotor elements. Holding is based on "comprehension," holding in mind, of the infant's mental state. Her primary maternal preoccupation (a partial withdrawal from activities other than the baby and a state of heightened sensitivity to her own self, her body, and the baby) helps the mother achieve a state whereby the baby is provided with the "illusion" that the mother responds accurately to his or her gesture because she is his/her own creation (a part of him/her).

Winnicott (1971a) saw object relating as arising from the experience of magical omnipotence. The breast in phantasy is the infant's creation. When self-object differentiation is incomplete, object representations are best designated self-objects. The infant's muscular "attacks" on the mother, and her survival of them, facilitate the development of the self and the mother's release from omnipotent control. The infant can perceive her as a real or separate other who can be used properly and not just omnipotently. Winnicott (1956) suggests that optimal development of self-esteem depends on the mother's capacity for affective "mirroring" of the infant. Inevitable failures and frustration are essential for ultimate adaptation in that they facilitate the breakaway from infantile omnipotence and give an opportunity to the mother to *repair* the inevitable hurt by permitting *regression* to complete fusion.

The idea of "transitional phenomena" shows how the infant uses the mother to facilitate independent functioning (Winnicott, 1971b). A favorite blanket may help to soothe the infant because it is grasped in the moment when the infant fantasizes about the breast-feeding situation, and it is associated with calling the mother (and the breast) to mind in her absence. The physical object is both the infant (the "me" aspect) and the mother (the "not-me" aspect); it is transitional in facilitating the move from the omnipotence that must occur in relating to a subjectively created object, to relating to the "real" mother who is seen as part of external reality.

The mother has to be "good-enough," but her failure is expectable and is the motivator of growth. Winnicott (1958b) stresses that the baby must not be challenged too soon about the

mother's "realness" (her independent existence) and asked to negotiate the "me and the not-me." The baby's omnipotence gives rise to the ego nuclei which will in time become integrated in the real experience of the *I* (the true self). Serious trauma at the developmental level prior to the stable differentiation of self and objects, creates a basic fault in the structure of the psyche, which Balint (1968) envisages not as a split or fracture, but as a profound misordering. A person manifesting a basic fault has an underlying feeling that something is not right about him or her; he or she is not resentful about this but invariably seeks a solution in the environment. The basic fault is seen as the developmental root of personality disorder.

#### **1.14.4.3.2 The Independent view of neurotic processes and severe psychopathology**

##### *(i) Splitting of the self*

The key contribution by Fairbairn (1944) is the proposition that early trauma of great severity is stored in memories which are "frozen" or dissociated from a person's central ego or functional self. This conception steps beyond the classical psychoanalytic notion of repression in developmental accounts of psychopathology. The classical model of pathogenesis (conflict → repression → reactivation of conflict → neurotic compromise) is still seen to apply to conflicts which reach the oedipal (3–4 year) level. The Independent model concerns disorders of the self, thought to arise out of traumatic events before that age. Anxiety, as all pathological states, is regarded as rooted in the conflicts over infantile dependence. The regressive wish to be dependent carries with it the threat of loss of identity. The progressive goal of separation generates anxiety over feeling isolated, alone, and unsupported. Whilst a retreat to the home base might offer short-term relief, eventually it creates anxiety over engulfment and loss of identity, thus reinitiating the conflict.

##### *(ii) Schizoid personality*

Schizoid personality (Fairbairn, 1952) arises out of the infantile experience that love is destructive for the mother and therefore has to be inhibited along with all intimacy. In schizoid states the ego is so split that the individual may be mystified about himself and is transiently disturbed about reality (finding the familiar in the unfamiliar and vice versa). These individuals resist perceiving others as whole persons; they hide their love, and, to protect themselves from others' love, they erect defenses designed to distance others, seeming indifferent, or even

hateful, giving themselves over to the pleasures of hating and destruction.

##### *(iii) The false self*

If trauma occurs at the stage of absolute dependency, an adaptation in the form of a "caretaker self" may develop (Winnicott, 1965a, 1971a). If the mother is unable to provide comprehension of the infant through her gestures, the infant will be forced into a situation of compliance, which has mimicry but which is alien to his or her true self. The infant, and then child, will have the capacity to "go through the motions" of interpersonal relationships, but these encounters will be with the false self which will serve to hide the true self (Winnicott, 1965b). At the extreme, it is only functional outside of intimate relationships, and when called upon within such relationships (by, for example, an intensive psychotherapeutic encounter) it may "break down" and leave unprotected an infantile and poorly developed sense of "true self."

#### **1.14.4.3.3 Strengths and weaknesses of the approach**

Post-war British psychoanalysis, particularly as exemplified in the work of Winnicott, has been immensely influential in bringing about the sea change in psychodynamic thinking which we have witnessed over the last 20 years. Winnicott, and other Independent object-relations theorists, became a source of inspiration to US object-relations theorists such as Modell (1984) and Ogden (1986). At the same time they have influenced the work of major French psychoanalytic thinkers such as Andre Green (1975). In addition, its impact on Kohutian thinking was considerable although not often acknowledged.

The major weakness of the British object-relations school is its naive reconstruction of infancy in the adult mind. Although infant research confirms some speculations and informal observations, it cannot sustain the developmental argument of a linear evolution from infancy to adulthood. Human development is far too complex for infantile experiences to have direct links to adult pathology. In fact, to the extent that such research is available, longitudinal studies of infancy suggest that personality organization is subject to reorganization throughout development, based on significant positive and negative influences (Block, Block, & Gjerde, 1988; Emde, 1988a, 1988b). The infantile experiences described by Winnicott and others are thus no less metaphorical and reductionistic than the ego psychological formulations they aimed to replace.

### 1.14.4.4 Kohut's Self-psychology

#### 1.14.4.4.1 Innovative aspects of Kohut's theory

Kohut (1971, 1977, 1984) proposed that the main developmental attainment for any individual is the achievement of a cohesive self. Self-cohesion is the primary motivational property guiding human behavior, and is derived from inevitable disappointments of grandiosity and exhibitionistic needs (Kohut, 1971).

Self-development is the internalization of the self-object. A self-object is defined as a person in the environment who performs particular functions for the self; these functions evoke the experience of selfhood (Wolf, 1988). To begin with, a mirroring selfobject (assumed to be the mother) allows the unfolding of exhibitionism and grandiosity. Frustration, when phase appropriate and optimal in degree, permits a gradual modulation of infantile omnipotence through "a transmuting internalization" of this mirroring function. Empathic responses from the mirroring selfobject facilitate the unfolding of the infant's grandiosity, exhibitionism, feelings of perfection and omnipotence, and this enables him or her to build an idealized image of the parent with whom he or she wishes to merge. The idealization of self-objects, also through internalization, leads to the development of ideals. The internalizing of the mirroring function and idealized self-object leads to the emergence of a "bipolar self" with its ambitions and ideals and the natural talents available to it. "Transmuting internalization" of the self-object leads gradually to consolidation of the *nuclear self* (Kohut & Wolf, 1978, p. 83). Kohut (1977) suggested that it is the "enfeebled self" which turns defensively towards pleasure aims (drives). Drives are breakdown products of disappointments to the self, usually involving failures in emotional attunement of selfobjects.

#### 1.14.4.4.2 Self-psychological model of mental disorder

When parents consistently fail to provide for the child's narcissistic needs, Kohut suggests that the archaic grandiose self and the idealized parent imago may become hardened and fail to be integrated into later structures. Akhtar and Anderson, however, (1982) summarize some of the behavioral features of narcissistic personality disorder included in Kohut's writing (see Kohut, 1971): rage as a reaction to threats to self-esteem, the need for revenge to deal with narcissistic injury, a lack of capacity to form and maintain relationships, perverse sexuality or a lack of sexual interest, lack of empathy with others, pathological lying, a limited capacity for

taking a playful or humorous stance towards the self, hypomanic states of exaltation, and overconcern with the bodily self. There is a defect in the self, predisposing the individual to experiences of threatened fragmentation and empty depression. These experiences are disguised by defensive and compensatory behavior such as the seeking of adulation and intense excitement.

The grandiose self persists in an unneutralized way because the child is not met with appropriate mirroring responses. The idealized parental imago will remain, and the child will be left with an unattainable, unrealistic, or partial system of values and ideas. Patients in Kohutian treatment are allowed to express both their idealization of the therapist and their own grandiosity without being met by confrontation or interpretation. The empathic stance taken by the therapist is believed to reactivate the developmental process, and, through the inevitable and gradual disappointment of the patient, the neutralization of both the grandiose self and the idealization of the caregiving figures will be resumed.

#### 1.14.4.4.3 Strengths and weaknesses of Kohut's model

Kohut's clinical approach has strong following amongst psychologists. This is principally because of the clinical relevance of his ideas and their usefulness in psychotherapeutic work. There are clear and readily implementable technical recommendations arising out of Kohut's approach based around the need to "restore the self" using therapeutic methods which perform the developmental task of transmuting internalization.

There are numerous critical reviews of Kohut's theory from within structural psychoanalytic models (see, e.g., Slap & Trunnel, 1987). Schwartz (1978), for example, finds Kohut's descriptions suffer from overinclusiveness. Fragmentations of the self are believed to include depressions, depersonalizations, and disorganizing anxieties, as well as temporary or encapsulated psychotic states.

A major problem in Kohut's more recent formulations is the implicit confusion between self and self-representation. The self is presented by Kohut in representational terms, yet Kohut ascribes to it motivational properties and tendencies such as goals, plans, and self-esteem motivation (see Kohut, 1971). In this way, the self comes to denote most, if not all, of the personality and therefore becomes a superfluous term, much as the concept of ego was overextended by ego psychologists (see, e.g., Schafer, 1976).

#### 1.14.4.5 Kernberg's Integrated Theory

##### 1.14.4.5.1 *Internalization and the construction of self, object, and affect triads*

In Otto Kernberg's model, affects serve as the primary motivational system (Kernberg, 1983). He suggests that combinations of a self-representation, an object-representation, and an affect state linking them are the essential units of psychic structure. He terms these self-object-affect triads. His model is based on reconstructions from the treatment of severely disturbed adults, which are strongly influenced by Kleinian theory. It is less concerned with the child's real experience and focuses on the force of introjects and fantasies.

Kernberg (1976) described self-image as a product of the process of internalization. For Kernberg (1976) internalization entails three processes:

(i) *Introjection*, the most basic, involves the reproduction of an interaction with the environment by means of the clustering of memory traces attached to the self- or object-image and the interactions of the two in their affective context.

(ii) *Identification* presumes the child's cognitive ability to recognize the variety of role dimensions that exist in interactions with others, and involves the capacity of the self to model itself after the object influenced by fantasy and affect. The individual's experiences of gratification and frustration influence affective states and determine the degree to which self-representation is flexible, true, and complex.

(iii) *Ego identity*, borrowed from Erikson (1959b), as the overall organization of introjections and identifications under the synthesizing influence of the ego.

##### 1.14.4.5.2 *Levels of pathology within Kernberg's system*

Kernberg suggested that all levels of disturbance are more complex in severe personality disturbance but exist across the entire spectrum of psychopathology. Kernberg (1984) sees neurotic pathology as regression to a relatively integrated, although repressed, infantile self, connected to relatively integrated although also unconscious representations of the parental object. Patients with a neurotic, rather than borderline, level of personality organization are able to integrate positive and negative representations of self and others. They have passed through developmental phases where good and bad representations of self and others were combined across affective valences and a

complex presentation containing both loving and hostile elements evolved.

Even such relatively integrated internal representations, however, contain dyadic units which reflect either a defensive or an impulsive aspect of psychic conflict. An individual is highly susceptible to anxiety when configurations of self- and object-representations are highly charged affectively, and are poorly differentiated. For example, a representation of the self as being weak and vulnerable may be coupled with an object representation of ruthless domination with a violent affective tone. When this configuration is activated in therapy or elsewhere, the individual may become highly anxious.

In more severe psychopathology, identification with self- or object-representations may rapidly alternate. For example, an individual may feel criticized, but very quickly the criticism can shift from the self onto the other; and the critic is now seen as the self who is hurt and mistreated, and the individual identifies with the critical stance. This oscillation of self and other accounts for many instances where impulses appear to change into their opposite (active into passive, good into bad). Very severe levels of character pathology are typified by primitive dissociation or splitting of internalized object relations which entail a lack of integration of self- and object-representations, projections of primitive superego nuclei, splitting, impulsivity, lack of empathy, and the unmodulated expression of libido and aggression (Kernberg, 1984). Splitting is a key feature of borderline personality organization, antisocial personalities, "as if" characters, patients with multiple sexual deviations in narcissistic personalities, addictions, and even in analytically approachable psychosis. The highly unrealistic, sharply idealized or (through aggression) highly persecutory self- and object-representations characteristic of this group do not correspond to any real relationship. As object relations are poorly integrated, the reversals of the enactment of self- and other-representations may be particularly rapid. This can make relationships with such individuals confusing and even chaotic. For example, love and hate may exist in a dissociated way side by side; several object relations may be condensed into single images.

##### 1.14.4.5.3 *Borderline personality disorder*

It should be noted that, for Kernberg, borderline is a level of psychic organization rather than a nosological entity. His criteria for the disorder include: (i) nonspecific manifestations of ego weakness (poor affect tolerance, impulse control and sublimatory capacity), (ii)

primitive defenses, (iii) identity diffusion, (iv) intact reality testing but a propensity to shift towards dream-like thinking. There is some empirical evidence in support of Kernberg's criteria (see Clarkin, Foelsch, & Kernberg, 1996; Kernberg, Goldstein, & Carr, 1981).

For Kernberg (1977), the root cause of borderline states is the intensity of destructive and aggressive impulses, and the relative weakness of ego structures available to handle them. Kernberg sees the borderline individual as using the developmentally early defenses (e.g., splitting) in an attempt to separate contradictory images of self and others to protect positive images from being overwhelmed by negative and hostile ones. Primitive idealization, also a consequence of splitting, protects the individual from the "all bad" objects through creating an omnipotent object in fantasy which is the container of grandiose identifications. Splitting also results in a "diffuse sense of identity" which is characterized by a confused internal representation of the "real" object, and an unintegrated primitive superego which sets unattainable ideals and internalized persecutory images.

Kernberg (1987) illustrates how the self-destructiveness, self-mutilating behavior, and suicidal gestures tend to coincide with intense attacks of rage towards the object. They can serve to re-establish control over the environment by evoking guilt feelings, or express unconscious guilt over the success of a deepening relationship. In some patients self-destructiveness occurs because their self-image becomes "infiltrated" with aggression, so that they experience increased self-esteem and a confirmation of their grandiosity in self-mutilation or masochistic sexual perversions. The caring professions can respond only with despair to these patients' obvious sense of triumph in their victory over pain and death. Their efforts seem futile to the patient, who at an unconscious level experiences a sense of being in control over death. Self-mutilation, such as cutting, may also protect from the identity diffusion (derealization) which is a constant threat to the fragmented internal world of the borderline individual.

#### **1.14.4.5.4 Strengths and weaknesses of Kernberg's model**

Kernberg's is perhaps the most systematically elaborated of all the psychodynamic models, yet it shares many weaknesses with all the other models we have considered. These can be briefly summarized as follows.

(i) It is logically erroneous to assume that just because some features of severe psycho-

pathology have characteristics in common with children's thinking that the functions served in the adult mind are identical in the two contexts. Thus the developmental metaphor cannot be used for explanatory purposes.

(ii) A related weakness is the emphasis on early development as the root of severe psychological disturbance. Many significant environmental influences do not make themselves felt until late childhood, adolescence, or even adulthood.

(iii) There is a lack of specificity in Kernberg's model which is apparent perhaps only because he, more than other writers, attempts actively to distinguish between subtypes of disorders. Yet it is not clear from his theory why, for example, borderline personality disordered individuals spontaneously improve with time (McGlashan, 1986; Stone, 1990).

### **1.14.4.6 Bowlby's Attachment Theory Model**

#### **1.14.4.6.1 Innovative aspects of Bowlby's approach**

The infant comes into the world predisposed to participate in social interactions. The British psychoanalyst, John Bowlby (1969, 1973, 1980) was the first to give central place to the child's biological proclivity to form attachments, to initiate, maintain, and terminate interactions with the caregiver and use him/her as a "secure base" for exploration and self-enhancement. Bowlby's (1969) critical contribution was his focus on the infant's need for unbroken (secure) early attachment to the mother. The child who does not have such provision is likely to show signs of partial deprivation: excessive need for love or for revenge, gross guilt and depression; or complete deprivation: listlessness, quiet unresponsiveness, and retardation of development. Later there are signs of superficiality, want of real feeling, lack of concentration, deceit and compulsive thieving. Later Bowlby (1973) placed these reactions into a framework of reactions to separation: protest → despair → detachment.

Bowlby's attachment theory is unlike most other psychoanalytic formulations in that it is, for the most part, prospective (Bowlby, 1969). Laboratory investigations such as those of Brazelton and colleagues provided important support for Bowlby in demonstrating the innate social disposition of the infant, and the adverse consequences if expectations of social responsiveness from the caretaker are not met (see Tronick, Als, Adamson, Wise, & Brazelton, 1978). He is also most bold in claiming that the infantile roots of pathology lie in *actual* realistically based fears.

Following Bowlby (1973), the attachment of infants to their parents is recognized across the social science disciplines as a fundamental psychological process affecting human development across the lifespan (see Goldberg, Muir, & Kerr, 1995). Secure (safe and stable) vs. insecure (anxious or ambivalent) attachment of the child to its parents has been identified as a primary influence upon the child's evolving adaptation to the environment. Quality of attachment can be successfully assessed in infancy using a simple laboratory separation between a 12–18 months-old child and the caregiver, developed by John Bowlby's closest colleague, Mary Ainsworth (Ainsworth, Blehar, Waters, & Wall, 1978). Upon reunion, secure infants actively seek contact with and are soothed by the caregiver. Insecurely attached infants either avoid the caregiver or resist the caregiver's attempt to comfort them. The pervasive influence of the quality of infant–parent bonds upon subsequent social, cognitive, and emotional development has been intensively studied (Carlson & Sroufe, 1995). A number of longitudinal studies show that attachment in infancy strongly influences many aspects of psychological adaptation, including social behavior (Skolnick, 1986), affect regulation (Erickson, Sroufe, & Egeland, 1985), cognitive resourcefulness (Grossmann & Grossmann, 1991), and psychological disturbance (Sroufe, 1989).

Infant patterns of attachment, however, do not invariably determine subsequent attachment relationships (Lamb, 1987), and this variability is probably due to qualities of the internal representation of attachment patterns.

#### **1.14.4.6.2 Attachment and psychopathology**

Bowlby (1969, 1973) suggests that disruption in the functioning of the attachment system will interfere with the child's developing capacities for regulating his behavior, emotions, and arousal. He argues that since children have many of their first experiences of emotional states (intense anger and anxiety, as well as love and happiness) in the context of their early attachment relationships, the quality of these relationships will determine their capacity for self-regulation at times of high stress. Insecurely attached children should therefore be more vulnerable to emotional and behavioral dysregulation, and have fewer opportunities to elaborate the capacity to regulate emotional experiences, than secure ones (see also Ainsworth et al., 1978). Further, Bowlby (1973, 1980) maintained that secure attachment will generate internal working models of relationships characterized by an expectation of emotional as well

as physical support, leading to positive self-concept and confidence in the availability and responsiveness of the other. Insensitive parenting will give rise to insecure models of relationships, characterized by lack of trust in the other and a self-representation as unworthy and undeserving of love and affection.

Broadly speaking, Bowlby's prediction that insecure attachments are associated with various later difficulties has been borne out by empirical research. Insecurely attached children appear to be more likely to experience fluctuating and unpredictable affective states, including intensely negative emotions such as excessive sadness and anger (Cassidy, 1994). Insecure attachments are associated with maladaptive functioning in other contexts, with problems of emotional dysregulation, heightened sensitivity to stress, pervasive anxiety and distress problems in interpersonal relationships, internalizing and externalizing disorders (Carlson & Sroufe, 1995).

#### **1.14.4.6.3 Strengths and weaknesses of Bowlby's theory**

Bowlby's approach is a general systems theory and, as such, has many strong features. It has close ties to empirical data. His approach is also consistent with the current interpersonal or relational emphasis in psychodynamic therapy (Greenberg, 1991). The notion of internal working model is a useful, if somewhat vague, metaphor (Dunn, 1996) which encompasses the three components of human goal-oriented interpersonal behavior: the self, an object, and an interactional field within which the two establish a specific pattern of relationship (Greenberg, 1991). Bowlby's theory is strong with regard to identifying "maintaining factors" in pathological transactions without placing an unduly strong emphasis on past experience.

The weakness of his theory is in failing to specify how change may be most readily achieved, or indeed what might constitute change. For interpersonalists, change is often seen as deriving from the development of a new relationship with the attachment figure of the therapist (Holmes, 1993). Traditional psychodynamic notions of aggression and conflict also are difficult to integrate with Bowlby's ideas.

### **1.14.5 EVIDENCE FOR PSYCHODYNAMIC THEORIES**

We have already touched on the speculative nature of many psychodynamic constructs. The tendency for psychodynamic theorists to identify evidence from their clinical work consistent with their theories is overwhelming and is most

likely responsible for the numerous overlapping formulations offered to fit comparable data sets. In this section we shall consider some of the areas of active research in which such interplay could have taken place more intensively given interest from either the research or the psychodynamic clinical community.

As has been emphasized throughout this chapter, the framework perhaps most consistently made use of is the developmental one. The psychodynamic assumption that childhood experience has complex causal relationships with the characteristics of adult adaptation has been supported by evidence from a number of fields of study.

There are numerous longitudinal studies which demonstrate that problems of adaptation are predictable on the basis of childhood characteristics. The longitudinal study of Block and Block (1980) demonstrated that both drug abuse (Shedler & Block, 1990) and depression (Block, Gjerde, & Block, 1991) can be predicted from childhood characteristics. Such findings are naturally equally consistent with genetic predispositions; however, many findings contain subtle specific associations which are more readily accounted for in psychodynamic terms than in terms of behavior genetics. For example, in the Block et al. (1991) report, men with depressive features were observed to have been impulse-ridden in childhood, whereas women with the same diagnosis had been overcontrolled.

Research using a wide range of methodologies uniformly reports that poor parenting practices are a major contributing factor to the development of psychiatric disorders. The balance of evidence now favors the case for the pervasive and apparently irreversible impact of inadequate early social relationships (e.g., Hodges & Tizard, 1989a, 1989b). It is also recognized that, in particular during early months, when growth and maturation are most rapid, the degree and quality of caregiver assistance may be critical for the long-term biological organization of the child (e.g., Greenough & Black, 1992). The accessibility of attachment patterns to empirical research (Ainsworth et al., 1978) has prompted numerous longitudinal studies. These have demonstrated that particularly secure mother-infant attachment predicts predominantly good long-term outcome (Carlson & Sroufe, 1995) and disorganized patterns of attachment are most likely to be associated with pathological patterns of adaptation (Lyons-Ruth, 1996). In particular, there is unassailable evidence to suggest that, at the extreme end, childhood maltreatment is a powerful cause of maladaptive behavior (Cicchetti & Toth, 1995).

As we have seen, one of the core assumptions of modern psychodynamic approaches concerns the representational processes which are assumed to underpin maladaptive behavior. Object relations approaches to psychopathology (Greenberg & Mitchell, 1983) assume that disorders are rooted in cognitive structures which bias the representation of interpersonal relationships. Object relations formulations of psychodynamic theory have much in common with approaches rooted in social cognition, as has been shown by Western (1991). Distortions or pervasive biases of social cognition are generally accepted as central to understanding manifestations of severe psychopathology, whatever the causes of such cognitive distortions may be (e.g., Mineka & Sutton, 1992). Blatt and his colleagues (e.g., Blatt et al. 1994) demonstrated cognitive distortions in the narratives of depressed patients, particularly marked deficits in the description of the self. Those patients whose experience of depression was one of emptiness, loneliness, and labile, diffuse negative affectivity are more likely to be diagnosed with borderline personality disorder. Such associations may be relatively easy to account for in terms of differences in self and object representations in borderline and non-borderline individuals. The quality of depressive experience reflects the sparsity of the internal world of the borderline individual.

Modern cognitive science has increasingly drawn on notions of unconscious cognitive processes to account for performance in the areas of memory (e.g., Schacter, 1992), thought (e.g., Jacoby & Kelly, 1992), and emotion (e.g., Murphy & Zajonc, 1993). That much cognitive processing occurs outside of awareness is an inevitable conclusion given our enhanced understanding of the complexity of these processes. Somewhat more controversial, but equally unassailable, is the idea that conscious and unconscious processing of the same theme may produce qualitatively different outputs. A study by Fazio and his colleagues (Fazio, Jackson, Dunton, & Williams, 1995) illustrates this point. They administered a measure of conscious racial attitudes to subjects at the same time as assessing unconscious or implicit attitudes by seeing the extent to which subjects' responses to adjectives was slowed down or speeded up by the presentation of black or white faces. The subjects' task was simply to categorize adjectives as good or bad. An implicit negative attitude was indicated by the face of a black person speeding up responses to negative words, and a white person's face speeding up responses to positive adjectives. The subjects then engaged in conversation with a black confederate who rated them in terms of their

friendliness as she discussed racial issues with them. Interestingly the priming task, but not the conscious attitude ratings, predicted the blind independent ratings of the confederate.

This necessarily brief survey has illustrated that psychodynamic notions are not “outlandish” from the point of view of empirical psychology. None of these studies can “prove” psychodynamic ideas, as the two pertain to independent realms of human functioning: the laboratory and the consulting room. However, taken as a whole, they point to the validity of underlying psychological processes implicitly posited by psychodynamic theoreticians when offering their model of human development and the pathological outcomes that may arise from it. Psychodynamic theory is unlikely ever to be comprehensively validated in empirical studies. All that we can hope for is that the general model of a person, painstakingly assembled on the basis of neuropsychological, social and cognitive-psychological investigations, will not be too dissimilar from the vision of human functioning which has emerged from the clinical endeavors of psychodynamically oriented practitioners.

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# 1.15

## Phenomenological, Existential, and Humanistic Foundations for Psychology as a Human Science

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### 1.15.1 INTRODUCTION

This chapter highlights the historical foundations of psychology as a human science, a rigorous, empirically-based study of humans *as* humans. That is, its approach to humans takes

into account human characteristics that include but go beyond those of the objects, materials, and processes that are addressed by the natural sciences. These human characteristics include our experiencing and acting in accordance with meanings, such as anticipated futures, pasts that

are alive in the present, and the world that we relate to personally.

In contrast, for the most part psychology has achieved its rightful status as a science by adopting the assumptions and methods of early twentieth century natural sciences, particularly physics and physiology. As this *Comprehensive Clinical Psychology* series attests, that adoption has been beneficial for clinical psychology as well as for our discipline's other specialties. However, there has been a cost: the content of traditional scientific psychology has been limited to subject matter that is amenable to the experimental and statistical methods of natural science: "If we can't count it, it doesn't count as psychology." As practicing clinicians we often have to figure out how to integrate the science we were taught with what was left out—human striving, hope, moral sensitivity, and the like—which are not reducible to biology and independent determinants. Although contemporary cognitive psychology allows much more of life into psychology, it both explains away some of it and does not quite address how uniquely human content is to be accounted for scientifically. Even as researchers and theoreticians, we find that we often have relied on aspects of life not addressed by our science in order to develop hypotheses and links among theoretical themes.

Phenomenological and existential movements have contributed philosophical foundations for a human science psychology. Humanistic psychology has promoted positive values in theory and practice. Briefly, phenomenological philosophy notes that the starting point of any science is the direct appearance (phenomenon) of subject matter to human observation. Analysis of appearances allows us to discover how humans construe meanings—the basis of all knowing, including that of the natural sciences. From a human science perspective, when we study biological (including neurological and physiological) aspects of being human, it is appropriate to use the procedures developed by those disciplines; but we should be mindful that these procedures, observations, and sense-making are all human undertakings and construals. Moreover, the studied biological aspects are always of a person; each implies and influences the other. Hence, we ought not automatically impose experimental designs and explanations based on theories and methods developed for studying nonhumans. Again, from a human science perspective, when we study humans as such, we ought to be faithful to human reality, and to develop research methods explicitly suited to that purpose.

Existential philosophy addresses the universal human condition of each individual having

repeatedly to make personal decisions and sense of his or her existence, with no guarantees except that everything human carries co-authored meanings and that life ends. Human science psychologists hold that when studying human situations as such, at some point researchers ought to attend to whatever existential themes become apparent, rather than "controlling" them out for the sake of "objective science."

The founding phenomenological and existential authors were European, primarily German and French. Humanistic psychology is a uniquely North American movement, arising in the 1960s both as a popular protest against dehumanizing trends in the culture at large, and as a course championed by certain psychologists. In its early years, it was characterized as the "human potential movement," reflecting its insistence that society in general and psychology in particular ought to address not only dysfunctions, limits, deficits, and their supposed causes, but individuals' potential for growth and for positive experiences such as joy, love, creativity, and community. Contemporary humanistic psychology continues to remind us that research, theories, and practices bring consequences to society and to individuals, and that we ought to be mindful of values as we practice psychology.

Applied human science psychology occurs within an existential–phenomenological or related hermeneutic frame. "Hermeneutic" here implies an approach which acknowledges that understandings necessarily are developed through perspectival meanings—interpretations. Human science practitioners are developing clinical and research methods, and bodies of understanding, appropriate to humans as beings who are always in relation to their world(s). In that these beings are always involved in interpersonal and societal, and hence, moral undertakings, humanistic psychology's concerns are ever present. Although a human science approach to psychology respects, and indeed in some instances counts on, the work of natural science psychology, it understands that work within a broader frame.

This chapter will now go on to address phenomenological, existential, and humanistic movements in more detail. Each section is written for readers not particularly familiar with its approach rather than for psychologists already working within this orientation. Intra-orientation disputes are bypassed. The chapter will then turn to human science psychology, which draws on the above traditions. The connections of human science psychology with hermeneutics, the "posts" (postmodernism, postenlightenment, postpositivism), and constructionism will be mentioned. Finally, because most readers are not familiar with the implications for

the practice of a human science psychology, separate sections are devoted to its mainstay research method, empirical phenomenological investigation, and to human science psychological assessment. These sections describe practical differences that flow from this alternative philosophy of science. Psychotherapy is not addressed in a separate section because it appears earlier in Section 1.15.4, and because a chapter on existential psychotherapy appears in a later volume of this series.

What follows is written in the United States, to psychologists, in the last couple of years of the twentieth century. This rendition is colored by my long affiliation with the Psychology Department of Duquesne University, which began to develop its existential-phenomenological approach to psychology as a human science some 30 years ago. Other routes to a human science psychology also could be written. Certainly hundreds of additional references could have been included.

### 1.15.2 PHENOMENOLOGY

The phenomenology addressed in this chapter is only remotely related to two common uses of the term in North America. The first refers simply to taking experience seriously, in its own right, as Carl Rogers did, or as a journalist does when summarizing reported experiences. In the present context these practices would be characterized as *phenomenal* rather than as phenomenological. That is, they refer to experience, but not to the further analysis of that experience which would develop its structure and implications for understanding the human construction of situations. "Phenomenology" in this chapter refers to just such study of ("ology") how an event or object appears ("phenomenon"). The second usage common in North America is a medical one, in which "phenomenology" refers to similar outward appearances despite diverse etiologies. For example, we might encounter an article on the phenomenology of schizophrenia, only to find that it dealt with common externally identifiable symptoms rather than with schizophrenic experience.

So-called beginnings always have their predecessors. Still, as we look back from contemporary phenomenology, its foundational philosopher is Edmund Husserl (1859–1938). Martin Heidegger (1889–1976), who addressed existential themes in his own phenomenology, has been more influential in contemporary applied psychology. Their critiques of psychology's philosophy of science continue to challenge our assumptions about humans, about ways of knowing, and about the nature of science and of psychology.

Husserl earned his doctorate in mathematics, and attempted in his first book, *Philosophie der Arithmetik*, published in 1891, to demonstrate that the basis of logic and mathematics was psychological, that is, that they were based only on the brain's operations. He later retracted and refuted this position, known as "psychologism," which was in large part an effort to establish absolute foundations for knowledge. In his critique, Husserl noted that psychological laws are generalizations from empirical observations; that is, logic is used by psychologists to gather data and validate findings. Logic thus turns out to be more reasonably viewed as a human means of checking on thinking, rather than being reducible to brain functions. Biology is necessary for thought, but not sufficient to account for human reasoning and self-reflection.

Three centuries earlier Descartes, too, had sought to establish a foundation for absolute truth. His approach was to systematically doubt all assertions until he reached the indubitable: "I think, therefore I am." The cost of this proof, however, was a radical separation of mind and matter, a separation with which psychology still contends. Husserl initiated phenomenology's resolution to that artificial distinction. In a very significant sense, Husserl *unseparated* mind and material. In contrast to Descartes' method of systematically doubting the existence of objects until ultimate certainty was discovered in the "I who thinks and doubts," Husserl's method was to return respectfully to things in the world, considering them in their own right. By "things" he meant any things of which we are conscious—*phenomena*. Experience thereby was expanded beyond sense perception, another shift of importance for psychology. Moreover, Husserl contended that consciousness is not isolated, separate from the world; consciousness is always of something, and that "something" is necessarily laden with meaning. The term "intentionality" in philosophy often refers not to purposiveness but to this inevitable attending to, intending toward of consciousness. Although we of course can distinguish our thought processes or perception (a "noetic" focus) from what we are conscious of (a "noematic" focus), the two imply each other.

Husserl referred to the everyday ways in which people are attuned to the world as the "natural attitude," a prephilosophical attitude. When we are being scientific, we adopt a much narrower stance. Within that specialized frame, we can accomplish positive advances in conceptions and technology. However, Husserl's critique remains salient for today's Western psychology: as we go about our lives as striving, hoping, creative, disciplined scientists, we have forgotten that we are more fully human than the

specialized attitude takes into account. Similarly, we are quick to explain and treat depression in terms of serotonin levels, while being forgetful that even in instances of biological predisposition, a person's struggles, losses, and defeats are also essential for the development of depression. In other words, we too readily explain all human activity in terms of behavior, sensation, neurology, or cognition, thereby ignoring or explaining away the perceiving, striving person. Working in terms of restrictive concepts can be decidedly useful for particular purposes, but our findings too are restricted when we fail to return to the experiencing person.

Husserl's philosophical method avoids severing the world (e.g., in the form of scientific categories and measurements) from the person who is in relation to that world. The phenomenological epoché involves questioning and for the moment putting aside (bracketing) one's presuppositions related to both theory and the natural attitude. As we then examine a phenomenon in its full variety, we become aware of surprises and of instances that do not fit our anticipations; we discover our assumptions, which we bracket to look anew. Husserl acknowledged, however, that although we can bracket even the question of whether the world is real, we cannot bracket our own consciousness which is an integral aspect of whatever we examine.

Husserl intended that phenomenology become a science of essences—of what was essential for any phenomenon to be that particular phenomenon. The *eidos* (essence) was known through remaining actively present to the object of consciousness and its variations, through forming language about what appeared, and through comparing these appearances to related phenomena. "Consciousness" thus is our active presence to whatever we are attending to. Husserl anticipated that various disciplines would pursue different phenomena and essences. He meant for this project to retain the unity of consciousness and world, in particular to bypass both subjectivism and empiricism. However, many of his contemporaries and many of today's philosophers and psychologists have regarded this eidetic phenomenology as moving into idealism, that is, as emphasizing thought at the expense of the world.

Nevertheless, Husserl's spelling out that consciousness is necessary for any experience, and that it is necessary for unifying our fleeting impressions into coherent ones, has had an enduring positive impact. In particular, in human science contexts, consciousness is regarded as an active contribution to our experience; nevertheless is consciousness only a passive recipient of input.

In part responding to criticism of his abstract, detached concern with essences, Husserl in his later years expanded his earlier view of the importance of the *Lebenswelt*—the lived world. The lived world is just that, the world as we live it prior to theorizing. The hyphens in expressions such as "being-in-the-world" are intended to convey that humans are not in the world in the same manner as coffee is in a cup, but rather that humans are always in a unitary relation with the world. That relation is the (necessary) context or horizon within or against which we perceive. In turn, as we study our perceptions of objects we also draw out themes of, and better understand, that lived background, world. Husserl's most influential books on this continent have been *Ideas: General introduction to pure phenomenology* (1913/1962) and *The crisis of European sciences and transcendental phenomenology* (1935–37/1970).

Heidegger studied with Husserl, was his assistant, and later was awarded what had been Husserl's chair at Freiburg University. However, Heidegger developed phenomenology in a decidedly existential direction. That is, he left behind Husserl's rather sterile focus on consciousness and its objects, and instead centered his study on being-in-the-world. Heidegger's academic background and the cultural context of his early adult years differed significantly from those of Husserl. Heidegger's basic graduate training was not in mathematics or science, but in philosophy and the history of theology. His historical context was a Europe coping with World War I, concerned about the effects of the industrial revolution, questioning society and its notions of "progress," and seeing both nationalism and finished philosophical systems as stultifying and dangerous. Søren Kierkegaard's (1813–1855) anguished and earnest writings, which came to be known as Christian existential works, were republished in this era. The educated public consumed Dostoyevsky's novels, with their vivid accounts of complex motivations, people caught in circumstances, introspection about responsibility, and life's uncertainties and agonies.

Within that context, Heidegger turned his attention to questions of human existence beyond Husserl's concern with consciousness. Heidegger explicitly eschewed metaphysical philosophy with its various claims of ultimate knowledge of the nature and organization of matter and causes. Unfortunately, his ontological philosophy is not as accessible to most readers as are the writings of phenomenological and existential psychologists and psychiatrists. In addition, Heidegger's penchant for relying on arcane etymological derivations for his distinctive use of German words has made his works



difficult to read even for Germans. Moreover, translators, faced with the dilemma of having no comparable terms in English, often have retained his original German. Heidegger's major, most influential, text is *Being and time* (1927/1962).

Heidegger set himself the task of developing an ontology—an explication of the meaning of Being. The capitalization indicates concern with the *nature* of existence and being, in contradistinction to concern with actual beings. Heidegger's term for human Being, "*Dasein*" ("da" = there; "sein" = being), emphasizes that human reality is always situated, always in relation to a surrounding "there," never without environment. However, *Dasein* does not refer to individual humans, as has sometimes been misassumed (understandably, and usually productively). Rather, for Heidegger *Dasein* refers to the character of existence as being both open to the world's possibilities and as always finding itself already in the world. We do, however, explore human Being through examining actual individuals' basic modes of being-in-the-world (*existentiata*).

Care is one such mode. We find that events and things matter to us; we cannot help but to care. But we care in authentic and in inauthentic ways. When we are open to the depth, newness, and possibilities of what we are attending to, and when we heed our responsibility for choosing that openness, we are living authentically. When we turn other beings into fixed objects or adopt a closed, unreflectively technological, or judgmental attitude, we are living inauthentically. Of course it is impossible to go about life being continuously or fully authentic. We are stuck with always being at least partially inauthentic; the challenge is to strive for authenticity. Our finitude, our always being-toward-death, is another of the basic modes of being-in-the world. Our finitude reminds us of our responsibility to make our choices within our limited time. We are always caught within temporal moments, coming from and going toward. Heidegger's distinguishing clock time from temporality is a particularly influential theme for psychology. As human-science researchers and clinicians we are keenly aware that lived time differs meaningfully from measured time (see Slife, 1993).

Heidegger extended his analysis of finitude and temporality to include our being inevitably historical—caught in historical times and moments. Our language and understandings are historically formed, hence we cannot arrive at an absolute or final interpretation of Being. However, *Dasein* is openness to future as well as to past. This basic human way of being (toward the future) has been ignored both by Husserl's eidetic phenomenology and by contemporary

mainstream psychology. In addressing Being beyond consciousness, Heidegger established a hermeneutics ("interpretation") of existence; his philosophical hermeneutics also addressed the assumptions of social sciences' methods.

While Heidegger focused on interpretation of Being, other philosophers with similar interests have focused on particular topics such as language (e.g., Gadamer) and symbolism in psychoanalysis and religious experience (Ricoeur). Psychologists have employed the hermeneutic philosophical tradition (see Hoy, 1978) both to examine individuals' ways of taking up their lives, and to research phenomena—to explore a particular experience and its lived-world ground across instances. The experiences as reported by individuals are referred to as phenomenal instances, instances of unexamined living of a phenomenon (such as being anxious, or bored, or whatever). A phenomenological comprehension is yielded by (hermeneutically) examining instances, asking what becomes evident about how phenomenal appearances reveal and instantiate general human foundations of meaning-making, such as caring and temporality, in the case of a particular phenomenon. See Packer and Addison (1989) for a broader range of hermeneutic psychological studies, where "hermeneutic" refers to the interpretive (meaning-making) character of the research, and where researchers acknowledge perspective, ambiguity, and the always unfinished aspect of understanding. That understanding begins in the life world, where the researcher reflects on possible understandings of the subject matter, then has a dialog with his or her own and others' prior understandings, and circles back to the life-world phenomenon to deepen and revise the earlier understanding. Hermeneutic interpretation does not explain the phenomenon in terms of external or underlying, or any other, variables; it remains with what the researcher is present to in the life world. This interpretation is *not* a translation into established systems of knowledge.

Readings in regard to Husserl, Heidegger, and other phenomenological philosophers include: Edie (1987), Kockelmans (1985), Natanson (1966), and Spiegelberg (1965). Also see *The encyclopedia of phenomenology* (Embree et al., 1997).

### 1.15.3 EXISTENTIALISM

Jean-Paul Sartre (1905–1980), probably the name North Americans associate most readily with existential philosophy, wrote in close dialog with the works of Husserl and Heidegger. In his own way, he appropriated consciousness as a constituting, construing character of being

human. He took up the notions of humans being temporal and situated—always in relation. His *Being and nothingness* (1943/1956) addresses consciousness, Being, and their relation. For Sartre, consciousness is forever striving either to be at one with Being or to become a concrete something. Sartre's famous sentence, "Existence precedes essence" refers to this necessity of constructing one's meanings, because they are not given in our merely existing. Hence also we are "condemned to freedom" to perpetually choose our meanings, especially through our actions.

In much of his work, Sartre emphasized the emptiness of our future being a "not yet" and of our past being a "no longer." For him, the basic character of consciousness is a perpetual dialectic between Being and Nothingness. Sartre certainly is not a philosopher of joy! Not all existential writers stress the anguish of dealing with the possibility of the void, but all do, in one way or another, emphasize the bad faith of not acknowledging both our situatedness and the availability of at least some degree of choice. Most existentially oriented philosophers and psychologists are sympathetic with Sartre's amplification of Heidegger's concerns about our falling into a technological attitude, characterizing others as "they," and living as though we too are objects—already complete, determinate. Sartre wrote primarily of such failures in our efforts to be open to our own and others' subjectivity, freedom, humanity. Others, especially Martin Buber in his *I and thou* (1958), have emphasized that it is through recognizing another person's humanity that we discover our own. Our social context renders us radically intersubjective. Authentic community is possible only through the efforts to affirm our own and others' openness to perspective and possibility.

This standpoint was at odds with psychoanalysis, of which Sartre was a serious reader and critic. He developed an alternative existential psychoanalysis, not as a system of treatment but as a study of motivation, conflict, and of what others identified as neurosis. He deciphered the dynamics of behavior in terms of a person's earlier fundamental choices and of his or her continuing existential projects. He published compelling existential analyses of Genet, Flaubert, and Baudelaire.

Sartre's impact on psychiatry and clinical psychology, as well as on the educated public, has been significant. Existential-phenomenological clinicians have emphasized that often what we diagnose as pathological depression is the state of a person struggling with choices that he or she sees as determining who he or she will be. In seeming paradox, it can be liberating to acknowledge that one is struggling with, "con-

demned to," an existential dilemma, rather than with "having a depression." (As will be seen in the following brief discussion of Merleau-Ponty's work, the body's chemistry can be seen as participating in the depressed state, rather than as originating it.) Even a person caught in a brain-damaged body, or living out a genetic predisposition to schizophrenia, nevertheless still participates in forming the meanings of his or her circumstance, in making continuing choices, and in shaping his or her brain-damaged or schizophrenic life.

Sartre's (1948) analysis of emotion as a "bad faith" attempt to magically escape from the meaning of one's situation has been useful in ordinary life and in clinical practice. The author's own empirical phenomenological research on becoming angry, for example, echoes Sartre in finding that the angry person's seemingly powerful protest against whatever is blocking his or her way is a self-deceptive effort not to attend to a sense of being made helpless or of feeling demeaned (Fischer, 1998).

Maurice Merleau-Ponty (1908–1961) was a contemporary of Sartre's, but from his position in psychology at the Sorbonne he addressed different issues, in particular those of then contemporary psychology: perceptual theory, behaviorism, and neuropsychology. In *The phenomenology of perception* (1945/1962), Merleau-Ponty bypassed the old mind-body split by describing persons as being embodied consciousness. We are our bodies even as we transcend their physicality. Our bodily being is an opening onto things; our body and things imply each other, for example, as one reaches for a cup of coffee. Moreover, we perceive only against horizons, against physical settings and biographical contexts. As horizons shift in accordance with our movement and our efforts toward clarity, perception too shifts. In this sense what we know through perception is always ambiguous, never once-and-for-all; likewise knowledge. This state of affairs is not to be lamented, but rather to be acknowledged against claims of fixed meanings and of absolute knowledge.

In the above work, and in his *The structure of behavior* (1942/1963), Merleau-Ponty described three interpenetrating orders of being. As bodily beings, we are part of the *physical order*, just like rocks, and other objects that "obey the laws of nature." We also participate in the *biological order*, just like other organic, animate beings. Finally, we participate in the *human order*, which is grounded in and limited by the other orders but also surpasses them. The human order affects the others: a strained life can occasion a heart attack or clinical depression. Reciprocally, meditation or a human-made pill can alleviate

biological strain. By now, as we enter the twenty-first century, these ideas do not seem the least bit controversial, but mainstream psychology has not adopted a philosophy of science that can accommodate such observations.

Let me mention a few other major existential authors before moving on to address existential psychiatry and psychology. Victor Frankl's (1905–1997) short, readable *Man's search for meaning: From death camp to existentialism* (1946/1962) has been widely read. Its compelling account of the psychiatrist's survival in a Nazi concentration camp gave credence to the importance of actively creating personal meaning. Rollo May, as first editor of *Existence: A new dimension in psychiatry and psychology* (1958), although writing for a professional audience, opened the way for English-speaking readers to explore what was happening in continental philosophy and psychiatry. Although in later years, May wrote from a more humanistic concern to wider audiences, the introduction he wrote for this book served as a solid introduction to existentialism and phenomenology for many North Americans. Adrian van Kaam's (1966) *Existential foundations of psychology* is a classic text. In more recent times, Irving Yalom (1980) has written a widely read version of *Existential psychotherapy* that presents as its guiding existential themes the necessity of dealing with death, freedom, isolation, and meaninglessness. The author's own, extended, version of existential themes includes finitude (limitation), choice and meaning, intersubjectivity, and lived body (see Leder, 1990, in addition to Merleau-Ponty, in regard to the latter; see Fischer, 1991, for an overview of phenomenological–existential psychotherapy).

#### 1.15.4 PHENOMENOLOGICAL–EXISTENTIAL PSYCHIATRY

From the 1920s to the 1960s, while North American psychiatry imported psychoanalytic theory and method, and North American psychology developed behaviorism and diagnostic assessment instruments, a significant segment of European psychiatry created an approach that addressed the work of both Freud and Heidegger (see Spiegelberg's (1972) *Phenomenology in psychology and psychiatry: A historical introduction*). The two major figures were Swiss.

Ludwig Binswanger (1881–1966) and Medard Boss (1903–1990) both described their work as *daseins-analysis* in contrast to psychoanalysis. In both cases Heidegger's *Dasein*—humans' always being in relation—was taken up at an existential rather than at an ontological level. Binswanger later wrote of his productive error in applying Heidegger's notions directly to

actual beings rather than understanding his characterizations as being about the nature of human Being. It was indeed productive. Binswanger's early training was with Bleuler and Jung at a clinic in Zurich. He opted out of an academic appointment, and instead served as director of a Swiss sanatorium (Bellevue), where over the years he hosted such foundational scholars as Husserl, Heidegger, Freud, Pfander, Scheler, Cassirer, and Buber.

Binswanger remained steadfast in his concern that psychiatry should be properly, rigorously, scientific; that is, it should be open to person-in-world rather than arbitrarily narrow its focus to presumed internal states and historical causes. His applied phenomenological method included “bracketing,” putting aside theory and philosophy while allowing more and more of a person's being-in-the-world to become apparent through a respectful, caring presence to that person. He collated his findings across instances of those ways of being-in-the-world we call schizophrenia, mania, and melancholia. Perhaps his best known individual analyses are “The Case of Ellen West,” found in May *et al.*'s (1958) *Existence*, and “The Case of Lola Voss,” found in Needleman's (1963) edited volume, *Being-in-the-world*. Binswanger never regarded his efforts as settled accomplishments nor even as building toward a complete system, but rather as explorations beyond the artificial constraints of a mechanistic psychiatry. Many of his adaptations of Heidegger's notions, however, have provided readers with an alternative access to ordinary living as well as to restricted or disordered existence.

Binswanger emphasized the we-relationship, a we-hood, between the clinician and the other person, and he studied not “personality” but rather the patient's worlds—his or her multiple relationships. Broadly, those worlds may be addressed in terms of an *umwelt*—one's relation to his or her nonpersonal environment, an *eigenwelt*—one's private world or relations with self, and a *mitwelt*—one's social relations. Always he attended to temporal and spatial unfoldings of personal topographies, such as Ellen West's ethereal, tomb, and action worlds. Binswanger described various “failures of *Dasein*,” notably those of losing one's way, finding one's meanings mixed up, and settling for a life of mannerisms.

Binswanger did not attempt to develop a system of therapy, but he did agree that his *Daseinsanalyse* carried implications for helping persons whose ways of being in the world had become problematic. He advised allowing the patient to explore how he or she had lost his or her way, and he encouraged the patient's re-exploring our shared world, both in and beyond

therapy sessions. He regarded therapy as an interpersonal encounter, and transference as a present encounter between the participants. He regarded dreams as reflecting one's relations, that is, one's lived worlds, and as indicating openings to future choices.

Medard Boss's work is more systematic and more widely published in English than that of Binswanger (see Boss, 1949, 1958, 1963; Moss, 1978). He respectfully bridged Freud's psychoanalytic method into phenomenological psychiatry. He received his medical education at the University of Zurich, worked with Bleuler, received Freudian psychoanalytic training, and continued his psychoanalytic studies in London and Berlin with Ernest Jones, Horney, and Fenichel. After a long collaboration with Jung, Boss ultimately found himself dissatisfied with the deterministic assumptions of both Freud's and Jung's theories. During World War II Boss studied Binswanger's work, delved into Heidegger's writing, and then consulted with Heidegger directly. Boss held seminars in Zurich at which Heidegger spoke to psychiatrists and other physicians.

Boss noted common references in Freud and Heidegger to openness and freedom. He regarded his own phenomenologically inspired *Daseinsanalysis* as remaining truer to Freud's insights than was Freud himself when Freud converted these insights into the language of the prevailing natural sciences. However, Boss found psychoanalytic *technique* to be highly compatible with Heidegger's writings. For example, following the rule of free association returns the patient to an openness to experience and meanings, through which he or she loosens self-imposed constrictions.

Free association and dream analysis uncover not just the past, but previously resisted futures, which now become newly possible in the context of an authentic, caring relationship. Freud's injunction to maintain "evenly hovering attention" is affirmed, but is accompanied by an injunction against translating the patient's reports into reductive psychoanalytic concepts. Reductive interpretations too often transfer the patient out of his or her restricted understandings into the restrictive understandings of the therapist.

Like Binswanger, Boss consistently attends to the patient's matrices of meaning, or lived worlds, as the place where the patient can experience invitations for new possibilities. He adopted Heidegger's distinction between "intervening care" and "anticipatory care," that is, a distinction between intervening by making decisions, providing interpretations, giving advice, or administering medical treatment, and intervening by alerting the patient to his or her

emerging possibilities. For example, a clinician might inquire, "So might this dream be an instance of your already relating to your family as possibly becoming accepting of you?" The clinician also guides the patient by noting resistance to his or own desires. Note that the goals are other than symptom removal or conflict resolution.

Consonantly, Boss distinguishes between neurotic guilt which originates in childhood transgressions and existential guilt which is a failure to remain open to one's possibilities. Both can be terribly constraining, as one avoids looking at continuing choice, lives in a past, and dreads the future. Remaining open, as Heidegger wrote, is not an achievement, but rather is a feature of being human; we are less fully human to the extent that we turn away from possibility amidst the conditions in which we find ourselves. Both attempting to override the limits of our situations and settling for closed objectifications of self or others are inauthentic. Patients are not so much encouraged to form relationships with others or to self-disclose, but rather are encouraged to allow fundamental "world openness" to occur. Boss replaced the psychoanalytic "why?" (does the patient behave in self-defeating ways, recapitulate earlier trauma, etc.) with an existential "why not?" (take your longings seriously, move on from stultifying stances, relate to others more intimately).

The above understandings contrast markedly with psychoanalytic notions of conflicts occurring internally. Rather than seeking internal resolution, Boss encourages revised and opened relationships (with self, other people, things, places, the past and future). Boss regards "acting out" not as resistance but rather as trying out possibilities in a new setting. Likewise, he regards "transference" as the patient's bringing old ways and old perceptions to the relationship going on in therapy now. For a clinician to deny his or her additional part in the therapy encounter is inauthentic and contrary to the project of the client trying out revised ways of relating. Boss, regards *Daseins analysis* as especially demanding of the clinician, who makes use of his or her experience with the patient to encourage greater openness. He regards therapy as a genuine encounter in which the clinician is relatively selfless and restrained and especially respectful of the patient's individuality.

There are, of course, many other contributors from this period in which Europeans sought to develop psychological approaches that respected humans' relational meaning-making. Among these are Johannes Buytendijk, Henri Ey, Karl Jaspers, Eugen Minkowski, Paul Schilder, Erwin Straus, J. H. Van den Berg, and Victor von

Gebsattel. Their work has been of interest to like-minded psychiatrists and psychologists around the world. However, historically there has been little communication between these authors and mainstream psychiatry and psychology, although the former group generally has been traditionally trained and makes use of a range of theory and data. Indeed, my characterization of my own orientation is probably accurate for most phenomenological–existential practitioners: our philosophical orientation is phenomenological–existential; we make pluralistic use of developmental theories (such as those of Erikson, Sullivan, Kohut, etc.); we integrate research studies into our ongoing understandings and questions; and within this frame we make eclectic use of a range of techniques. Unlike other major approaches, no basic therapeutic method is identified with phenomenological–existential clinical practice, as the case is for psychoanalysis, behaviorism, and cognitive psychology. Several psychotherapists have developed particular techniques, useful within many frameworks; examples are Frankl's (1946/1962) paradoxical intentionism and Bugental's (1965) "What's stopping you?"

In closing this section, I offer several corrections of common misunderstandings. As reflected in the preceding sections, existential philosophy and practice do not posit unfettered free will. Nor do they emphasize dread and anguish. A second point: Europe is no longer a fountainhead of phenomenological–existential psychology and psychiatry. North American behaviorism and research design were quickly imported by academicians in Europe, Great Britain, and Scandinavia. Now, interest in human science alternatives for psychology is developing in all continents just as it is in North America.

### 1.15.5 HUMANISTIC PSYCHOLOGY

Humanistic psychology emerged as a full-blown professional movement in the 1960s. The California-centered countercultural "happening," which protested society's suppressions, is similarly named and is sometimes conflated with a reflectively planned movement within psychology (cf. Smith, 1990). The happening, in which assorted citizens, professionals, and many psychologists participated, also stressed the importance of aspects of life neglected by education and the social sciences: joy, creativity, love, self-affirmation, and spontaneous expression of affect and belief. Overlap between the popular and professional movements did occur. American Psychological Association (APA) annual conferences in those days were

attended by sandal-footed, knap-sacked, openly breast-feeding members of the Association of Humanistic Psychology (AHP), whose meeting overlapped with the APA conference. The establishment types were dismayed as some members of the other organization interrupted symposium speakers from the audience with proclamations such as, "I just have to tell you that I love your soul!" One psychologist who earlier had published in staid journals propounded nude group therapy in swimming pools.

Unfortunately, for those psychologists who have not kept up with the literature of humanistic psychology, those earlier dramatic times still color the movement. To the contrary, the AHP's journal, *The Journal of Humanistic Psychology*, has consistently been a major source for well-considered work that explores and supports central human phenomena that are still underaddressed by mainstream education and psychology: our spiritual, creative, spontaneous, caring, transcendent, and transpersonal moments (see Bynum, 1994, in regard to transpersonal psychology). Even while acknowledging the painful and darker sides of existence, the AHP continues to champion human potential for positivity and to promote responsibility for self and to others. Other positive legacies of the mixed beginnings of humanistic psychology have been its contributions to the growth of self-help organizations, and to education and psychology at least nodding to notions of the "whole person"—the experiencing, thinking, valuing, emotional, bodily, spiritual, choosing, interpersonal, behaving person.

Indeed, dissatisfactions with the 1950s partitive, deductive, and reductive experimental research model, with sterile and deterministic behaviorism, and with pathology-oriented psychoanalytic theory, led Abraham Maslow to form a mailing group of kindred professionals. He referred to their efforts as the "third force" in psychology's evolution, following psychoanalysis and behaviorism. Their exchange of writings eventually grew into the *Journal of Humanistic Psychology* (founded in 1961). Among the first board of editors for the journal were Andras Angyal, Charlotte Buhler, Kurt Goldstein, Dorothy Lee, Rollo May, Lewis Mumford, and David Riesman. In 1963 the founding meeting of the AHP took place in Philadelphia, where nearly 100 like-minded professionals confirmed the importance of inserting the place of values into psychology's subject matter and activities. Gordon Allport, Jacques Barzun, George Kelly, Clark Moustakas, Gardner Murphy, Henry Murray, and Carl Rogers were among nationally known figures

who attended the Old Saybrook (CT) follow-up conference in 1964. International meetings followed. The first psychology departments dedicated to a humanistic approach were Sonoma State College (CA) in 1966, West Georgia College in 1969, and the Humanistic Psychology Institute, HPI (San Francisco) in 1970. The HPI has since become Saybrook Institute (see deCarvalho, 1994, for an organizational history of this period).

In the 1950s and 1960s, as psychology moved increasingly out of academia and into practice, Carl Rogers' *Client-centered counseling* (1951) and *On being a person* (1961) were welcomed. He emphasized persons' capacity for growth through self-understanding, enhanced through the empathic, authentic presence of the counselor. He referred to "counseling" rather than to psychotherapy or analysis in large part because those terms belonged to the legally protected domain of medicine. Rogers' work was in striking contrast with psychiatry's emphasis on psychoanalytic theory and psychopathology, and with psychology's emphasis on learning theory and behavior modification. In academia, determinism dominated psychology, which identified itself as a science that could "predict and control" behavior. Maslow's *Toward a psychology of being* (1962) and his *I-thou knowledge in a psychology of science* (1966) were welcomed as alternative frameworks for thinking about ourselves and our clients, see also Bugental (1963).

In 1971, Division 32 (Humanistic Psychology) of the APA was established, largely through the efforts of AHP members. Its purpose was to bring the concepts, theories, and philosophy of humanistic psychology to the research, education, and professional applications of scientific psychology. Among its nationally known first board members were David Bakan, Leonard Blank, Albert Ellis, Carman Harari, James Klee, and Everett Shostrom. Among the presidents of the APA who have also been leaders within humanistic psychology are Carl Rogers, Abraham Maslow, and Brewster Smith. The Division's journal, *The Humanistic Psychologist*, publishes articles on theoretical and philosophical issues, and on methodological advances in human science research. Many members are also actively involved in the APA's Division of Theoretical and Philosophical Psychology.

Accounts of the founders of this movement, their writings, and the issues they dealt with can be found in deCarvalho (1991b), Royce and Mos (1981), and Welch, Tate, and Richards (1978). Division 32's contribution to APA's centennial publications on the divisions' histories and contributions may be found in Wertz (1994).

This brief overview is concluded with a backward- and a forward-looking note. By now, the humanism of earlier centuries has been a touch point for humanistic psychology, but in fact it was not explicitly raised as a source of inspiration in the founding years of the latter movement. The phrase "humanistic psychology" was adopted for the AHP's journal only after rejection of options such as "person psychology," "self psychology," and "orthopsychology" (Greening, 1985; Sutich, 1962); Gordon Allport had first used the term with its present meaning in 1930 (deCarvalho, 1991a). From the Renaissance onward, "humanism" has referred to humans' unique values, creativity, and reason, which exceed both Church doctrine and our material nature (see Bullock, 1985). Humanistic psychology has helped to put psychology in touch with both historical humanism and contemporary arts. Humanistically informed psychologists continue to remind us both of the place of values in our personal and work worlds, and of the importance of examining the assumptions and implications of our scientific practices.

#### 1.15.6 HUMAN SCIENCE PSYCHOLOGY

Human science psychology is an approach to psychology's subject matter. It is not a philosophy or a theory. The approach is intended to respect humans as a subject matter that differs from nonhuman material. A thorough human science approach would encourage study of, and work with, humans' physical, biological, and meaning-making character all at once. Many clinicians attempt to practice in just that way, but when they are not familiar with an explicit foundational framework, their efforts are not as consistent as they could be.

Many researchers could work within a human science frame while continuing to pursue their psychological studies through experimental and other statistical designs, which are efficient for dealing with large samples and with the biological (and chemical, neurological, etc.) aspects of being human. However, these studies would take into account that variables do not explain behavior or experience, but rather are superimposed grids for organizing the orderliness of psychological matters. An analogy is the field of economics, where explanations and predictions are made in terms of unemployment rates, gross national product, and so on, although the human order underlies those created and imposed measures. Ultimately the measures reflect the actions of individuals living within various circumstances and working, enjoying, competing, making decisions, spending, investing,

striving, risking, and so on. In both psychology and economics, the imposed grids are definitely useful, but they are most useful when we take into account that we made them up, and that we understand and serve people best when we return from our constructions to reflect on individuals going about their lives. These reflections allow us to revise our constructions.

At this point human science psychology is a critique and a call. It is a critique of mainstream assumptions and practices for their being forgetful of the fuller human context, and it is a call to develop a thorough alternative. Adopting a human science framework for psychology's ongoing work would eventually obviate the need for the term. Of course by then other critiques would emerge, and psychology would accordingly advance in new directions. Positivism similarly was adopted as a corrective to nineteenth and early twentieth century arm-chair philosophizing, and much of the achievements of North American psychology are the product of that adoption. By now newer generations of psychologists ask "What's positivism?," not because it is no longer around, but because it is not being taught as such. Even though many theorists say that positivism served its purpose, and now has few strict adherents, in the absence of widespread alternative views, implicit positivism determines the research designs that are supported by grants, publications, and so on. Positivism has become the invisible approach of mainstream academic psychology. Because psychology no longer has to develop and prove itself as a science, philosophy of science courses are rarely taught. Slife and Williams (1997) have called for specialty training in philosophical/theoretical psychology so that departments will have such expertise available and, once again even within experimental programs, will engage in discussions of assumptions and their implications.

Toward that end, let me briefly rehash the historically-based approach of a human science psychology, and some contemporary trends with which it is joined. William Dilthey (1833–1911) urged that psychology become a rigorous and systematic science, a model for all the *Geisteswissenschaften* (the disciplines of mind, spirituality, and human affairs). He argued against the new discipline modeling itself on the highly successful disciplines of time, mathematics, and the natural sciences (see Dilthey, 1894/1977; Hodges, 1944). Nevertheless, even though Wilhelm Wundt also wrote a social psychology that was closer to Dilthey's proposed human science, and William James wrote extensively on consciousness, it was their fledgling laboratories in Germany and the United States, respectively, that were taken

up by the new discipline. Their philosophical essays and books were ignored in favor of hands-on, technically-oriented practice. Nevertheless, a sampling of books and essays in developmental, clinical, and social psychology through the 1940s shows that most authors were more holistic, and more historically and philosophically minded, than is the case today.

Although North American psychology has always housed a broad mix of theorists and practitioners, by the 1950s "scientific" psychology was equated with experimental psychology. By the 1960s, many of us who were not satisfied with that situation discovered the translations of existential phenomenology that were finding their way from Europe. Within this country, many critiques and calls for returning persons as such to our studies were published, for example, those by Gordon Allport, David Bakan, Roger Barker, Joseph Lyons, Robert McLeod, and Nevitt Sanford. In 1970 Amedeo Giorgi published *Psychology as a human science: A phenomenologically based approach*. Trained in experimental psychology, he had encountered its limitations as a natural science, and turned to Dilthey and phenomenological writers for foundations appropriate to studying humans as humans. His book has been a touch point for diverse readers who have looked for support and inspiration for their own kindred reflections.

Institutionally, Duquesne University's Psychology Department, shaped in the 1960s by Adrian van Kaam and Giorgi, has been a center for the systematic development of psychology as a human science. Its work, however, has been foundational and corrective and has not yet embodied a full human science research program, which would include our cultural, biological, etc., dimensions. Nevertheless, at Duquesne alone, nearly 200 empirical phenomenological research dissertations have been completed; gradually substantive content area comprehensions are taking shape. Moreover, as qualitative research methods are increasingly practiced across disciplines and continents, their practitioners are increasingly identifying themselves with a human science tradition.

Saybrook Institute, which also offers a doctorate, stressing humanistic psychology, offers substantive work in a human science tradition. The masters degree psychology programs at West Georgia College and Seattle University are dedicated to humanistic/human science psychology. Other institutions include clusters of colleagues pursuing this approach, and of course there are hundreds of individual faculty members doing likewise along with thousands of nonacademics. Many individuals do not use the "human science" title, and many are working within overlapping interest areas. A

small but representative sampling would include constructivist psychotherapists, who have advanced George Kelley's (1955) analysis of how we construct our worlds and can be encouraged to construct them differently; narrative therapists, who help clients to see how they have storied their lives and might co-author them differently; and action researchers, who not only acknowledge but plan and track their influence on their field subjects. Related areas of scholarship include social constructionism, which explores how science as well as society and individuals are inevitably shaped by our ideas and interactions, and which finds that we have no access to truth aside from these constructions; linguistic studies, which explore the inherent role of language in our sense of reality and possibility for action; and feminist studies, which in a variety of ways highlight the culturally situated social construction of gendered possibility. (Leading sources on social constructionism include Berger & Luckmann, 1967, and Gergen, 1985, 1994; sources on personal construct therapy include Epting, 1984 and Leitner, 1985; linguistic and discourse studies include Barnard, 1998, and Gavey, 1997; feminist works include Harding, 1987; and Hawkenworth, 1989.)

Many other approaches and practices that nowadays carry one of the "post" labels also dovetail with human science psychology. "Post-enlightenment" refers to orientations that emerged in response to disenchantment with the enlightenment era's assumption that God had created a mathematically ordered world which mortals could uncover through empiricism and logic. "Postmodern" refers similarly to contemporary movements across the arts and sciences that have ceased to look for absolute Truth. Reality is always known only from perspectives, which are necessarily local (not universal). Contrary to some protests, this position is not one of "anything goes" relativism, but rather one that respects subgroup realities, and calls for accountability through specifying the motives and history through which particular understandings emerge (see Aanstoos, 1990). Finally, "postpositivism" refers to having superseded the notions that science requires that its knowledge be based on sense data that have been processed through mathematics based on Aristotelian logic.

To review, human science psychology differentiates itself from positivistic traditions by drawing on broad hermeneutic traditions and/or phenomenological descriptions of persons as always being in relation to the world, always temporally on the way from and toward, and on existential extensions of those descriptions of persons as always being in relation to lived body

and to the social world, and as inevitably participating in making meaning and choices no matter how constrained. The mind-matter split is undone. The moral dimension of human matters is more clearly proper to psychology. The old goals of predict and control become those of describe, understand, and influence. Data developed using natural science methods, including neurological and biochemical data, are comprehended within this frame. To the extent that a body participates in the human order, its biological order is part of that structure; even sciences of the body are human constructions. Description of human perception, experience, and action is narrative rather than reductive, and it respects ambiguity as sometimes inherent to the subject matter rather than as a deficient account. Accounts are structural—retaining the mutuality of differentiated aspects of whatever is described. Understanding and influencing require respecting persons as choosing in accordance with both contingencies and personal meanings. Validity and objectivity become matters of specifying means and perspectives, and of sharing data, comprehensions, and practical outcomes, toward consensual agreement. Progress evolves as new perspectives are continually brought to bear.

Below are concrete illustrations of approaches to research and to psychological assessment that flow from a human science orientation. Representative psychotherapeutic practices have been described in Section 1.15.4 on existential psychiatry and psychology. I have drawn on work with which I have been intimately connected, and which has been developed within the Duquesne community for over 30 years, in order to provide examples of what the foregoing discussion might come down to. A wide range of nonphenomenological substantive work can be characterized as human scientific. Human science psychology will develop and contribute to the extent that variations and innovations are undertaken and discussed.

Further examples of human science, broadly conceived, can be found in the *Duquesne Studies* series edited by Giorgi and colleagues (1971, 1975, 1979, 1983) and in the series edited by Valle and colleagues (1978, 1989, 1997).

### 1.15.7 EMPIRICAL PHENOMENOLOGICAL RESEARCH METHOD

The term "empirical" in "empirical phenomenological research" refers to directly observed, pretheoretical experience (one's own or another's) in contrast to data derived from the



use of predetermined categories and through measurement. "Empirical" also refers to the availability to other researchers of data and analytic steps, so that they can check the extent to which they come to similar findings. The basic method, developed and elaborated for over 30 years at Duquesne University, is a systematic means of addressing psychological phenomena in their own terms, that is, in terms of how they were lived. Empirical phenomenological analyses of "being in privacy" and "being impatient" will be reported below. "Phenomenological" here refers both to the more immediately accessible "what it's like" for individuals (to be impatient, etc.) and also to what researchers learn when they ask what else these phenomenal descriptions can reveal about the process of being human in that situation. In the latter regard researchers repeatedly rediscover their subjects as being temporal, always in relation and on the way, restricted and choosing, and so on. At Duquesne we have referred to a cohesive representation of these themes as they appear in, and shape, the phenomenon as its phenomenological structure.

Empirical phenomenological research is existential in that psychologists have departed from a purer phenomenological philosophy, to address people in their necessarily existential situations. "The method," moreover, is a great variety of variations and innovations, as von Eckartsberg (1986) helpfully described in *Life-world experience: Existential-phenomenological research approaches in psychology*. For other examples and discussions of variations of the method, see Aanstoos, 1987; Colaizzi, 1973, 1978; Giorgi, 1985; Richer, 1978; Walsh, 1995; and Wertz, 1983.

Qualitative research in general addresses the experience and action of persons as they engage in particular situations. Findings are in terms of the life world rather than transformations into categories and measurements. Traditional quantitative findings of course provide ground for reflection in concert with qualitative description. Many nonphenomenological qualitative research methods are also appropriate for a human science psychology. These include ethnography, linguistic and conversation analysis, case studies, ethnomethodology, and some forms of grounded theory. Psychology has been slow to become involved with qualitative research methods in that they do not fit our received, rather narrow, notions of science. In contrast, sociology, education, gender studies, human development, nursing, and counseling all have established or are building major qualitative research traditions. About a third of the papers presented at the American Educational Research Association conferences

are qualitative studies. At last count, five nursing journals publish significant qualitative research. About a third of the research presentations at the Society for Psychotherapy Research conferences is qualitative. Well over half of the research papers presented at the Association for Women in Psychology Conferences use qualitative methods.

Many variations on the EP (empirical phenomenological) research procedures described here have been, and could be, undertaken. Guy Kashgarian (1997) employed a variety of linguistic analysis as part of his phenomenological analysis of instances of "being criticized" reported by persons diagnosed as narcissistic personality disorder and those not diagnosed. Martin Packer (e.g., Packer & Scott, 1992) encourages EP analyses based in part on ethnographic data.

A thorough human science research program would accommodate biological, cultural, and psychological dimensions of any studied circumstance. It is to be hoped that before long psychologists will undertake studies of the mutuality of biological and psychological depression, for example. In the meantime, this chapter focuses on Duquesne's EP research method because it offers a crucial approach to understanding human phenomena otherwise missing from psychology's efforts. EP research is the appropriate method when the question is "What is such-and-such a phenomenon?," as in "What is privacy?" or "What is being impatient?" When our questions have to do with absolute amounts and with amounts of change, traditional measurement and experimental methods are appropriate. Unfortunately, historically psychology typically has not asked the What question, and has instead resorted to operational definitions (defining something in terms of measurement criteria, as in hunger = percentage of body weight loss, or of food reduction).

#### 1.15.7.1 Procedures

Long before beginning a formal study, one has noted instances of the phenomenon in one's own life, talked with colleagues and friends about instances in their lives, and noted occurrences in newspapers, novels, and so on. Research and theoretical literatures have been consulted for their accounts. As in all research, formal participants for the study are chosen on the combined grounds of practicality/availability and of being appropriate for the issues and literatures one hopes to address. Because EP analyses are so arduous and time-intensive, a dissertation study might involve only five or six

formal participants. One funded project with multiple researchers included 50 formal subjects (Fischer & Wertz, 1979). Regardless of the number of formal participants, the study's preliminary findings are compared with other instances, both to reconsider one's work, and to make comparisons with regard to differences among participants in different situations.

#### ***1.15.7.1.1 Collecting instances***

Although EP research need not be constrained to analysis of verbal reports, which is the tradition that developed at Duquesne, through faculty members' projects, more than 200 doctoral dissertations, the work of graduates, and the work of others who have based their research on the Duquesne tradition. Requests for descriptions from pilot participants result in a refined formulation of the research request that is presented to the formal participants. An example: "Please recall a situation in which you became angry. Please write out what was going on before that situation, what happened as the situation evolved, and what happened then. Include details that will help us to understand what it was like to be you throughout the situation. Thank you." Participants have agreed in advance to provide a description, and hence have been recalling long before they sit down to write. Some researchers have recorded interviews rather than requesting written accounts; usually those accounts are briefer on the one hand or rambling on the other. Written reports allow participants to organize and elaborate their recollections.

The researcher types any recorded or handwritten accounts, and reads and rereads them before returning to the participant for a follow-up interview, which is taped and later transcribed. Typically, the researcher asks if there are additions to the account that the participant might have thought of since providing the description. Then he or she is given a copy of the transcript, from which the researcher reads aloud any sections that seem to require clarification. Open-ended requests for elaboration are made, in the form of, "Could you tell me more about that?" Only at the end of the interview might the researcher ask direct questions such as "Did that occur on the same day?" or "Would you say that this instance of being impatient is typical for you?"

#### ***1.15.7.1.2 Organizing the data***

The researcher typically recasts each account into chronological order, inserting interview elaborations (in a contrasting font) into the

relevant text. At this point the researcher decides on a system for breaking the text into workable form. Usually this involves numbering "meaning units"—segments of text that seem to be cohesive and that are followed by a shift in the reporter's account. Some researchers number phrases, some entire paragraphs. The point is both to have a way to check that one has included all the text in the analysis, and to later be able to cite sections in every report to illustrate each finding.

#### ***1.15.7.1.3 Data analysis and forms of results***

Some researchers have met regularly as a group, exchanging their own descriptions, or those that they have collected, working together to formulate their findings. Others have met regularly with the providers of accounts, to collaboratively revise their findings in discussion with the providers. Most researchers working on dissertations have worked singly, in consultation with a faculty committee. Of course one can combine these approaches.

Although variously formulated, most of the work at Duquesne includes the following processes. One immerses oneself in the descriptions, "dwelling" in them, as we say, becoming familiar with their internal cohesion and their nuances at a prearticulate level. Of course at the same time, one is noting recurrent themes, and jotting down phrases and metaphors that hint at what seems to be present in all the accounts. The method section of the research report itemizes the formal steps that were conducted and documented, and that a reader may follow along to see whether the reported findings are evident, and whether the procedures seem to have been followed. However, the formal steps do not characterize all that goes on as one becomes more deeply immersed in apprehending and giving expression to the phenomenon under study. For example, while supervising a student psychotherapist, I realize that a phrase she has just uttered is perfectly suited to characterize an aspect of what my research participants have reported. While doing my morning physical exercises, a just-right metaphor seems to come out of nowhere. Upon rereading a transcription for the umpteenth time, I realize this person's wording fits all my participants' instances. While reviewing my notes for a lecture on object relations, I somehow realize that I have been forcing a distinction on my data.

One is not discovering external, independent, features of an object, but rather is making a phenomenon visible and differentiable from related phenomena. The process requires four kinds of rigor: diligence in describing and

following formal procedures, faithful presence to data, disciplined and systematic reflection on what is appearing in instances of the topic, and finally, creativity in expressing—representing—making visible—the unity and differentiations of the phenomenon. We struggle with the limitations and possibilities posed by language and by our being historically and culturally situated.

The formal steps usually include uncluttering each participant's report by dropping out information not essential to that person's experience for it to be an example of the particular phenomenon. For example, in Ms. Smith's account of being criminally victimized, we might drop out the names of her children and the color of her handbag. We might condense references to having done three loads of washing, made all the beds, and vacuumed to "after a morning of heavy housework . . ." The resulting condensation might be referred to as an individual or situated description, and can serve as one form of the study's results.

Next, each condensation is analyzed by asking what is being said about where this person was going in his or her life, how he or she was in relation to self, others, and the world, and how he or she was living a past and the future. The researcher might then attempt to represent this understanding by writing a narrative version that stays close to the participant's own words but draws out experienced meanings more explicitly and in relation to the research question (e.g., "What is the experience of being criminally victimized?"). Here, the researcher might say, in part, "Having completed her planned morning of heavy housework, Ms Smith gathered her children into the family car, anticipating a successful shopping trip." This level of analysis can be presented to the participant as a check on having rendered her explicit and unarticulated experience faithfully. This version of the findings again is both one form of findings and a step toward further analysis. This step, which might be a half-page to two or three pages in length, is referred to as an individual phenomenological description or structure. By "structure" we mean that the descriptive account is cohesive in its explication of differentiable but essential aspects, and that none is made more basic than any other; each aspect implies the others.

Finally, the researcher examines each individual phenomenological structure, identifies the themes that are shared by all, and formulates a single phenomenological structure that expresses what was necessary and essential for all the accounts to be instances of the phenomenon under study. This level of finding is of course more abstract than the preceding ones. The researcher's implicit and explicit questions to the data are evident in the findings. For

example, my own societal and clinical concerns become evident to me at some point as I reflect further on my findings. Levels of abstraction vary among research authors, as does style of representation. Some writers put greater stress on evoking readers' lived sense of a phenomenon, and others place greater emphasis on highlighting themes that lend themselves to dialog with philosophy. In any case, the findings must be demonstrable in every instance of formal data, and in all instances encountered outside of the study. No settling for probability levels here! Quotations from the participants' original accounts document and illustrate the findings.

### 1.15.7.2 Clarifications

When undertaking a study, researchers first put aside theoretical positions and what they already know about their biases, and then specify what they know about their guiding interests (e.g., making a rape victim's experience available to law enforcement, medical, and court personnel). However, throughout these steps and in the findings, the researcher repeatedly discovers his or her preconceptions (for example, that being impatient is a negative stance, or that privacy is privacy-from). He or she makes formal note of the discovery for later discussion, and in the meantime tries to hold the assumptions in abeyance. We recognize that we cannot thoroughly separate ourselves from our historical, cultural, biographical contexts nor from motives for undertaking a study, but we hold ourselves accountable for bracketing all that we can, and specifying all that we identify about the perspectives from which we have viewed our data.

Researchers follow different steps and different exercises within those steps depending on the subject matter, their own route into phenomenology, and so on. In one way or another, all follow what can be called a "dwelling and hermeneutic presence" to their data. Analysis does not occur in a finished step by finished step fashion. Rather, the researcher repeatedly immerses him- or herself in the original descriptions and in the forms of findings developed so far. Sometimes this delving is a roaming around, noticing nuances. Other times it is a reading of one account with another account in mind, allowing synergistic meanings to emerge. When a new insight or understanding emerges, the researcher returns to the earlier understandings, and reworks them in the light of the newer perspective. This systematic circling back to revise one's interpretation, the hermeneutic movement, is difficult to represent in a procedures section.

### 1.15.7.3 Example: Being in Privacy

The following structure of being in privacy has been useful in distinguishing privacy from secrecy. It also makes a case for the importance of solitude, even in public settings, for the development of wonder, reflection, and so on.

Instances from which the analysis was developed included being immersed in a novel until interrupted by telephone solicitor, considering a painting in an art gallery until a tour group came through, working on a math problem until the teacher looked over one's shoulder, enjoying a flower garden until chimes reminded one of obligations.

These characterizations are paraphrased from Fischer (1971) (pp. 153–154):

As we look back, it seems that in privacy one's sense of self falls away, and one flows with whatever is being attended to. One seems nearly, but not quite, merged with that something. This attending is recalled as including an aura of familiarity, at-homeness, of things fitting even when they do so unpleasantly or newly. In privacy one's relation to subject matter may occur through reflection, action, imagination, whatever. The relation includes relative openness to and wonder at the object's possibilities, facets, realities, even when the person is in a problem-solving mode or is reflecting on an unfortunate aspect of one's life.

One's attention eventually turns, seemingly in its own course, to other matters, usually things to be done. However, when privacy is disrupted prematurely, attention is torn from its prior focus, and shifts repeatedly among intruder, self as caught by the intrusion, the peripheral world, and the lost focus. One now contends with contingencies—time, space, demands. Affect varies with desire and felt capacity to deal with the intrusion, and to recapture the disrupted presence to the previously evolving subject.

### 1.15.7.4 Example: Being Impatient

The following Condensed General Structure is taken from Donna Coufal's (1997) dissertation. Among other themes, the dissertation discusses the surprising discovery of a moral dimension, and compares being impatient with phenomenological analyses of being angry, frustrated, and anxious. Instances provided by her participants (Ps) included being stuck in an automobile bank teller line, coping with a client who seemed not to be hearing the counselor, and being lost on a country road while trying to get to a social event.

The awareness of time as protracted but imminently fleeting is central to the experience of being impatient. What was initially taken for granted, that time is sufficient to meet P's goals and that the

future will unfold as P anticipates, is no longer certain. P's sense of time feels distorted. An earlier absence of concern about the future gives way to intensifying affect in the face of waiting. In spite of obstacles in the way, P is poised to move forward, resisting the temptation to become emotional.

As P's focus narrows, the complexities of the situation go unnoticed, particularly as P becomes tired and frustrated. P tries to force the situation to evolve as planned and cannot imagine alternative courses of action. The situation is one that demands something new of P who nevertheless grasps at the familiar in the face of the unknown. P overlooks evidence that would help in navigating the situation more efficiently. In attempting to avoid waiting, P creates further delays and finds him- or herself frustrated or defeated.

In denying limitations of a habitual style, P is confronted with the annihilation of previously unchallenged assumptions about him- or herself. P becomes vigilantly focused on preserving a shred of his or her identity in failing to meet his or her own expectations. P admonishes self and others while waiting. Innocent delays are experienced as moral affronts. P casts about for targets of blame, alternatively believing delays to be purposive on the part of others or reflecting inadequacies in P's own participation.

In spite of the awareness that being impatient worsens the situation, any attempts to relax are defeated by P's rigid alertness. P tries to hurry in order to feel less vulnerable, but remains tense or becomes angry. Although P has restrained him- or herself from becoming emotional, when looking back on the situation, P identifies his or her responses as disproportionate to the demands of the situation.

Having failed, P reflects on the situation and becomes open to its particular meanings and possibilities. Now the present and each small step P takes become more figural, while larger, future oriented goals become horizontal. Nevertheless, P arrives at destination too deflated or exhausted to enjoy any sense of accomplishment except for the modest satisfaction of having modified his or her initial plan.

In contrast, when patient, P experiences openness to unexpected events. Anticipation is tolerable, even pleasurable, and P responds imaginatively when action is not yet possible, appreciating the limitations imposed by the situation, but not assuming a catastrophic outcome.

### 1.15.7.5 Comments

Another researcher or reader may suggest a more evocative, accurate, felicitous, or otherwise preferable word or phrase, may identify additional assumptions on the researcher's part, may point out relations to other phenomena, and so on. That is how bodies of understanding evolve. Note that I have referred throughout to "understandings" rather than to "knowledge" in order to emphasize that what we know, even

though through our natural science or human science methods it is always an interpretation.

What is phenomenological research good for? It provides a means of addressing experience as such. It enhances consideration of the human order of events and how we participate in them. Within such descriptions, we discover choice points; we are freer than our psychology's literature on determinants would lead us to believe. Phenomenological research also helps us to see the relations among phenomena, and how one can turn into another, which again helps to guide our choices. This type of descriptive psychology puts us in touch with the humanities, art, literature, and drama, making them available to our psychological theories.

Adrian van Kaam's dissertation (1958) at the University of Chicago, on the experience of feeling really understood in psychotherapy, was a forerunner of the research method developed at Duquesne, where van Kaam brought together the group who initially began the department. Amedeo Giorgi took the lead in developing conceptual and procedural foundations for existential-phenomenological research. Other faculty members and graduates have contributed to and furthered their development, for example, C. Aanstoos, S. Churchill, P. Collaizzi, C. Fischer, W. Fischer, S. Halling, and P. Richer. Among phenomenologically oriented researchers who developed their research approaches independently of Duquesne are Howard Pollio at the University of Tennessee and Joseph deRivera at Clark University. Further illustrations of EP research and writings on method can be found in the *Duquesne studies in phenomenological psychology* series (Giorgi et al., 1971, 1975, 1979, 1983), in the *Journal of Phenomenological Psychology*, and in the journal *Methods: A Journal for Human Science*.

### 1.15.8 HUMAN SCIENCE PSYCHOLOGICAL ASSESSMENT

The author developed the following approach and practices while teaching psychological assessment within the human science psychology program at Duquesne University. One does not have to adopt the orientation of this chapter to incorporate the practices into assessment. I do think that accommodating this chapter to one's own assumptions about the nature of being human can allow one to engage a client more fully and consistently as a co-assessor, and to participate in developing tailored, viable changes in his or her life. Stephen Finn (1996) and his colleagues at the Center for Therapeutic

Assessment in Austin and Leonard Handler at the University of Tennessee have developed similar approaches from orientations that overlap mine. Finn and others have published research reports on the effectiveness of, and client satisfaction with, assessment collaboration and feedback. Slowly, my textbook (Fischer, 1994), *Individualizing psychological assessment*, originally published in 1985, is increasingly referenced or adopted in graduate courses. It seems to me and my colleagues that our work in collaborative assessment validates the private inclinations of practitioners, whose university training, in contrast, had followed the laboratory tradition—in which one gathers data and draws inferences, without consulting with the subject. Availability of an explicit human science framework encourages efforts to involve clients as co-investigators.

#### 1.15.8.1 Developing and Contextualizing Issues for the Assessment

If an assessment referral has been made by a third party, I ask that person to tell me more concretely what the issues are. Often the referral remains abstract until I ask what decisions might be influenced by the assessment. For example, in one instance, a director of an adolescent program requested an assessment of a youngster's "personality structure." Upon discussion he initially revised his request to be "evaluate Axis II diagnoses," and then after further discussion, to "assess capacity for empathic relations." When asked about what decisions he was faced with in regard to the young woman, he said that an immediate one was whether she could be placed in a therapy group that required mutual support among the members and an ability to introspect. He agreed with the assessor that he also was trying to determine how psychologically developed the client was so as to anticipate her level of involvement with the overall program and to determine whether several months of participation might or might not make a difference. The assessor then asked for information and observations that had been gathered on both sides of the issues. It turned out that the client's family had been unstable in many ways all her life, and that she had been truant from school regularly (and was failing most classes), had a reputation for defending herself from insults through physical retaliation, was suspected of being responsible for items missing from the school lunch room, and was seen as a loner at school. On the other hand, she had earned a "B+" from an English teacher she seemed to like, had taught a younger brother to read, and

had impressed the Program's social worker as being sad rather than as defiant and as having a definite, albeit dry, sense of humor.

Often, this "getting down to earth" process suggests next steps that enable bypassing formal assessment. On the other hand, just a few consultations, such as that above, with referring staff members, results in assessment requests that are already accompanied by concrete events from a client's life and by a clear notion of how the assessment might address the client's situation. The assessor encourages the referring parties to discuss their dilemmas directly with the client, and to ask for experiences that might weigh on one side or the other. Inevitably, staff members discover that clients are more capable of entering these discussions than was assumed. In addition, explaining and discussing issues with clients, without professional jargon, clarifies staff notions while revealing the clients' understandings and concerns. This process is constructive in itself.

When a formal assessment is arranged, whether on referral or from a direct request from the individual, I continue the above procedure. I ask the client for his or her understanding of the referral, and I clarify whatever I know about it. Later, after we have developed ways of working together, I may ask for the above clarifications again. At that point I am sometimes told that a psychiatrist or therapist wants to know whether the client is really crazy, suicidal, or hopeless. Almost needless to say, it is imperative that I have previously obtained the referring party's permission to discuss the presented issues with the client, so that I can be honest, reassuring, and exploratory, as called for.

I ask what the client would like to learn from the assessment, and I ask what the client already understands about his or her, and others', concerns. As we talk, I try to use his or her terms in order to keep us both grounded in the client's life rather than in abstractions. I help the client to "deconstruct" any personality constructs, and to provide examples of the "when-nots" as well as the "whens" of problematic reactions and actions. For example, a woman tells me that she shares her therapist's concern that she might be pushing beyond her "natural ability," that she might not have "enough intelligence" to pursue a promotion to a managerial position. We discussed what the managerial job requirements would be, and agreed that we would look into her styles of approaching problems and would relate our observations to the managerial position.

Before we began testing, we had discovered that Fran has gotten along well with co-workers and subordinates, and has been regarded as

efficient and conscientious. But she has backed away from projects that seemed to require "jumping in and taking the ball" into "unknown territory." A when-not, however, had occurred when she decided to have an addition built on her home. She had written numerous notes and questions in a binder, consulted several contractors, then consulted friends who had built additions, and checked in regularly with the contractor and subcontractors. We agreed that with the freedom to consult she had indeed taken the ball into previously unknown territory.

Fran offered another issue for assessment, namely whether she was a "dependent personality." We de"construct"ed that notion and wound up discussing times that she had and had not deferred to others' judgments when in fact she could have acted on her own. From there we went on to the Minnesota Multiphasic Personality Inventory (MMPI) and Rorschach to see what else we might find in regard to her questions. I reminded Fran that I would summarize our findings after our next meeting, and then would send a copy of my written report to both her and her therapist. The beginning of the assessment process has already been collaborative, individualized, and oriented toward the life world.

#### **1.15.8.2 Using Tests**

Any assessor should be knowledgeable in the realms of test selection, profiles, and norms, and in their relations to theories of, and research on, personality patterns, psychopathology diagnostic systems, styles of coping and thinking, and levels of effectiveness. This expertise allows the assessor to form impressions, be surprised, and form alternative understandings. To be most useful, however, the assessor must also know how to use this expertise to gain access to a client's life. Within a human-science approach, our primary data are life events. As above, after locating referral issues in life situations, we then turn to assessment tools, to theories, and to research to gain access to and to explore those life events. If written assessments end instead with scores, categories, and diagnoses, I would say that they are unfinished, that they have reported our tools rather than findings pertinent to an individual's life. The life world should be our point of return from testing, as well as our point of departure into testing.

Test scores do not reveal anything more basic or explanatory than we could find by following a person around through various contexts and observing his or her reactions and actions. Of course the latter "option" is not efficient or

usually even feasible. At any rate, the assessor's explorations with the client culminate in a description of how the client has contributed to his or her successes and failings, and how the client might recognize alternative routes to revised goals. Our understandings weave a story, a narrative account, in contrast to assessments that explain in terms of "underlying variables."

Examples of using test patterns in discussion, and then of using individual tests interventionally, follow. I had been asked to conduct an assessment of an electrical engineer whose MMPI-2 had been too defensive to allow immediate clearance into restricted areas of a nuclear plant. He and I agreed that he had tried to look as unproblematic as possible in order to obtain clearance, but also that he has always seen himself as "a good citizen," that he does not look very deeply into personal matters, and that a retest would probably result again in what would look defensive to others. I remarked that although he had not flagged in filling out several pages of background forms I had given him, he struck me as being tired. He said no, that it had just been a long day. "Well," I asked, "how about this possibility?: Although you aren't depressed [he nods], this asterisk here [I point to the subtle depression score on a computer print-out] often happens when a person has been kind of down, not as alert or enthusiastic as usual, even though people at work and at home haven't noticed." Mr. Kern looked up at me with surprise, nodded quizzically, but said he did not know much more about it. I then mentioned that on the Rorschach he had been attuned to a lot of things "just happening" [inanimate m], like a leaf that was being blown in the wind, and a lit candle melting. I reported to him that when people are able to see that kind of "just happening," that their lives are often in a holding pattern, that they know where they would like to be going, but it is as though they just have to tread water. To this, Mr. Kern nodded decisively, and when I suggested that most people know what that holding pattern is all about, and that they also find themselves tossing in their sleep about it, he explained to me that his entire work unit had been waiting for eight months to hear whether the corporation was going to close them out of existence. He volunteered that during the same time he and his wife were waiting to see whether they would be able send their son to the college he preferred. Mr. Kern explained then that perhaps he was feeling "worn down" but that he was not "tired," which meant to him beginning to lose hope or not being able to pay attention. Although Mr. Kern had not wanted to examine his current life at all, he found that he had done so in a way that affirmed his previously

nonconscious sense of his circumstances, and that unburdened him a bit.

Individual tests and techniques allow both client and assessor to observe the person going about an activity. Similar past situations become available to the client in ways that talk alone would not have afforded. For example, Ms. Rose had wanted to know if we might discover how it was that her boss gave her low performance ratings for efficiency, although she gets more done than anyone else in the office by the end of the day. As I watched her copy the Bender designs, I wondered if my reaction was similar to that of her boss. Ms. Rose looked at each design for a good while, stretched, then held her pencil lightly with one hand and anchored the paper with just two fingers of the other. She drew parallel lines for the square, then half the circle, then finished the box, then the circle. She continued in her unorthodox manner through the nine designs. She never looked at me, and never looked back at the stimulus card after first observing it. When she had finished the designs, I was astounded to see that they were geometrically precise and neat, and that she had finished in considerably less than the average time. We laughed together as she realized that she similarly has created her own world in the busy office, where others cannot see what she is up to as she does things out of order, stretches, seems oblivious to the clock, and so on; others had not noticed the nicely completed projects among the part projects arranged on her desk. Through our work, Ms. Rose was already finding that she could make sense of her circumstance; she had discovered how she had been contributing to others' perceptions of her. We then role-played doing the Bender again with her making comments that allowed me to know what she was doing and that she was indeed following directions. She remarked later that she was now "liberated" both to be herself and to win the approval of her boss.

### 1.15.8.3 Collaborating and Intervening

Finn has pointed out that this approach to assessment can be undertaken as a therapeutic activity in its own right. Note the therapeutic process in the following examples of intervening into the client's usual course. On the Rorschach, Harriett had hesitatingly reported a fight between two bears who were both injured. On the next card, instead of the usual humans cooperating on ordinary tasks, she reported "warriors from another planet; they make me laugh—they're too stupid to know that they're hermaphrodites." I pointed out a similar sequence with other cards,

and asked her what she already knew about a pattern of running from being afraid or from being in touch with the danger of being injured, and then trying to hide through being silly, voicing put-downs, and so on. Harriett readily provided examples within her family and her personal relationships. We used life instances and the Rorschach instances to help her to recognize landmarks which could indicate that she had entered frightening territory. She could then look for turn-off points in order to change course if she found herself veering into silliness or offensiveness. One landmark she identified was an inclination to convert her fearfully tightened lips into a sneer. We agreed that she might then purposively transform the coming sneer into an expression of determination to stay her course while checking out whether she was indeed in dangerous territory.

On other occasions an assessor might intervene during a client's telling of a Thematic Apperception Test (TAT) story. For example: "Geez, John, is this going to end dismally like all the rest? Can you tell it so the fellow figures out a plan?" This was John's first recognition that he had been co-authoring his own dreary circumstances outside the assessment.

Note that this collaborative, interventional approach affirms the client as an active agent, sometimes self-defeating, but also capable of changing course. It focuses on actual situations, whether during assessment or the client's larger life, rather than on drives, traits, cognitions, determinants, and so on. The client is seen as always in relation to goals, to pasts, to obstacles, to invitations. We characterize the person in terms of his or her construing situations and of being formed through choices in those situations. Although not illustrated in this chapter, assessor and client often agree to disagree, or agree that they cannot spell out what it is they are in touch with. In that process, they become at least peripherally aware of both the necessity for, and the inadequacy of, language. Shared humanity and understandings between the participants move the assessment along, despite personal differences. The client knows he or she has been met by someone attempting to deal authentically with limits and possibilities, even when the assessor comes to conclusions not in the client's favor, such as findings of neurological impairment, or of unsuitability for unsupervised visits with a child.

#### **1.15.8.4 Writing Reports**

In human science assessment reports, "findings" are both clarifications of past life events and identification of personally viable options for the client. So reports are crafted to remind

readers of the client's agency—his or her power to initiate shifts in course. Constructs are eschewed as implying that external factors account for the person's actions. Any necessary jargon is explained in everyday terms. When psychologists cannot write their findings in terms of everyday events, they literally do not know "what in the world" they are talking about. Reports are written in first or third person (Mr. Jones, I, we) rather than referring to "the examiner" or to "this patient." We write with verbs rather than nouns whenever possible, in order to move from abstractions to actual behavior. For example, instead of "John's low self-esteem keeps him from entering the contest," we might write, "Fearing that he would be made fun of, John has not entered the contest." Yes, it does take extra effort to locate and contextualize the actual instances of what has been abstracted into nouns, and it usually takes more writing to describe them than to name constructs. But the worthwhile outcome is understandings of actual events shared by all participants (client, family, teachers therapists, etc.). We also write in the past tense rather than in the present tense, to indicate situatedness and the possibility of change. For example, instead of "Janine sees authority figures as demeaning," we might say, "Janine has seen her bosses and teachers as likely to demean her." We try to own our perceptions rather than attributing all conclusions to test data. For example: "As Ms Gertz glared at me when I asked personal questions, I thought of the MMPI's references to 'nondisclosive' and 'hostile,' which I see as a defensive stance on her part."

Some impressions are only implied or evoked rather than spelled out, not reflecting deficient understanding so much as respect for the ambiguity inherent to the life world. A report that is faithfully descriptive does not attempt false clarity. For example: "Something about the contrast between Mr. Hersh's stiff upper body and his causally stretched out legs struck me as being as disjunctive as was his account of how he had been arrested."

We may add a technical appendix to reports, with commentary on assorted test features, for psychologist readers. This information is regarded as a tool, data for further reflection, not as findings. Again, "findings" are clarifications of life events. We explain any diagnoses contained in the report, in the client's language. We often provide the appropriate pages of the *DSM (Diagnostic and statistical manual of mental disorders)* for the client to read and then discuss.

The report is regarded by all as a progress report, a report of progress to date in understanding the client's situations, and in developing concrete suggestions. After addressing any



referring party's concerns, the report ends with a section itemizing suggestions that client and assessor developed. For example, "(2) When you've arrived at a solution instantly, remember to slow down to show others how you got there, or how they might get there. You'll recall that we talked about this in regard to my not seeing your "battlefield" on the Rorschach until you guided me through it. We then related that to times your co-workers and supervisor have thought that you were just shooting from the hip, until you helped them to find their way to your insight."

We include a section for the client to write commentary about the report. Often we find that the client has developed further understandings since our last meeting (usually two meetings occur prior to reading the written report). Even when the client does not want to write much on the report, the invitation affirms the client as a collaborator in the process. Knowing that the client is going to read the formal report also encourages the author to find expressions that are true to the client's life.

#### 1.15.8.5 Judging Validity

In that the report deals with direct observations and reports of actual events, we are not faced with the task of justifying inferences. Instead, the criterion is that client and readers must recognize in the report's descriptions touch points with their interpretations or experiences of this client or similar clients, and therein a consensual validity is formed. Ambiguity of the life world is respected when certain findings are implied or evoked rather than spelled out. Differences in perspective are respected as inevitable, and it is understood that further observation and reflection will result in continuing clarifications and revisions. No portrait, description, or other interpretation can be final. In the meantime, the practical utility of the concrete, individualized, suggestions is a strong form of validity.

#### 1.15.8.6 Comments

Working within a human science frame does not bypass the critical contributions of neuropsychology, medicine, cognitive psychology, object relations theory, diagnostic systems, and so on. We take these contributions most seriously but do not adopt the reductive philosophies within which many of them were developed and/or frequently are practiced. For example, although it is definitely helpful to know that a person matches criteria for borderline personality disorder, that match

does not explain the person. Although various knowledge domains do provide information about a person's present restrictions and possibilities, we still must explore that person's ways of going about life, so we can locate when-nots, help that person to develop personally viable pivot points, and so on.

Even when intending to work within a human science orientation, we find ourselves falling into our culture's and our discipline's reductive attitude all too easily. Hence, in my assessment courses I include numerous exercises to remind us of the primacy of the life world, and that test data are always about a life that we are responsible for getting back to. On the blackboard I draw the first Bender design, but with the circle and square overlapping instead of adjacent. The class offers ideas about how an overlapping rendition might occur. The ensuing range reminds us not to rush to interpretation from manuals, but rather to be open to a range of possibilities: being in a rush, attending to just getting through the task rather than trying to be precise, resenting having to go through an assessment, leaning on others for comfort or support or to avoid the possibility of not connecting, being unmindful of boundaries. In another exercise, Rorschach students write descriptions of their own situations in "Rorschachese": "There I was, M<sup>a</sup>ing toward Fd, when a large H AG'd into the line in front of me; I found myself Hxing (H) images, and imagining him MOR." To further ground Rorschach ratios in life, we act out introversive and extratensive styles of entering the classroom late. In still another exercise, we observe slides of the American painter Andrew Wyeth's representational art, and talk about how we too re-present our subjects. We do so without "artist's license" to change features, but with awareness that we too are creating a portrait, from our perspectives and with our lives as points of access.

#### 1.15.9 CONCLUDING REMARKS

Through emulation of the natural sciences, psychology rightly has achieved status as a rigorous discipline. From that secure place, however, we now note that the experimental method, when based on a residual positivistic philosophy of science, is not fully adequate for studying human affairs. An approach that studies humans in terms of their being subjects (agents) as well as objects is human science psychology. This approach respects data formed through natural science methods, but looks more broadly to lived-world contexts to reflect on their significance.

As fewer psychologists identify with radical behaviorism or a strict logical positivism, and more hope for psychology to do greater justice to *human* phenomena, psychologists increasingly are adopting the term “human science” to characterize their own leanings. Simultaneously, qualitative research methods are finding their way from other disciplines into psychology. Through our national organization, psychology advocates respect for cultural and individual differences, and urges that we become familiar with what it is like to be in other people’s situations. In these and many other ways we find ourselves increasingly heeding persons’ unitary relations with their worlds. I have written this chapter as an invitation to revisit the human science literature, which affords promising possibilities of unifying our discipline. Although human science psychology has been outside the mainstream, its history is as long as that of prevailing psychology, and its grounding philosophy is at least as formidable.

Studies exploring the mutuality of biology and psychology, integrative research methods, and transdiscipline cooperation are all still in their beginning stages. However, their emergence is now accelerating through the work of diverse communities of scholars and practitioners. The second edition of *Comprehensive Clinical Psychology* will probably include in its foundations volume the growing literatures of hermeneutics, linguistics, feminist studies, constructionism, and other endeavors that acknowledge human participation in what we take to be reality. Indeed, perhaps the second edition will explicitly summarize work exploring how it is that our clinical practices necessarily draw on our life worlds. These are exciting, promising times.

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# 1.16

## Family Systems and Family Psychology

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### 1.16.1 INTRODUCTION

The place of family systems theories and couples and family therapies within psychology has vastly expanded in recent years. Once seen as a radical departure from the more traditional focus on the individual that has typified the mental health disciplines, family systems viewpoints have now been with us for over 40 years and gained wide acceptance. Many systemic theories and therapies have been developed, and couple and family therapies are now among the most widely practiced. Several prominent guild organizations supporting the practice of family therapy have blossomed, including The American Association for Marriage and Family Therapy, the American Family Therapy Academy, and the Division of Family Psychology of the American Psychological Association, as have a number of prominent journals including *Family Process*, *Journal of Marital and Family Therapy*, and *Family Therapy Networker*. The Division of Family Psychology within the American Psychological Association now has over 6000 members, while the American Association for Marriage and Family Therapy has over 25 000 members.

The essence of what has driven all this attention lies in the emergence of a broad recognition of the importance of the family in the life of the individuals within it and the society made up of families. Whether we consider the impact of a depressed parent on a child, the role of poor parenting practices as a risk factor for conduct disorder in children, or the impact spouses have on one another, both clinical experience and much research point to the enormous influence of the family. Gurin, Veroff, and Feld (1960) found that 42% of all people who had sought professional help for psychological problems viewed their problems as related to a marital problem, and another 17% viewed their problems as pertaining to family relationships.

However, family therapy is truly unified only in the shared belief that relationships are of at least as much importance in the behavior and experience of people as are internal processes within individuals or broader social forces. As Gurman, Kniskern, and Pinsof (1986) have suggested, family therapy includes therapists from many professions, has no unified theory, and few techniques that are specific to it. There are many distinct systemic therapies, which differ enormously from one another. Some are directed to the treatment of families, some to subsystems within the family (e.g., couples), and others abandon the specific focus on the family entirely, aiming at the broader social nexus. In this chapter, we overview basic systems concepts

and the field of family therapy, highlighting both the common threads underlying these methods and the differences that have emerged across the schools of practice.

### 1.16.2 DEFINING THE PURVIEW OF FAMILY THERAPY

Delineating what is meant by family therapy is far more complex than might be thought. The simplest way of labeling a therapy as a “family” therapy is to look at who is seen in treatment. From this vantage point, family therapy occurs when more than one member of a family are seen together in psychotherapy. This definition has considerable advantage in its parsimony. Accepting this definition, it is clear when family therapy is occurring and when it is not, an appraisal that can be made through a simple head count. From this viewpoint, the meeting of a couple, a parent and son, or a multigenerational family with a therapist, all constitute family therapy, whereas meetings of therapists with one individual or with unrelated individuals in groups do not. In most such usage, the term “family therapy” is restricted to instances when family members are seen together at one time (also more specifically termed conjoint family therapy), although at times the definition has been expanded to include therapies in which various members of the family are seen in different sessions, termed concurrent treatment (e.g., when a child is seen in some meetings and parents in another).

Despite the attractiveness of such a simple straightforward definition, most family therapists have not found this way of conceptualizing family therapy either satisfying or sufficient. Instead, most family therapists view the essence of family therapy as centered on maintaining a focus on the system rather than who is seen in treatment. They point to therapies in which multiple members of families are seen but in which the focus remains on an individual (e.g., as when the focus is exclusively on changing the symptomatic client) as failing to meet the criterion for family therapy, and to other therapies that directly involve only one client but focus on the social system (e.g., Bowen Therapy) as family therapies. It has been the presence or absence of a systemic focus of treatment that has emerged as most important to the majority of those who practice family therapy. Based on such a notion, Gurman et al. (1986) offered the following classic definition of family therapy: “Family therapy may be defined as any psychotherapeutic endeavor that explicitly focuses on altering the interactions between or among family members and seeks to improve

the functioning of the family as a unit, or its subsystems, and/or the functioning of individual members of the family.” This definition best fits with the zeitgeist of family therapy, but leaves some room for debate about whether a particular treatment is “family therapy.”

A related issue in demarcating the territory of family therapy concerns the place of couple therapy, specifically whether couple therapy represents a subset of family therapy or a separate endeavor. Some have argued that because couples therapy calls for a distinct set of skills, it should be considered separately (Alexander, Holtzworth-Monroe, & Jameson, 1994). More typically, couples therapy has been regarded as a subset of family therapy (Lebow & Gurman, 1995). Most prominently, those who emphasize the importance of a systemic viewpoint as the essence of family therapy view the treatment of couples as simply work with one family subsystem.

It also should be mentioned that in the late 1990s “marital and family therapy” has become “couple and family therapy.” Respect for the diverse forms of family in our society (Walsh, 1993) has led to this fundamental change in nomenclature.

### **1.16.3 THE VARIETY OF FAMILY THERAPIES**

Family therapy is actually a number of different activities linked by a few common understandings. Systemic concepts merely set a frame for possibilities. They do not limit the range of intervention. Both the mediating and ultimate goals of treatment may vary, as may the theoretical frame, the treatment strategies, and the specific interventions employed (Gurman, 1978). Therefore, it makes conceptual sense to subdivide family therapies into ones that share common characteristics.

Family therapies can be classified best along two axes: the first consisting of who is seen in treatment (e.g., individual, couple, nuclear family, extended family), and the second, the theoretical perspective on which the therapy centers (e.g., structural, strategic, object relations, or a type of integration). Both who is seen and the theory of the approach contribute to the differences between therapies at the level of operations.

It is also crucial in examining this literature to keep in focus the importance of treatment goals (Gurman, 1978). In couple and family therapy, change in family process is always both a mediating and an ultimate goal. Most couple and family therapy also has an additional goal, be it a change in the behavior of an individual or

in a broader dimension of relational life, for example, marital satisfaction. In examining family therapy, we must maintain a focus on the impact on both family process and other treatment goals.

### **1.16.4 A BRIEF HISTORY OF COUPLE, FAMILY, AND SYSTEMS THERAPIES**

To understand the enormous variation among family therapies, it is important to grasp the diverse origins of the field. The earliest couple and family therapy was framed as a direct extension of existent models of individual therapy. Although much of this work was conjoint, the essence of these methods consisted of helping to uncover individual patterns to be altered. In these therapies, some of the core techniques for working with more than one client in the room were developed (e.g., early variants of communication training), but these therapies were largely considered adjunctive to what was viewed as the more important work of individual therapy.

Family therapy leapt to prominence through the work in the 1950s and 1960s of such figures as Nathan Ackerman, John Bell, Ivan Boszormenyi-Nagy, Murray Bowen, James Framo, Jay Haley, Donald Jackson, Salvador Minuchin, Virginia Satir, Carl Whitaker, and Lyman Wynne, who shared a common belief in the core importance of the family system as well as a great deal of personal charisma. In contrast to earlier couple and family therapies, this work actively questioned and argued against the traditional individual oriented view of problem development and treatment. The early work of these pioneers included a wide range of family-focused intervention, crossing the boundaries of schools of family therapy that have subsequently emerged. Each developed theories and methods of intervention, and several ultimately shaped their methods into the foundation of a school of treatment. Each incorporated aspects of systems theory into their work, most prominently the view that causality is best conceived of as a circular process in which behavior is seen as interdependent and subject to mutual influence. Within the zeitgeist of the time, the behavior of “identified” patients (i.e., those with symptoms who were labeled as having the problems) were seen as a reflection of underlying family process, that is, the family was viewed as the principal locus of the problem, central in its development, and the most appropriate context for treatment. The first generation of family therapists emphasized systemic concepts with the kind of fervor that

goes with those who have discovered an as yet undiscovered truth, and were highly critical of traditional methods of mental health intervention in which individuals were seen out of their natural context in the social system.

The influences on this generation of pioneers themselves were enormously diverse. Some had backgrounds in psychoanalysis (e.g., Ackerman, Framo) that they brought to bear. Some had backgrounds in fields other than the mental health professions, such as anthropology (e.g., Bateson, Weakland) and communication (e.g., Haley). In general, new and exciting ideas were welcome and sought out. Thus, for example, Haley became quite interested in the work of Milton Erikson, bringing core techniques of his variant of hypnotherapy, emphasizing paradoxical intervention, into the mainstream of methods of practice in family therapy. Observation of the importance of difficulties in communication in troubled families, based on the work of the double bind project (Bateson, Jackson, Haley, & Weakland, 1954) and the parallel work of Wynne, Ryckoff, Day, and Hirsch (1958) and Lidz, Cornelison, Fleck, and Terry (1957), also exerted an enormous impact, focusing emphasis on changing these deviant communication patterns.

Through the 1970s and 1980s, the field of family therapy passed from the shared excitement about the core importance of the family system to emphasizing differences across the many distinct schools that emerged. Some of these schools accepted or transmuted a range of concepts from individual therapy, creating schools that are psychoanalytic (Ackerman, 1958; Scharff & Scharff, 1987), experiential (Greenberg & Johnson, 1988; Whitaker & Keith, 1981), and behavioral (Patterson, 1982). Other schools rejected virtually all aspects of individual models of treatment, remaining exclusively focused on aspects of the social system such as structure (Minuchin, 1974) overcoming family homeostasis (Watzlawick, Beavin, & Fisch, 1974), or intergenerational process (Bowen, 1978). Across these schools, a vision developed of a powerful therapist (sometimes literally referred to as a wizard), jousting or performing some version verbal judo to free up the family from its patterns. Through this time, family therapy grew enormously in popularity and began to enter into the mainstream of practice.

In the 1980s, voices began to emerge within family therapy that were highly critical of aspects of the schools of practice that had developed. Most prominently, the feminist critique pointed to the numerous male assumptions that were endemic to most models in family therapy (e.g., that fathers should hold the

major executive position in the family). Feminists called for a more egalitarian family therapy (Goldner, 1985; Hare-Mustin & Maracek, 1988). Other criticisms focused on the basically homeostatic vision of family life in family therapy, in which families were seen as highly resistant to change and readily relapsing into dysfunctional patterns. Alternative visions developed emphasizing family resilience and the natural tendency in families toward change (Walsh, 1982, 1993).

Other questions were raised about whether the therapist needed to be the powerful enactor of change depicted in many of the earlier models of treatment. Theoretical precepts of second-order cybernetics were advanced (Hoffman, 1981) that emphasized the view that there were no "objective" observers (Von Glaserfeld, 1984), and that therapists, like all others, become part of the system, being influenced by it as well as influencing it. Following Gergen (1981), social constructivism further argued that knowing and knowledge are socially constructed through language and discourse. Models of family therapy developed that emphasized collaboration (Anderson & Goolishian, 1988) and the personal construction of narrative (White & Epston, 1990).

Still others became critical of the exclusive focus on the family as the locus of change in systemic models. Movement has clearly been away from the earlier simplistic notion that the family was the sole etiologic agent in the development of difficulties, and that family therapy was the sole preferred method of intervention across all difficulties. An integrative viewpoint is emerging that includes not only the concepts from various family methods of intervention, but also interventions at the level of the individual (Lebow, 1984, 1987a) and larger system (Breunlin, Schwartz, & Karrer, 1992). Some have even called for a basic redefinition of systemic therapy, moving from a specific focus on the family to a broader vision of consultation with social systems, including but not limited to families (Wynne, McDaniel, & Weber, 1986).

Family therapy has also been examined through the lens of several other pertinent and powerful vantage points including culture (Boyd-Franklin, 1989; McGoldrick, Pierce, & Giordano, 1988), life cycle development (Carter & McGoldrick, 1988), and postmodernism (Hare-Mustin & Maracek, 1988). This has been a field boiling over with ideas and concepts, and ways of examining the family and how to have an impact on it. It has been a continually developing field, in which treatments have emerged and been refined, theory has undergone considerable revision, and assumptions



have been continually examined within the emerging vantage points about family within the broader society.

### 1.16.5 CORE SYSTEMIC CONCEPTS

A few core assumptions underlie all family therapy, the most central being the core importance assigned to the social system in influencing individual behavior. Families are seen as having a powerful effect in shaping and maintaining individual patterns of behavior. There have been a range of ways of conceptualizing this influence represented in the various models of family therapy.

One set of core concepts that have been prominent within a wide range of models derives from general systems theory, developed by Von Bertalanffy (1969) as a way of understanding all systems, animate and inanimate. The central tenet of general systems theory is that the whole is more than the sum of its parts. Therefore, to understand any part (e.g., an individual), one must grasp its relation to the whole of which it is a part (e.g., the family).

Within general systems theory, humans are viewed as part of what is termed an "open" system, one in which there is ongoing exchange with those outside, be they other individuals, families, or other systems. Open systems remain subject to influences from outside. Systems (e.g., families) are also made up of subsystems (e.g., a couple, children), which affect one another, and sum to more than their parts. General systems theory focuses on the most global principles of how systems evolve, be they particles in space or human families.

A principle that assumes great importance in general systems theory is equifinality. Equifinality suggests that there are many ways of reaching particular configurations within the system and that the particular pathway by which a configuration has been reached does not matter. End states reached through different pathways are equivalent. Applied to family systems, an emphasis on equifinality focuses concern on the state the family is presently in and not how the family reached that state. To the extent equifinality is stressed, history and individual motivation assumes lesser importance, while the topography of how the system is presently organized assumes more.

In general systems theory, how behavior is understood is viewed as a function of the context in which it is conceived. In a classic example cited by Watzlawick, Beavin, and Jackson (1967), the meaning of seeing a man quacking at ducks is significantly altered by the knowledge that this man is Konrad Lorenz, engaged in experiments about imprinting.

Without that piece of information, the behavior looks eccentric or psychotic, but sense can be made of this behavior in the appropriate context. Early in the history of family therapy, a focus on context became the cornerstone of the belief that the behavior of all family members would make sense if only the meaning of the behavior in the appropriate context could be deciphered. In particular, severe mental illness was seen as the product of behavior that made sense in a particular context (e.g., within the family process), although it appeared to make little sense when seen outside of that context.

A common extension of these concepts early in the history of family therapy was to label the behavior of the family members displaying psychopathology or other problematic behavior as "identified patients." These "identified patients" were typically seen as the victims of labeling of their behavior when considered out of the appropriate family context. Within this viewpoint, the real patient and bearer of the problem is the family, and therefore, family therapy is the most appropriate method of bringing about change. A frequently encountered correlate of this set of beliefs was dismissal of the biological basis for severe mental disorder (e.g., Haley, 1963).

General systems theory also emphasizes circular paths of causality. Rather than focusing on linear pathways of actions followed by reaction, attention centers on recursive patterns of mutual interaction and influence. If the behavior of one person affects that of another (e.g., a father punishes his child), attention still must focus on how the response of the second person affects the first (e.g., the child's aggressive behavior leads to the parent's punishment, the parent's punishment leads to the child temporarily stopping the behavior, followed by further aggressive behavior). From the perspective of general systems theory, the system, not a single person, is responsible for the behavior that is maintained through such circular pathways.

Some of the critiques and reappraisals of systems theory have been directed at these concepts. One line of criticism has centered on the notion of "identified patients." Much research has shown very real disabilities in those with mental illness. Many now question the use of this term when applied to situations in which there is psychopathology. In particular, the psychoeducational movement has called attention to some of the costs of this approach in alienating families from the treatment they need, because of the sense of shared family causation that accompanies usage of the term "identified patient."

Others have argued the limits of circular notions of causality (Dell, 1986), pointing to the dangers inherent in the argument that all parties are equally responsible for sequences of behavior. In particular, the example of family violence has been cited as an instance where individual responsibility and lineal arcs of causality need to be highlighted, lest the observer be left with an inappropriate sense that batterer and victim are coequal in responsibility for violent behavior (Dell, 1986, Goldner, Penn, Sheinberg, & Walter, 1990). There is considerable agreement among family therapists that both lineal and circular pathways of causality and problem maintenance need to be considered in assessing family systems.

Cybernetics, the science of communication and control in man and machine, developed by Wiener (1961) and others, added to the understandings developed through general systems theory among family therapists. In cybernetics, systems are viewed as self-correcting, influenced in an ongoing way by feedback. Feedback is the process by which a system gains information to self-correct to maintain a steady state or move toward a goal. Positive feedback describes input that increases deviations from the steady state, negative feedback describes input that reduces such deviations. Homeostasis is the powerful force moving the system toward a steady state. Early family therapy was profoundly influenced by the idea that human systems were homeostatic, that is, moving toward the reduction of change. As a result, most first-generation family therapies were based in the notion that powerful interventions needed to be created to reorganize the family, overcoming the homeostatic forces.

Systemic thinking has given more weight to morphogenesis, the natural force moving the system toward change. A systemic emphasis on morphogenesis creates very different implications for psychotherapy than a homeostatic emphasis. Models emphasizing morphogenesis suggest that initiating the process of change is likely to kick off a positive chain reaction, very unlike the minimization of change thought to be active in a perspective emphasizing homeostasis. Thus, in morphogenetic models, launching small changes is seen as likely to be productive in creating movement within the system, while in homeostatic models, only the most powerful of interventions are seen as likely to produce change. In a similar vein, Maturana and Verela (1980) highlight the process they termed autopoiesis, whereby the internal structure of the living system determines its behavior. Following the notion of autopoiesis, the therapist can only perturb the system to make changes that the client system itself produces.

Another important emphasis in systemic theory has been on communication processes. The point of entry for these ideas in family therapy was the double bind theory of schizophrenia developed by Bateson et al. (1954), which suggested that the psychotic process was the product of disturbed communication. In the double bind, two or more parties are involved in an important relationship that is ongoing. A primary injunction is given, such as "show me your feelings." A second injunction is given that conflicts with the first, such as "The feelings you have are unacceptable and should not be verbalized." Given that the recipient of the communication cannot leave the field, he or she is in a double bind that induces anxiety. The double bind theory suggested that the repeated exposure to such binds results in responses that break out of the bind through psychotic processes.

Although empirical research never was able to demonstrate high frequencies of such double binding on the part of parents of schizophrenics (indeed, in the research, the presence of double bind communication could not even be reliably rated as present by observers), this work has remained highly influential in family therapy in focusing interest on deviant communication processes in families. Beginning with the premise that "you can't not communicate" (Watzlawick et al., 1967), this work sought to understand the communication which was occurring, and alter patterns of communication so that the system could use this communication to reorganize in a more functional manner.

### **1.16.6 MODELS OF FAMILY THERAPY**

Many specific couple and family therapies have been developed. Some of these models have been directed to specific difficulties (e.g., Kaplan's treatment for sexual disorders), but the majority have been directed toward a broad range of problems. Some family therapies are aimed at resolving difficulties that are explicitly labeled as being about family relationships (e.g., couple therapy aimed at marital dissatisfaction; family therapy aimed at overcoming differences between parents and their grown children), whereas other family therapies utilize a family systems perspective for intervention with problems that manifest themselves in the behavior of an individual, and which from another perspective might be seen as "individual" problems (e.g., depression). The numerous couple and family therapies can be divided into a few distinct categories on the basis of emphasis. These are structural, strategic, cognitive-behavioral,

psychoeducational, intergenerational, psychodynamic, experiential, narrative, and integrative schools of family therapy.

#### 1.16.6.1 Structural Family Therapy

Structural family therapy, developed by Salvador Minuchin, is an example of a family therapy largely drawn purely from systemic concepts. Structural family therapy emphasizes the power of the social system, as manifested through family structure. Health and dysfunction are directly viewed as products of the effectiveness of the family structure. By family structure, Minuchin means the regulating codes as manifested in the operational patterns through which people relate to one another in order to carry out functions. The three primary dimensions of structure are boundary, alliance, and power.

Boundaries are the rules defining who participates and how, who is in and who is out of an operation, regulating contact. The strength of boundaries vary, ranging from rigid, resulting in disengagement, to very permeable, resulting in enmeshment. At the disengaged end of the spectrum, families act like they have little to do with each other, leaving the individuals substantially disconnected. At the enmeshed end, there are violations of function boundaries, in which family members intrude into functions that are the domain of other family members. Structural family therapy aims to move families away from the extremes of enmeshment and disengagement.

Alliances are the joining or opposition of one member of a system to another in carrying out an operation. Alignments are inevitable. They become dysfunctional when they become fixed and unchanging (stable coalitions) or when they are primarily cross-generation. Triangulation describes the process of two people demanding that a third join with them against the other. Structural family therapy aims to create alliances that are functional (e.g., parents with one another), while at the same time not becoming rigid.

Power describes the relative influence of each family member on the outcome of an activity. Power can be functionally distributed with the primary locus in the older generation or can become rigidly held by one individual or coalition, or there can be insufficient executive authority (weak executive function). Power is seen as best held in the hands of an executive (e.g., a parental coalition), but in such a fashion to leave everyone with some degree of power.

Treatment in structural family therapy primarily consists of efforts to change these elements of family structure. Given the homeo-

static vision of systems at the base of structural therapy, therapists seek to create powerful in-session experiences to work to alter the family's organization. Frequently, a transaction is created by the therapist that promotes in-session family members' habitual patterns of relating (called enactment). There is much emphasis on joining with the family through such interventions as tracking (adopting the symbols of the family's life), accommodation (relating to the family in congruence with the family's patterns), and mimesis (joining with the family by becoming like the family in manner or content). Ultimately, interventions are directed to fully restructuring the system. Symptomatic change in "identified" patients is assigned a much less important role than change in structure of the system.

The structural approach remains the most influential school within family therapy. Some of its ideas, such as the importance of boundary, alliance, and power in family systems have come to be broadly accepted by most family therapists. However, there has also been considerable criticism of some of the assumptions of structural therapy. Most emphatically, the highly gender-based nature of some of the assumptions about the roles of men and women and boys and girls within structural family therapy has resulted in considerable criticism, particularly from feminists. The emphasis of this approach on equifinality and, thus limited concern with history or the internal process of individuals, has also been attacked. Minuchin (1996) has recently moved to a more gender aware version of the structural approach, and acknowledged a greater appreciation for the importance of history.

#### 1.16.6.2 Strategic Approaches

Strategic approaches are the most purely systemic of the family therapies. These models simply ask, "What is the most expeditious route to promote systemic change?" Strategic approaches also share an orientation toward brief focused intervention. From a strategic viewpoint, change is a discontinuous process. The goal is to intervene, find a new way of functioning that works better, and promptly end the treatment.

Strategic models have also been closely identified with several specific methods. One common thread is the use of "paradoxical" interventions, in which directives are offered which if acted on would move the family in the opposite direction from that which is desired. Although direct interventions are also common in strategic methods, the rapier-like effort to find the simplest and most expedient pathway to

change is best represented by the use of paradox. Strategic methods have also been closely associated with the use of team approaches, utilizing observers behind one-way mirrors as part of the intervention process, typically offering commentary or directives to the therapist and family. Strategic methods have been further associated with cool detached stances on the part of the therapist (e.g., the MRI model), although there have been significant exceptions to this trend. Strategic therapies center on altering feedback cycles within the family, but do not seek to enable insight within the family about such cycles. Change, not learning about the change process, is clearly the center of attention. The strategies of change invoked vary significantly with the specific strategic model.

#### **1.16.6.2.1 The Mental Research Institute**

The first systems-based strategic model within family therapy was the "Mental Research Institute" (MRI) or "Palo Alto" model developed by Jackson, Watzlawick, and Weakland, and their colleagues (actually the first MRI model, as there have been subsequent other MRI models). The MRI model derived from a mix of systems theory, cybernetics, and the study of communication processes.

In the original MRI model, problems are viewed as a natural part of family life that families regularly deal with on their own. The need for intervention is seen as stemming not from the problem itself, but from how family members treat the problem. When families become stuck in systemic patterns and in efforts to solve problems ("more of the same"), changes in behavior, termed "first-order change," are seen as unlikely to resolve the problem, and possibly making the problem even worse. Therefore, therapy focuses on the creation of "second-order change," an alteration in the rules of the system that govern interactions. Treatment consists of identifying the ways problems are maintained by the behavior within the system, examining the rules that lie beneath these behaviors, and then changing the rules. Reframing and paradoxical interventions are viewed as the most powerful tools for initiating second-order change. Reframing consists of active efforts by the therapist to create a new and different understanding of old events that has a more benign meaning. For example, in recasting behavior that has been seen as bad as being out of the control of the individual, a different reality is created. Reframing has become perhaps the most common intervention in couple and family therapy, employed by therapists regardless of their orientation.

Paradoxical directives capitalize on the force in social systems to move against the direction toward which it is directed. For example, the therapist may list reasons why change is not likely to be productive or even harmful. Although much more controversial than reframing, paradox has become common within family therapies, especially strategic ones.

Treatment within the MRI model always remains brief and focused. The therapist's cool detached stance is not designed to make for long-term attachment, and termination is encouraged by the therapist as soon as problem resolution has been substantially initiated.

The MRI model stands as the exemplar of a strategic family therapy, and has been highly influential among all other strategic therapies. Several of its core concepts (reframing, "more of the same," first- and second-order change) have been adopted within a broad range of family approaches. Nonetheless, although highly influential, this approach today is rarely seen in practice in its pure form. The paradoxical bent of the intervention strategy and the detached stance of the therapist have caused this approach to receive a great deal of attention, but have also led many who have tried this approach to move on to other models that emphasize a more collaborative approach between clients and therapist. In addition, little empirical support is available for this approach.

#### **1.16.6.2.2 Haley's problem-solving therapy**

Haley's (1976, 1980) problem-solving therapy and the closely related work of his colleague Madanes (1981) combines a strategic use of paradoxical techniques with goals that typify structural family therapy. Problem-solving therapy strongly emphasizes grasping and working with the function that behaviors serve within the system. Most often, this function is conceptualized as a struggle for power and control.

An initial stage of the therapist and client becoming acquainted is followed by a stage in which each person's perspective about the problems which exist is elicited. Specific observation by the therapist focuses on triangles (who supports whom in interaction) and hierarchy (who has what power), but these ideas are not directly shared with the clients. Directives to the family follow from a consideration of solutions that have been attempted, and aim to engage the family in new and different behaviors. Many of the techniques utilized by Haley and Madanes derive from the hypnotic work of Milton Erickson, aimed at increasing suggestibility and openness to change. One example is the pretend technique

(Madanes, 1981), in which the family is directed to have children pretend to have symptoms and parents pretend to help them, a paradoxical technique that suggests the possibility of overt control over patterns thought to be out of conscious control. Other commonly employed techniques more directly aim at establishing a coalition between parents to help adult children leave home.

Haley remains highly controversial in his adherence to some of the earliest systemic conceptualizations of family therapists, for example, the view that “identified patients” carry symptoms entirely due to the function these symptoms serve within the family and a denial of the existence of mental illness. For Haley, psychopathology is always the product of a dysfunctional social system, not due to biology or individual psychology. These ideas, once a welcome contrast to the determinism of biological and psychoanalytic formulation, now appear rigid and stale in the wake of the development of the considerable literature delineating the biological and psychological basis for severe disorder, and the emergence of highly effective psychoeducational treatments that demonstrate how an approach can be family based and yet consistent with the best data about schizophrenia and other mental illness.

#### **1.16.6.2.3 Milan systemic therapy**

A number of strategic therapeutic approaches have been developed in Milan, Italy, by Selvini-Palazzoli, Boscolo, Cecchin, Prata and their colleagues in various combinations. Versions of these models have varied enormously, although all have maintained a strategic focus.

In the classic Milan therapy that brought worldwide recognition to this group (Selvini-Palazzoli, Boscolo, Cecchin, & Prata, 1978), sessions are held approximately once per month, almost always involving a team situated behind a one-way mirror. The team forms a hypothesis about the family, to be modified over the course of treatment. During a break in each session, the team formulates an intervention. The therapist then brings the intervention to the family, most often through positive connotation or the prescription of a ritual.

Positive connotation consists of reframing behavior in a positive light, most frequently through suggesting how the behavior serves the goals of the system. Positive connotation aims to change the family view of dysfunctional behavior, while also decreasing resistance by allowing each family member to emerge with a positive view of their own behavior.

The rituals prescribed in Milan systemic therapy move to exaggerate or move against rigid patterns in the family. Most of the rituals have an ironic quality and engender confusion, although some (e.g., one called “odd and even days,” in which control is given to each parent on alternating days) merely serve to call attention to patterns in the family, and thereby move the family to see their ability to impact on the situation and resolve their difficulties. The early Milan approach strongly stressed the importance of therapist neutrality in delivering these interventions.

In the most influential variant of the Milan methods, Boscolo and Cecchin (Cecchin, 1987) moved away from directives toward the use of what they termed “circular questions.” Circular questions are questions used to learn about differences in the family that might provide clues to recursive family patterns. Circular questions include ones about differences in the perception of relationships (who is closer?), differences between before and after something else happened (were you more depressed before or after the birth of the baby?), and hypothetical differences (if you had not married, how would your life be different?). Curiosity is the essential ingredient in circular questioning. The aim is not to move the family toward a specific goal, but to initiate thought and conversation in order to create greater understanding of how the present situation and the family’s behavior in it came about, what the systemic patterns are that help keep the family from resolving their difficulties, and what are the most productive pathways toward change. Work in this model becomes much more collaborative than in the earlier version of Milan therapy.

Selvini-Palazzoli (1986) added another variant of the Milan model. Selvini-Palazzoli came to believe that disturbed patients were inevitably caught up in what she termed the “dirty game,” a power struggle between parents in which patient’s symptoms help support one parent. Her response was what she called “the invariant prescription,” applied to all families. In the invariant prescription, the therapist suggests to parents that they tell family members that they have a secret and go out together mysteriously without warning other family members, and that they then observe the family’s reaction. The “invariant prescription” aims to help strengthen the alliance between the parents and enable understanding of dysfunctional patterns in the family. Although rooted in some of the same observations about triangulation in disturbed families as those made by Haley, Bowen, and Minuchin, this approach has acquired little support because of its highly pathological view of family processes, its

ignoring of the mounting evidence demonstrating the importance of expressed emotion to recidivism, and its failure to respond to differences among families.

More generally, the Milan approaches have proven to be highly influential. Although only a small number of family therapists practice any of the variants of Milan therapy, the attitude of curiosity and prompting of circular questions have come to serve as the base of investigation for many family therapists. There has been very little empirical testing of the Milan approaches.

#### ***1.16.6.2.4 Solution-focused therapy***

Emerging into prominence since the mid-1980s have been a number of approaches that take positive connotation and reframing a step further, attempting to move discussion fully to thinking in terms of solutions rather than problems. Among the best known of this solution-focused set of approaches is the work of DeShazer (1985, 1988), Berg (1993), and O'Hanlon and Weiner-Davis (1989). The solution-focused approaches assume that clients want to change and reject the notion of deeply ingrained pathology. Instead, these approaches seek to introduce ways of thinking about and facing difficulties that are different, and can kindle the family's own process of resolving their difficulties. One favorite technique is to look for exceptions, times when problems have not been present or overcome. Another has been to nurture and help clients notice small changes from which they can build larger ones. For example, De Shazer asks clients to observe what happens in their lives that they want to continue. DeShazer and colleagues also employ the "miracle question": "Suppose one night, while you were asleep there was a miracle and this problem was solved. How would you know? What would be different?" All these techniques are designed to help clients begin to think in terms of solutions and the ability to resolve difficulties rather than in terms of problems and one's difficulty in resolving them.

Solution-focused approaches have been among the most widely influential in the 1990s, especially in the context of the mandate of managed care for brief therapy. The positive focus and optimistic frame of these models has proven most welcome to families and therapists alike. Criticism has focused on the repetitive use of the same few interventions (e.g., the miracle question), and on a very simplistic notion of problem development and resolution implicit in the model. Ultimately, whether such parsimony and positive ideas are sufficient is an empirical

question. Unfortunately, given their promise, solution-focused approaches remain among the least studied of the family therapies.

#### **1.16.6.3 Cognitive-behavioral Approaches**

Cognitive-behavioral models extend behavioral principles to the treatment of family systems. These models have primarily been utilized in work with child behavior problems (especially conduct disorder and delinquency) and with difficulties encountered by couples (especially marital dissatisfaction). Cognitive-behavioral methods begin with the assumption that thoughts and behavior are central to all aspects of functioning and that the most efficacious pathways to change directly address dysfunctional thoughts and behavioral patterns.

Classical and operant conditioning are the central mechanisms for shaping behavior within a behavioral paradigm. In behavioral family therapy, operant conditioning has assumed particularly great importance.

Central to operant conditioning is reinforcement: presenting some event or behavior that increases the rate of a particular response. Humans are seen as inevitably affected by the reinforcements they receive. However, behavioral family therapy is not fully the product of classical learning theory, but instead its application in the social context, called social learning theory. Here, learning is not simply the product of primary reinforcers such as food, but of social reinforcers, such as approval. Social learning also occurs both directly from experiences that reinforce or punish, and indirectly through processes such as modeling, in which learning occurs through observation of contingencies. This array of processes shape social behavior.

Social exchange theory (Thibaut & Kelley, 1959) also has had a prominent place in cognitive-behavioral approaches. Social exchange theory suggests that individuals strive to maximize their outcomes, to increase the rewards they receive, and decrease the costs. Behavior from one person is viewed as likely to be met with reciprocity to behavior from another, so that positive behavior will lead to positive behavior, and punishment to punishment on the part of the other. In particular, couples are regarded as likely to develop social exchanges that can become mutually supportive (each emitting positives to the other) or coercive (each emitting punishing behaviors). Problem behavior is viewed as primarily the product of either skill deficits that stem from a lack of knowledge, or from the establishment of coercive exchange. Skill training is aimed at providing the knowledge and experience needed

to engage in appropriate social behaviors, be they as a spouse or a parent. Positive exchange is altered directly by helping clients become more aware of patterns of exchange, and by negotiation of a more satisfying *quid pro quo*.

The cognitive theories that make up the cognitive part of cognitive-behavioral emphasize the development and maintenance of dysfunctional or "irrational" thought processes, and direct efforts to alter these cognitions through learning in therapy. Cognitive interventions examine the ideas that lie behind behavior and emotion for the presence of core distortions. The emphasis lies in being able to understand the importance of the thought that lies between an experience and the resultant feeling. Cognitive interventions principally help clients to understand and alter the tendency to overgeneralize, personalize, or be overly negative about events that are occurring. Homework is essential in tracking and assessing beliefs, just as it is essential in accomplishing behavioral goals.

Early in the history of behavioral couple and family therapy, there was little that was systemic in the approach. Systems theory was not part of the theoretical base. Instead, the emphasis was on ways of promoting individual learning to change family patterns. It was not unusual for behavioral family therapists to meet exclusively with parents to train them in better parent practices to shape the behavior of children. Indeed, much of this work was called behavioral parent training rather than family therapy.

More recent work by cognitive-behavioral therapists has been much more systemic. For example, Patterson and Chamberlain (1992) clearly explicate the reciprocal influence of child and parent in conduct disorder, and emphasize how therapists if they do not remain sensitive to client needs can engage in too frequent teaching behaviors that promote noncompliance with therapeutic tasks. Alexander and Parsons (1982) have added the systemic notion of function to behavior analysis. In their functional family therapy, attention centers on identifying the function of behavior, much in the manner of the Haley or MRI models. Once identified, behavioral interventions are introduced to accomplish this function in a less damaging way.

Cognitive-behavioral therapies are more similar to one another than other groupings of family therapies, such as strategic or intergenerational. It is a strength of these models that the work of each investigator builds on that of others. Even if models have different names and slightly different components, they are essentially similar in how to treat specific problems. Couples therapies are like

one another, as are treatments for children and adolescent problems.

Weiss (1978), Jacobson and Margolin (1979), and Stuart (1980) have all articulated similar approaches to couples therapy. Each approach begins with a behavioral assessment that includes the use of instruments to assess general levels of relationship satisfaction such as the dyadic adjustment scale. However, the primary focus of the assessment is on delineating problematic exchanges, specific target behaviors, and themes in the relationship that require change, evaluated through client recording of these behaviors between sessions, therapist observation of typical interactions, and clients completing self-report forms. The results of the assessment are directly shared with the couple, highlighting the areas in their relationship that require attention, leading to the development of a blueprint for change. Much of couple dissatisfaction is viewed as the product of the low level of positive reinforcement and high level of coercive exchange within the relationship, an often replicated finding in maritally distressed couples.

Many interventions are brought to bear to change these interactions. The monitoring of behavior, through tracking and sometimes including the use of videotape feedback, help couples to objectify their behavior, to see it from the perspective of an outsider. Where specific skills are lacking, skills training is employed to develop competencies. Typical skills training includes the development of communication skills such as attending, reflecting, listening, and speaking, and of problem-solving skills such as the abilities to define problems, generate alternative solutions, and reach naturally satisfying outcomes. Behavior exchange is specifically addressed through the development of contracts between the parties about these exchanges, most based in a *quid pro quo*, in which the behavior of one party is directly exchanged for the behavior of the other.

Behavioral couples treatments have a particularly strong record of demonstrating success in empirical studies, at least in short-term effectiveness (Lebow & Gurman, 1995). The great conundrum for behavioral couples therapy lies in addressing the aspects of the relationship that are not simply about behavior, those that involve feeling states, particularly love and caring. Behavioral couple therapists have therefore stretched the model to accommodate the obvious importance of this aspect of the relationship. In early formulations, some form of noncontingent loving behavior was prescribed, called "caring days" or "love days." Jacobson and Christenson (1996) have emphasized the importance of developing accepting

behaviors in addition to other skills. Other criticisms of behavioral couples approaches have more recently emphasized the lack of attention to history, meaning, and internal dynamics in the approach (Gurman & Knudson, 1978). The addition of cognitive theory and interventions have mitigated some of this criticism, but not all.

In sex therapy, a range of specific techniques for dealing with sexual problems are added to couple therapy. At times, interventions other than behavioral ones are included, as in Kaplan's (1979) widely circulated integrative model, but sex therapy always retains what primarily is a behavioral core. Much of this core, developed by Masters and Johnson (1970), derives from the well-demonstrated insight that anxiety is antithetical to sexual response and, that through classical conditioning, relaxation can replace anxiety. Sex therapy invariably includes the use of what are termed "sensate focus" techniques to induce relaxation. Other specific techniques are added which are specifically tailored to each sexual dysfunction. Sex therapy numbers among the most effective therapies in outcome studies, although the rates of success reported by LoPiccolo and LoPiccolo in 1978 are considerably lower than those reported by Masters and Johnson.

Much of the treatment of child problems in behavioral family therapy has exclusively focused on the parents in behavioral parent training. Given the theoretical orientation emphasizing reinforcement as crucial in behavior, and the large body of data available suggesting that the parents of problematic children help shape their dysfunctional behavior and respond poorly to it, many behavior therapists have concluded that time in therapy is best spent with the parents who control the reinforcers rather than with the children, especially when children are small. As in behavioral couple therapy, parent training begins with an assessment phase in which patterns of thought and behavior are recorded and connected to the target behavior of concern. This leads to a functional analysis of the problematic behavior, from which a plan is formed specifying the skills that need to be mastered and changes in contingencies that need to occur for the problem to be eradicated. Focus may center on caring behaviors as well as on establishing control. If the problematic behavior on the part of the child is restricted to a single area of concern, specific contingencies may be created in response to that behavior. For example, a program may be constructed of reward for school work. When problems are encountered in a number of areas, more comprehensive contingency programs are de-

veloped. Home token economies and point systems provide ways for credit to accrue for positive behavior and debits for problematic behaviors, with rewards dispensed for overall performance. In all programs, the preference for positive reward over punishment in shaping behavior is emphasized.

Although behavioral parenting programs have been very successful in helping parents to deal with a wide range of problems, they have not been sufficient to deal with more difficult problems. Here, the developers of these treatments have refocused their attention on the need to find ways to overcome resistance. Patterson and his colleagues (Patterson & Chamberlain, 1992) have highlighted situations in which therapist directives merely increase resistance, and sought ways to intervene that respond to client reactance. In a similar vein, Alexander and Parsons (1982) have directed functional family therapy for adolescent delinquent behavior toward ways of examining function and using that knowledge to enable engagement and cooperation in treatment. Further, in the work of groups led by Patterson, Alexander, and Henggeler, the behavior of the child or adolescent and the impact of peer groups and other relevant systems is accorded equal footing with the intervention with parents. These models assume a systemic perspective and resemble typical family therapy far more than parent training.

Behavioral family approaches to child and adolescent problems are among the most researched and validated of psychotherapies. Although the early variants of these approaches could readily be criticized for their simplistic analysis and failure to consider a range of important variables (e.g., history and meaning), recent work has been auspicious, leaving these among the most sophisticated as well as validated psychotherapies.

#### **1.16.6.4 Psychoeducation**

Psychoeducational approaches to the treatment of disorders are based on the notion that syndromes that seriously impair functioning have a biopsychosocial basis, and that illness models of these disorders are to be taken seriously. Sometimes illness models are fully incorporated as part of the approach, while in other variants they are examined with families as possible explanations for the disorder. Flowing directly from these ideas is the corollary that affected individuals and their families can gain a great deal from developing their understanding of the relevant problem and acquiring the requisite skills that best fit with



that particular problem. The goal is to establish a collaborative partnership with families who suffer from mental disorders, providing them with the kind of information and skills most needed. Information is viewed as a major route to coping. Beyond this constant, psychoeducational treatments include an eclectic mix of interventions derived from individual and family therapy that have particular relevance to the particular syndrome, as well as psychopharmacological interventions.

Psychoeducational family treatments were developed in the context of schizophrenia, where the treatments have been most extensively refined and tested, and extended to other disorders, most notably manic-depressive disorder. In the context of schizophrenia, a group at Western Psychiatric Institute in Pittsburgh (Anderson, Reiss, & Hogerty, 1986) and another at UCLA (Falloon, Boyd, & McGill, 1984) developed related, although somewhat different, psychoeducational methods. Each featured medication for the person with schizophrenia, along with education for the family. Each approach centered substantially around the notion that expressed emotion in families must be reduced. These approaches followed the extensive studies by Leff and Vaughn (1985), among others, demonstrating that when schizophrenics are exposed to much expressed emotion in families, consisting of criticism and high negative affect, high rates of recidivism and symptomology ensue.

Anderson et al.'s unique contribution lies in what they termed "survival skills" workshops, which over a full day, present the current state of knowledge about schizophrenia to families. These workshops seek to impart information, increase the sense of social support, and reverse the negative interaction families of disturbed individuals often have with mental health providers. Families are regarded as full collaborators and taught in these workshops both what is known and what is speculative about schizophrenia. The Anderson et al. (1986) family model also accented work in therapy designed to alter dysfunctional aspects of family structure, and a minimalist approach to intervention in sessions that included the schizophrenic, with one constant goal being to keep expressed emotion to a minimum.

The methods of Falloon, Boyd, and McGill (1984) also seek to involve family and reduce expressed emotion, but place greater emphasis on behavioral skills training. There also is a greater emphasis in this model on crisis management when the inevitable crises develop in the lives of these families. Both the UCLA and the Pittsburgh groups reported remarkable levels of outcome in sophisticated clinical trials,

which have been followed up with replication. With both methods, expressed emotion has been decreased in these families, compliance with procedures increased, and recidivism and symptoms decreased in the index patient. As such, this research suggests these treatments offer the most consistent cost-effective treatment for schizophrenia. This work has been followed up with similar procedures for families dealing with manic depression (Miklowitz & Goldstein, 1990).

One striking aspect of these models has been the inclusion of family in treatment in a way that has proved highly acceptable to these families, in contrast to earlier methods of dealing with these families that left many families blamed and highly dissatisfied. Much of this earlier work questioned the very existence of mental illness or even of disturbed internal processes in the schizophrenic. Some (e.g., Haley, 1976) even suggested that medication for the schizophrenic is harmful since it further establishes the patient in the sick role, and obscures what were regarded as the inevitable systemic issues. The strong evidence for the efficacy of psychoeducation, coupled with the lack of evidence that family therapy works in these samples without the use of medication, suggests that the psychoeducational form of family therapy is clearly superior to the earlier variety. Indeed, it appears clear that the highly stimulating family therapies of old frequently seen in the tapes of many masters of family therapy in treating schizophrenia provide exactly what is not needed: a highly stimulating environment likely to be difficult for the patient and an environment in which families are likely to feel blamed for the problem. All told, for schizophrenia and other severe mental disorders, psychoeducational treatments number among the most successful family therapies, and are becoming widely disseminated as the standard for care.

#### **1.16.6.5 Bowen Therapy and Other Intergenerational Approaches**

Bowen (1978) developed a prominent form of family therapy, now called Bowen family systems therapy, that incorporates systems theory, along with a unique language for considering the relationship of the individual with family. The crux of the Bowen approach lies in the concept of differentiation of self, which essentially amounts to the ability to distinguish thoughts and feelings. For Bowen, psychological and systemic health is a direct function of level of differentiation. When individuals differentiate themselves from family processes, they are viewed as less susceptible to

the pathology-inducing aspects of the system. Differentiation is clearly distinguished from cutoff, the establishment of rigid boundaries that minimize contact with family, which is viewed in this system as innately problematic.

In Bowen's theory, individual development is largely shaped by the family system. Bowen envisioned what he termed an "undifferentiated family ego mass," of beliefs and feelings within families that are transmitted through what he termed a "family projection process" across generations. The position of the individual in the family, in part determined by birth order and in part by other factors, is also viewed as of key importance in shaping the individual. A key element of family processes lies in the creation of triangles, in which the interaction between two individuals are affected by the presence of a third. Triangles are viewed as inevitable but also treacherous for individual development.

In Bowen family systems therapy, each member of the family involved is helped to increase their ability to manage their own anxiety. In individual differentiation lies the basis for better family relationships. Therefore, much of the work focuses on the relationships adult clients have with their families and, most specifically, their families of origin. Family of origin is typically not seen directly in treatment, but examined through forays outside sessions in which the client learns about family processes, experiences them, and finds new ways to cope with them. In this part of the therapy, termed coaching, the therapist helps the client develop a plan for investigation and examine the thoughts and feelings that emerge. Exploration involves both direct contacts with living relatives and efforts to learn about and experience feelings in relation to deceased family. Genograms, diagrams of the multigenerational family systems of participants, are employed to help in this examination, to shape exploration and set goals. Because this is the essential process of treatment, much of Bowen family systems therapy is conducted with only a single client in the office, although the work is principally centered on their family relationships.

Bowen family systems therapy represents a bridge between individual and family therapy. Although couched in systemic terms, many of Bowen's ideas about differentiation resonate with the concepts of object relations and cognitive models of therapy. Although much of the unique terminology developed by Bowen adds little (with a few clear highly salient exceptions such as "coaching"), Bowen developed a method that has enabled an exciting and moving voyage of exploration of family processes by innumerable clients. Although the clinical experience of many family therapists

suggests that this is a highly satisfying and effective treatment for clients, there has unfortunately been a paucity of research investigation of this approach.

There have been numerous other approaches that have centered on the kind of examination of intergenerational process developed by Bowen. Most prominent are approaches that create family rituals that serve as cathartic events for negotiating the emotional turmoil resulting from multi-generational legacies (Imber-Black & Roberts, 1993). Others have focused on explicating and adapting the family myths that have evolved.

#### **1.16.6.6 Psychodynamic Approaches**

Although psychoanalytic formulations have often served as the foil in expositions of several family therapies against which the value of focused systemic therapies could be highlighted, there has also been a long standing tradition of family therapies that have incorporated psychodynamic concepts (Reiner, 1997). Early in the history of family therapy, Ackerman, Boszormenyi-Nagy, Framo, Sager, Steirlin, and others created treatments that blended systems concepts with specific psychodynamic theories. More recently, the refinement of object relations concepts within psychoanalysis has led to the emergence of several family therapies that take object relations one step further, to consider those dynamics directly in the context of the family in treatment (e.g., D. Scharff & J. S. Scharff, 1987; Slipp, 1993).

Psychodynamic family approaches share a number of common characteristics, despite there being considerable variation with the particular psychodynamic formulation included. These approaches have roots in psychoanalytic concepts, but in all cases there is a significant transformation in the family context.

Most basic to psychodynamic formulations in couple and family therapy is the notion of an active dynamic internal process within individuals. Psychodynamic approaches share the belief that unconscious mental processes are extremely important and that early experience has a crucial influence on later behavior and experience. Psychodynamic family therapy has retained from its individual therapy counterpart an emphasis on the processes that occur within individuals.

Psychodynamic therapists also emphasize maintaining the frame of treatment, the formal arrangements such as frequency, time, and length of sessions. Creating an appropriate frame is viewed as leading to the development of a holding environment (D. Scharff & J. S. Scharff, 1987), in which the therapist tolerates

clients" anxieties and tensions while remaining empathic with their emotional experiences. The creation of a holding environment also allows for observations of deviations that arise from the frame. Another important route into unconscious process is the understanding of transference: the client's displacement or projections onto others of feelings, impulses, defenses, and fantasies from important past relationships or conflicts. These projections help recapitulate important aspects of clients' earlier relationships in therapy or in the family relationships. In psychodynamic family therapy, transferences are observed as much in relation to other family members, particularly spouses, as in relation to the therapist.

Psychodynamic couple and family therapies also accent the therapist's awareness of their own feelings in the therapy process. Countertransference, the therapist's reactions to the client based on client transferences or on the therapist's own personal experience, are viewed as important sources of information about client process. Most importantly, in projective identification, the therapist may be induced to feel or behave as the client has in the past or as others have behaved and felt toward the client. In most psychodynamic couple and family therapy, the understanding and owning of projective identification on the part of the client assumes an especially important place in the process.

Therapists offer interpretations that provide meaning to behavior by explicating unconscious processes. Change is seen as the product of working through one's issues over time, often as the product of these interpretations. Understanding resistance, the process, often rooted in anxiety, that moves against therapeutic goals, is also important in enabling change.

In many instances, the language and interventions of psychodynamic family therapy merely extends those of individual psychodynamic therapy and, most especially, object relations theory (e.g. D. Scharff & J. S. Scharff, 1987). Other family therapies begin with similar concepts, but draw on quite different methods, and language. For example, Boszormenyi-Nagy and Spark (1973) examine much of the same territory as an existential psychodynamic psychotherapy, but look at relationships in terms of what they term "invisible loyalties" rather than using traditional language in their contextual therapy. Their work aims at exploring multi-generational processes in families with an eye to what they term "ledgers," the balance of what has been given and received by each individual. The central tenet of the therapy lies in helping clients deal with and balance the ledger they bring from their families of origin.

The stance of the therapist toward the family, termed by Boszormenyi-Nagy and Spark "multidirected partiality," is also much like the hovering attention basic in more recent forms of psychoanalytic approaches, but carried over to the family context with new language and intervention strategies.

The work of Framo (1981) also draws heavily on the psychodynamic tradition, but adapted to the context of the family therapy. Framo developed what he termed family-of-origin sessions as part of couple or family therapy, in which the adults in the therapy would meet for a few sessions with members of their own family of origin, in order to understand better and resolve the outstanding issues that derive from that experience.

Although pure-form psychoanalytic therapies are relatively infrequently encountered in couple and family therapy, psychodynamic principles are central in the practice of many family therapists. These concepts have been highly influential, especially in their important place in a variety of integrative therapies. Many of the intergenerational and experiential family therapies also include a number of psychodynamic concepts. Psychodynamic therapies have rarely been evaluated through research. The demonstrated effects of a variant of psychodynamic therapy, insight-oriented couples therapy (Snyder & Wills, 1989), suggests the likelihood of a promising future for these treatments in research, should this research ever be carried out.

#### 1.16.6.7 Experiential Approaches

Prominent experiential couple and family therapies have been developed by Whitaker (Whitaker & Keith, 1981), Satir (1983), and Greenberg and Johnson (1988). Each of these approaches places the emphasis on felt experience, that is, in the therapy restoring liveliness and connection. In each, the primary instrument is the therapist, who uses self as an instrument toward change.

Each experiential family therapy employs different intervention strategies. Whitaker utilized a wide array of techniques, ranging from direct commentary to physically wrestling with clients, all aimed to fight emotional deadness. Satir employed exercises from the human potential movement with which she was so long associated, and Greenberg and Johnson utilize methods of focused interaction between couples derived from Gestalt therapy. Although there are relatively few family therapists trained in these specific methods, this work remains enormously influential in

highlighting the importance of the person of the therapist, and the need to maintain liveliness and authenticity in the work of couple and family therapy.

#### **1.16.6.8 Narrative Approaches**

At the time of the publication of this volume, narrative concepts have captured the greatest recent attention among family therapies. Michael White (White & Epston, 1990) has emerged as the major figure in the narrative movement. Other prominent figures include Anderson and Goolishian (1988), Combs and Freedman (1990), and Hare-Mustin (Hare-Mustin & Marecek, 1988). Their approaches vary in specifics, but all have roots in social constructivism (Gergen, 1981, 1991), the notion that knowing is socially constructed through language and discourse, and depends on the context of the observer. Each places a strong emphasis on thought processes and beliefs, and in each difficulties are envisioned as the product of stories that have been socially created and can be reconstructed. Problem-oriented descriptions are replaced by stories of accomplishment.

White emphasizes externalizing problems, that is, seeing them as separate entities from the individuals involved. Much like solution-oriented and MRI therapists, White also emphasizes the outcomes that occur when individuals have been successful in overcoming problems. Anderson and Goolishian (1988) offer the ultimate extension of this type of approach, fully replacing the notion of the expert therapist with the idea of therapist and clients as equal partners in conversation.

Rather than merely opening discourse, these approaches also accent the freeing of repressed voices. For White, following Foucault, the dialogue needs to be as much about overcoming societal oppression as about family processes.

Although sometimes these therapies do involve seeing families together, these therapies move far from the family emphasis of early family therapy. Much of the work is done with individuals, with only passing references to the family system. As yet, we also have little in the way of outcome research testing the effectiveness of these models. However, narrative models have already gained many proponents, and have influenced many more family therapists, in creating a frame for treatment that emphasizes a co-equal collaborative conversational style that de-emphasizes the therapist's role as expert, in emphasizing the importance of the client's voice, and in directly working with client stories about their lives to help create more workable realities.

#### **1.16.6.9 Integrative Approaches**

Integrative methods have become commonplace in couple and family therapy. Not only has a considerable literature emerged concerned with integration (Lebow 1984, 1987a, 1987b; Liddle, 1984; Grunebaum, 1988; Moulthrop, 1981, 1989), and numerous integrative models have been developed and widely disseminated (e.g. Feldman, 1985, 1990; Gurman, 1981; L'Abate, 1986; Pinsof, 1995), but the move to integration has become so much part of the fabric of our work that it largely goes unrecognized.

Integrative models merge the raw material of the various approaches. This merger occurs at three distinct levels: theory, strategy, and intervention. Because there are numerous therapies to merge, and several levels along which to merge them, integrative models vary enormously in content. Some integrative approaches accent each therapist's building of a personal method, while others offer highly prescriptive delineations of therapeutic ingredients and a specific map for when to do what. Moulthrop's work (1981, 1989), describing the therapist's personal odyssey, and Lebow's (1987a), suggesting guidelines for each therapist's model development, offer examples of conceptions focused on the therapist as an agent of integration. Jacobson's (1992) couple therapy and Liddle, Dakof, and Diamond's (1992) treatment for adolescent drug abusers and their families offer examples of highly specific prescribed routes to intervention, which can be specified in treatment manuals. Other models, such as Pinsof's (1983) problem centered therapy or Gurman's (1981) integrative marital therapy, bridge this chasm through prescribing ingredients, but allowing varying levels of room for improvisation, especially for more advanced practitioners.

Most integrative efforts combine behavioral notions of learning, with a systemic understanding of the family process, and the individual psychodynamics that are brought to bear in these patterns (Feldman, 1985, 1990; Gurman, 1981; Kirschner & Kirschner, 1986; Nichols, 1987, 1995; Pinsof, 1983, 1995; Sander, 1979; Wachtel, 1992, 1995; Wachtel & Wachtel, 1985). Pinsof's (1983) "Problem centered therapy" offers a highly refined version of this type of model, in which self-psychology is the internal system. Gurman (1981, 1992) has developed a combination of object relations, behavioral, and systemic procedures for working with couples. Feldman (1990) adds an integration of similar ingredients envisioned as linked in a multilevel circular process.

Much of the creative edge in integration has been concerned with the development of specific treatments for specific populations. Goldner et al. (1990) have merged feminist, narrative, systemic, and psychodynamic concepts in the treatment of abuse within couples. Liddle et al. (1992) and Piercy and Frankel (1989) have brought structural, systems, and behavioral principles together along with a developmental perspective in the treatment of adolescent chemical dependency. Similarly, Kaplan (1974) has brought an integrative approach to sex therapy; Addis and Jacobson (1991) to adult depression, Rolland (1994), Wright and Leahy (1994) and Wood (1994) to families with physical illness; Wachtel (1995) to families with young children; Steinglass (1992) to alcoholism; Alexander and Parsons (1982) to adolescent acting out; Markman (1979) to premarital couples; Trepper and Barrett (1989) to sexual abuse, and Harkaway (1989) to eating disorders. Each of these methods provides a hearty integration, that includes theory, methods of intervention, and particular values that guide treatment.

Feminists (Goldner, 1985; Hare-Mustin & Marecek, 1988, 1990) and those who offer treatment in diverse cultures (Aponte, 1985; Boyd-Franklin, 1989, McGoldrick et al., 1988) have focused attention on the obvious importance of race, class, and gender, and on the value of therapists' shaping treatment in relation to these factors. This has resulted in the development of several integrative treatments, specifically designed for particular cultural groups or gender-related issues (Boyd-Franklin, 1989; Goldner, 1985; Szapocznik & Kurtines, 1989; Watts-Jones, 1992). These models move beyond the notion of one method for all to a better understanding of which methods work best in what combination with various populations. Culture and gender have also been incorporated as anchors in some broader efforts at integration (Breunlin et al., 1992). The feminist and cultural perspectives have also helped elucidate the underlying assumptions about gender and culture within treatment models, leading to a more informed discussion of what is being integrated.

It should be noted here that even though feminist family therapy presumes an integration of feminist ideas and methods of family therapy, feminist work has seldom been labeled as integrative. Nevertheless, feminists have provided some of the richest integrative frameworks; many models include a wide range of precepts and interventions. Similarly, the integrative nature of the models of those working with specific cultural groups often goes overlooked in considerations of integrative treatment models.

As yet, research has only assessed a small number of these integrative treatment models. Nonetheless, the results of the existent research are very promising. The integrative methods studied have proven highly effective at dealing with a wide range of problems.

### **1.16.7 THE EFFICACY OF COUPLE AND FAMILY THERAPY**

Three decades of research have confirmed and reconfirmed the effectiveness of couple and family therapy. Reviews of the literature conclude that the outcomes achieved by treatment groups have exceeded those of control groups (Gurman & Kniskern, 1978, 1981; Gurman et al., 1986; Alexander et al., 1994; Jacobson & Addis, 1993; Baucom & Hoffman, 1986). With more and better research emerging the evidence for overall effectiveness has become unequivocal (e.g., Jacobson, Dobson, Fruzzetti, Schmaling, & Salusky, 1991; Snyder & Wills, 1989). Although the majority of studies have focused on behavioral treatments, there is now also a considerable base of nonbehavioral treatment studies that point to treatment efficacy. Further, the earlier stated concerns about possible confounds of treatment method that might mitigate this conclusion (Beach & O'Leary, 1985; Gurman & Kniskern 1978; Bednar, Burlingame, & Masters, 1988; Lebow, 1981) can now be put to rest. A range of excellent methodological studies confirms the general findings of efficacy found in less rigorous research.

Nonetheless, there is clearly a differential amount of evidence supporting the efficacy of the various couple and family therapies (Lebow & Gurman, 1995). Behavioral approaches largely have extensive bodies of research support, particularly in treating childhood and adolescent conduct disorder, marital dissatisfaction, and adolescent acting out. Structural approaches also have considerable support, particularly in child and adolescent conduct disorder. An experiential approach to couple therapy, emotionally focused therapy (Greenberg, Ford, Alden, & Johnson, 1993), and a psychodynamic approach, insight-oriented (Snyder & Wills, 1989), also have garnered research support. A considerable body of research evidence points to the efficacy of treatments that integrate individual and conjoint treatments, including psychoeducational treatments of schizophrenia (Hogarty, Anderson, Reiss, Knornblith, & Greenwald, 1986), cognitive-behavioral treatment of depression (O'Leary & Beach, 1990), multisystemic treatment (Henggeler, 1993), and functional family

therapy for adolescent delinquent behavior (Alexander & Parsons, 1982), and multidimensional family therapy for adolescent drug abuse (Liddle, Dakof, & Diamond, 1992). There exists very little research support for a variety of widely practiced couple and family therapies including Bowen, narrative, humanistic, strategic, and solution-focused approaches. Differences in knowledge about models are even more pronounced than earlier (Gurman et al., 1986).

Looked at from the perspective of the presenting problem, couple and family therapies have been demonstrated to have considerable value in treating depression, anxiety disorder, panic disorder, schizophrenia, alcoholism and marital maladjustment in adults, conduct disorder, autism, and drug abuse in children and adolescents (Lebow & Gurman, 1995; Pinsof & Wynne, 1995). Most of these demonstrations of effectiveness include a combination of couple and family therapy with other interventions. Offered alone, couple and family therapies emerge as the only demonstrated effective means for impacting on couple and family issues (e.g., couple conflict and marital maladjustment). Frequently it appears that even a small amount of family involvement adds immeasurably to treatment effectiveness and increases acceptability and participation in treatment (Lebow & Gurman, 1995). There are indications that treatments diminish in their effectiveness over time. Although outcomes are quite impressive in the short term, effects often dissipate (Lebow & Gurman, 1995).

An outstanding body of research is now also available associated with family process and family development (Cox & Paley, 1997). This work implies many hypotheses for treatment research. For example, Gottman and associates have carried out several studies that have added immeasurably to our knowledge of patterns and sequences of dysfunction in marriage (Buehlman, Goldman, & Katz, 1992; Gottman, 1991, 1992, 1993; Gottman & Krokoff, 1989), such as showing particular patterns in couples conflict that directly lead to decreasing levels of marital satisfaction and ultimately to divorce. Similarly, powerful bodies of research concerned with such issues as patterns in divorcing and remarried families (Bray & Hetherington, 1993), family transitions around the birth of children (Cowan & Cowan, 1992), and patterns in the alcoholic family (Steinglass, Bennett, Wolin, & Reiss 1987) have clear clinical implications.

A few other trends in the research that have emerged are particularly noteworthy. We are seeing a trend toward more clinical trials research comparing treatments. Paradoxically, research also shows the impact the intense study

of process can focus on a few cases, particularly when the cases are selected by outcome status and the methods for assessing process clearly focus on change events rather than engaging in a hunting expedition (Greenberg, 1991). We also are beginning to see a great deal of research on treatments that transcend the labels "individual," "couple," or "family" therapy, just as clinical methods are moving to transcend these boundaries (Lebow, 1987a, 1987b). We are also seeing more efforts to be conscious of gender and culture in research. No longer is the assumption made that findings are necessarily generalizable across gender or culture. Perhaps most promising, family therapists and family researchers have begun to engage in dialogue, suggesting that the notable gap between research and practice may narrow.

### **1.16.8 TRENDS IN COUPLE AND FAMILY THERAPY**

A number of trends are clearly emerging in family therapy. These include:

(i) Most family therapies have moved away from radical systemic positions, which focused solely on interpersonal processes and viewed the etiology of disorder, treatment, and the assessment of change exclusively in terms of the properties of systems (e.g., circular causality and homeostasis). These radical systemic viewpoints showed little respect for any linear epistemology, including the traditional methods of empirical research, and focused attention on a complex set of variables that were extremely difficult to measure. A shift has occurred in which interpersonal processes, feedback loops, and the system's role in problem generation and resolution are still emphasized; but attention broadens to include individual functioning, the larger social system, and other levels of analysis.

(ii) The boundaries among individual, couple, and family therapy and between schools of family therapy are blurring. There are more and more integrative therapies that include couple and family treatment. Many therapies that include significant aspects of systems theory and interventions with couples and family do not label themselves "family therapy" (Pinsof & Wynne, 1995).

(iii) Methods of practice in family therapy are becoming more generic. Therapists practice in ways that are more integrative, and utilize strategies and interventions that cross a range of schools of practice. Concepts like therapeutic alliance (Pinsof & Catherall, 1986), loss (McGoldrick & Walsh, 1983), life cycle (Carter & McGoldrick, 1988), cohesion (Olson, 1986), isomorphism (Liddle, 1984); macrosystem

(Imber-Black, 1988), and differentiation (Bowen, 1978); interventions such as enactment (Minuchin & Fishman, 1981), ritual (Imber-Black & Roberts, 1993), and genogram (McGoldrick & Gerson, 1985); and tactics for dealing with such processes as engagement (Minuchin & Fishman, 1981), resistance (Anderson & Stewart, 1983), and termination (Lebow, 1995) are used by a wide range of family therapists, transcending the approach in which they were first utilized. We have also seen the development of a common language that transcends approach (Simon, Stierlin, & Wynne, 1985), and the beginnings of catalogs of interventions that transcend orientation (Figley & Nelson, 1989; Minuchin & Fishman, 1981).

(iv) Biology is clearly emerging in the literature as an important variable in family life, in such disparate contexts as conflict in marriage (Gottman & Levenson, 1992) and processes around schizophrenia in the family (Leff & Vaughn, 1985). Family reactions to crises in health (Rolland, 1994) and, more broadly, the field of family systems medicine have come to occupy much attention.

(v) Many models now feature a very strong cognitive emphasis on intervention strategy. Behavioral models have added cognitive theory and intervention, strategic models a heavy emphasis on reframing, and narrative models fully focus on the creation of new stories about events.

(vi) There is a growing tendency to develop specific variants of treatment to respond to the specific issues in particular kinds of cases, especially different diagnostic groups. Such treatments as psychoeducational intervention for schizophrenia (Hogarty et al., 1986), cognitive-behavioral couple treatment of depression (O'Leary & Beach, 1990), multisystemic treatment (Henggeler, 1993) and functional family therapy for adolescent delinquent behavior (Alexander & Parsons, 1982), and multidimensional family therapy for adolescent drug abuse (Liddle et al., 1992); all are fully structured and adapted to the specific population in focus.

(vii) Much attention has been focused on the meaning and methods for intervening in family violence (Avis, 1992; Goldner et al., 1990). In attempting to deal with family violence, many assumptions of systems theory have required modification, for example, the absolute centrality of circular causal explanations.

(viii) As previously noted, gender and culture have emerged as vital considerations in shaping practice. No longer is the assumption made that the same treatment will be appropriate across varying groups (McGoldrick et al., 1988). Patterns of behavior and expectations

vary and generalizations about family process must always be viewed through the lenses of gender and culture.

This focus on gender and culture has taken two forms. Some have offered critiques of the concepts of family therapy, explicating the ethnocentric and gendered aspects of the most widely taught models of treatment. For example, Green and Werner (1996) have focused on the tendency to view closely connected families, frequently found in many minority cultures as pathologically enmeshed. Green and Werner argue that closeness can be and often is a positive value. Similarly, Goldner (1985) has described the gender assumptions implicit in most schools of family therapy. The second tact has been to explicate specific methods that fit with the culture of particular groups of individuals (McGoldrick et al., 1988) or powerful gendered issues (Carter, 1996; Goldner et al., 1990).

(ix) Others have become increasingly concerned with the impact and relationship of systems larger than the family. Early family therapy focused on the family and its dynamics. More recent thinking has viewed the family as only one of multiple systems impinging on the individual. The family system is viewed in relation to a number of other influences, such as schools, the legal system, poverty, and peer groups. Multisystemic treatments targeting multiple systems are beginning to emerge (e.g., Henggeler, 1993; Liddle et al., 1992). Some family therapists (Wynne et al., 1986) now even refer to themselves as system consultants.

(x) An increasing emphasis in family therapy has been on a life cycle perspective (Carter & McGoldrick, 1988). It has become typical to consider a family in relation to where they are within the developmental process, and shape interventions accordingly.

(xi) There is much more emphasis than earlier in family therapy on health, normality, and resilience, as opposed to homeostasis, resistance, and pathology (Walsh, 1993). Not only do the specific approaches that have captured the most recent attention feature these assumptions (e.g., solution focused and narrative), but most family therapy now emphasizes resilience and coping.

### 1.16.9 THE DEVELOPMENT OF FAMILY PSYCHOLOGY

From its beginning, couple and family therapy has been a multidisciplinary endeavor. Perhaps more than in the development of any other psychotherapy, psychologists, psychiatrists, social workers, psychiatric nurses, and

others (even including anthropologists and sociologists) have worked together in harmony. Indeed, many of the central developments in the field have been carried out in family institute settings, away from the organized centers of training in specific disciplines.

The downside of this pattern of development has been that it has taken considerable time for family therapy to occupy a central place within the specific disciplines, including psychology. Well after the emergence of family therapy as a much practiced endeavor, few training programs within the mental health disciplines offered training in systems therapy. For example, Stanton (1976) reported that as late as the mid-1970s, only 10 psychology programs offered family therapy training. However, since the mid-1970s, this pattern has substantially changed, as structures have evolved in training and practice that recognize the importance of the family system in psychology.

Foremost has been the creation of a sub-discipline, family psychology. The American Psychological Association has recognized the importance of family psychology in creating the Division of Family Psychology (Division 43) and a journal to represent scholarship in this discipline, *Journal of Family Psychology*. In surveys, 63% of psychology programs with special treatment services had family therapy clinics (Sayette & Mayne, 1990), 19% of all faculty had systemic theoretical orientation (Mayne, Norcross, & Sayette, 1994), and family research was the second most represented area of research across departments (Sayette & Mayne, 1990). Eighty-five percent of psychology internships reported some marital and family training (Soloman, Ott, & Roach, 1986). In many departments, a specific specialization in family psychology has been created (Green, 1996). In addition, many psychologists continue to receive training in postdegree programs in family institute settings.

As this is being written, discussion is underway about whether family psychology should be regarded as a specialization on a par with clinical and counseling psychology or whether it should be regarded as a proficiency lying within clinical psychology. Green (1996) has argued cogently for the former, suggesting that "there is nothing about clinical or counseling psychology that makes them inherently more generalist than a family psychology program would be." Green further suggests the creation of a full predoctoral specialization in family psychology as well as a postdegree specialization. Whatever the outcome of this debate, it is clear that family psychology has moved to a central place within psychology, just as systemic therapies have become among the most widely practiced

therapies, and as couple and family intervention has come to be seen as essential in the treatment of a wide array of problems.

### 1.16.10 SUMMARY

Systems formulations of family process and family systems orientations to psychotherapy have been prominent methods of conceptualization and intervention since the early 1960s. A variety of distinct forms of couple and family therapy have developed and become widely practiced, including structural, strategic, experiential, inter-generational, cognitive-behavioral, and psycho-educational methods. Approaches to couple and family therapy are increasingly integrative, incorporating a range of concepts and intervention strategies, and including a focus on the individuals within the family as well as on the broader social system in which the family resides. In the future, we are likely to see the further development of such therapies that feature a systemic orientation, but which are less distinctly "family" therapies, in which therapists move their focus across levels of social system: from family, to couple, to individual, to the larger system.

Knowledge derived from research about the family system and family therapy also is growing. Much of this research supports the tenets of systems theory as an explanatory set of concepts (Cox & Paley, 1997). This research is also helping to build a base of greater understanding of family processes. Research assessing the efficacy of couple and family therapy has consistently demonstrated its efficacy. Specifically, couple and family therapy appears to have a unique impact in alleviating relationship difficulties, to contribute in important ways in the multimodal treatment of psychopathology, and to help engage many clients who cannot be engaged in other forms of treatment.

Family psychology is emerging as a prominent field within psychology. In the twenty-first century, we are likely to see it occupy an even more central place within the discipline, with greater formal recognition of the specific skill base needed to work with couples and families.

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