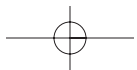
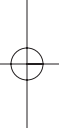
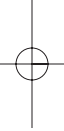
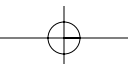
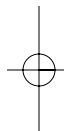
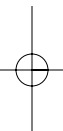
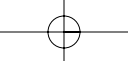


PART ONE

DEVELOPMENTAL SCIENCE

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CHAPTER ONE

THE SCIENTIFIC BASIS OF MENTAL HEALTH INTERVENTIONS IN CHILDREN AND ADOLESCENTS: AN OVERVIEW

Dr. med. univ. Hans Steiner

"Granted that mental processes are caused by brain processes, what exactly are these mental events? . . . Mental states are simply higher level features of the brain . . . a feature of the entire system, but not a feature of the microcomponents of which the system is composed."

JOHN SEARLE (1994)

". . . The brain is the organ of the mind, the organ of speech and thought, the organ of politics and of human individuality."

GERALD EDELMAN (1994)

"Introspection cannot teach you a thing about the brain as a physical object, even though consciousness is a property of the brain, and outer perception cannot give you any access to consciousness, even though consciousness is rooted in the observable brain."

COLIN MCGINN (1999)

"The barrenness and confusion of psychology (and psychiatry) cannot be explained by the fact that it is a young science . . . its lack of progress is not explained by the fact that it is comparable to physics in its beginnings. For in psychology (and psychiatry) we have scientific methods, but conceptual confusion."

LUDWIG WITTGENSTEIN (1953)

This chapter and the thirty-sixth are bookends for this entire *Handbook*. In this section and its overview, we will put forth what we think are the most salient developments in the sciences underpinning mental health interventions in children and adolescents. The purpose is not to provide a complete discussion of scientific

developments in the mental health sciences, which would be beyond the scope of this volume. Instead, we are providing a snapshot of those areas of scientific inquiry, which at the present time are most relevant to the practitioner. The intent is to orient the practitioner to progress, which has influenced or is about to influence mental health interventions.

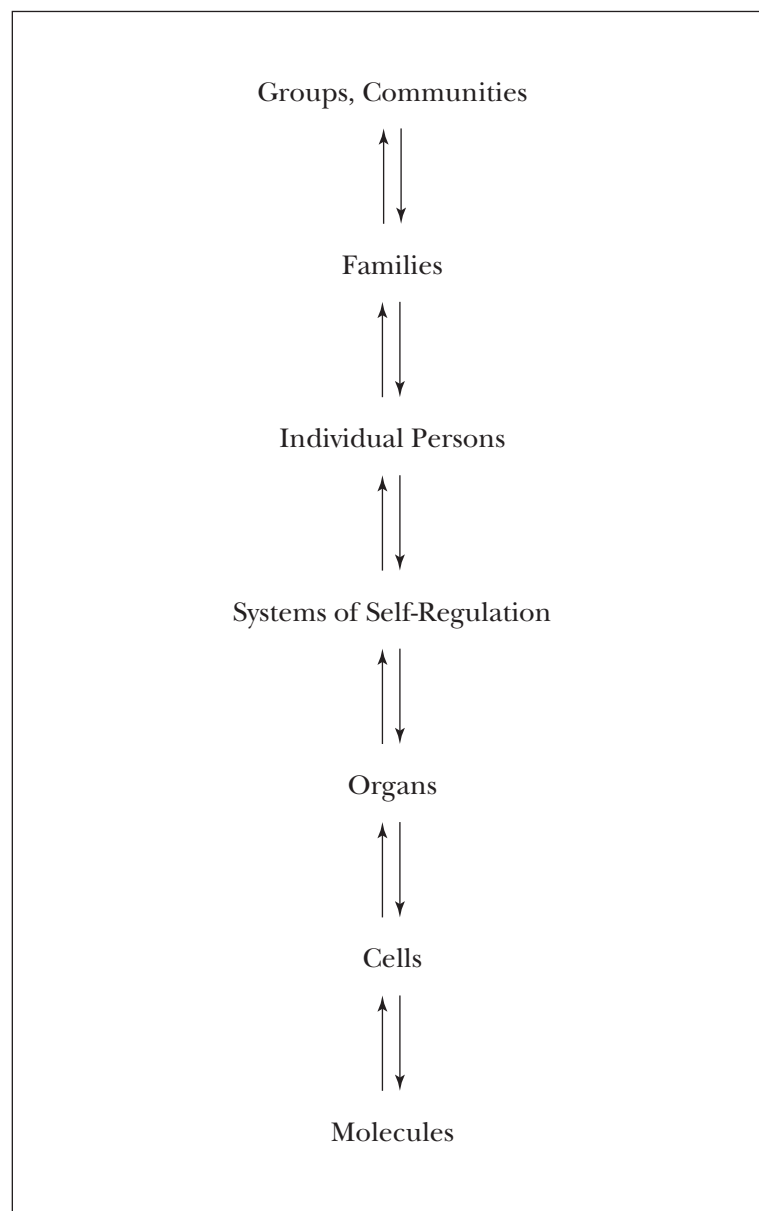
Psychiatry has always straddled uneasily the social and biological sciences. At different points in the development of our discipline, we have emphasized and de-emphasized this. Currently, we are in a period of mental health sciences and their applied disciplines where reimbursement pressures and the enormous advances in neuroscience are synergistically creating a subtle or not so subtle pressure to reductionistic forms of conceptualizing psychopathology. Such pressures also affect treatment: we are supposed to do more with less, and rely predominantly on medications to treat. We think this development will not serve our patients and our professions well. This *Handbook* is intended to be an effective antidote to any such pressures. By giving mental health practitioners access to the full range of exciting developments in all the fields relevant to our practice, we hope to give incentive to practitioners to carefully examine the best available, not just the cheapest interventions. Premature orthodoxy, as we witnessed in the case of psychoanalysis in the 1950s, is also ill advised for the biological sciences today as they contribute to solving the puzzles of mental health and its disorders.

Figure 1.1 represents an array of levels of abstraction and organization relevant to current clinical practice. Psychopathology, at our current level of understanding, can be caused, precipitated, and maintained by problems originating from any of these levels of abstraction, and interventions targeting any one of these levels can be helpful.

At our current stage of knowledge, we have to embrace this entire range of sciences more explicitly. Additionally, practice needs to be informed by the laboratory. And the laboratory needs the feedback of practice. Science proceeds by the valid experiment, which may tell us little about a particular patient. Practice proceeds by the controlled clinical trial, which may not tell us much about a disease process. In contradistinction to a weak bio-psycho-social juxtaposition advocated by some, we advocate for a strong integration of these sciences and practice on the basis of developmental principles to guarantee continued and rapid progress (Steiner & Hayward, 2003).

How do we select the sciences that are most relevant for this section? In order to do so, we must describe a model, which defines the boundaries of what is relevant and what is not. For the purposes of this book, we use the developmental model of psychopathology. For reasons that should become clear this model has the most promise to be helpful because it most closely reflects the realities a given case represents and the dilemmas a given clinician has to struggle with. We first describe the essential features of the *developmental model*, then discuss briefly the

**FIGURE 1.1. LEVELS OF ABSTRACTION AND ORGANIZATION
RELEVANT TO MENTAL HEALTH SCIENCE AND PRACTICE
FROM THE VIEW OF DEVELOPMENT.**



selected areas of scientific inquiry and their most poignant lines of research pertinent to clinical practice. Finally, we outline the principles of evidence-based practice against which any of the following chapters outlining a particular treatment are to be judged in terms of how solidly they are backed up by empirical and clinical facts.

The Developmental Model of Psychopathology

In the past 100 years, the mental health sciences have seen the rise of several developmental theories, as they apply to special domains of functioning (Crain, 2000; Jones & Elcock, 2001; Thomas, 2001). These also have been critiqued in an excellent compendium by Miller (1992). The “big” ones deal with cognition (Piaget, information processing; see Miller, 1992), psychopathology and the human life cycle (Freud & Erickson; see Miller, 1992), learning and adaptation (behaviorism and social learning theory; see Miller, 1992), and animal models of adjustment and adaptation (Bowlby, Lorenz, & Tinbergen; see Miller, 1992). From these theories, we can distill certain characteristics, which are core to a developmental approach, and apply them to our model of psychopathology and treatment. The following list summarizes these characteristics.

The Essence of the Developmental Approach

- Captures change (qualitative/quantitative)
- Builds complexity (systematic, molar, and molecular)
- Examines context (temporal and social)
- Looks for continuities/discontinuities (normality/pathology; isomorphisms/non-isomorphisms; causal process oriented)
- Historical: pathways and trajectories of change (person + disorder)
- Captures adaptive/maladaptive function (outcomes)
- Is narrative: synthetic across domains (unit of observation = person)

The developmental model of psychopathology first and foremost captures *change* and is interested in defining changing entities. This contrasts sharply with the description of static disease entities. Children and adolescents are involved in dramatic progress and alterations of their competences and levels of abilities at rates that are not paralleled in any later period of humans. This is especially true for adolescence (Steiner, 1996). Change in emotions, cognition, motor skills, and multiple other domains is evident in every individual and poses special demands on what counts as abnormal. Because so little stays the same over time, there is always a question as to whether something is normal or abnormal, a central question to

the enterprise of psychopathology. Another question, which the model attempts to address, is how change occurs: in large quantum leaps or in slowly accumulating doses? Quantum leaps can be more easily mistaken as pathology, as the new state seems to appear out of nowhere and is so different from what was just true. A more gradual and slowly accumulating alteration is much less suspect and can be deceptive regarding the severity of the problem it poses.

Inherent in the concept of developmental change are two related concepts: change is in a certain *direction* and occurs in more or less discrete *phases*. Change is usually in the direction toward differentiation and complexity (Werner, 1948) when things go well. This is not the change of entropy, where organization becomes less; it is in the direction of more organization. Change is the product of internal organizational forces, which direct the final outcome. In interaction with experience, though, inherent programmed potential becomes what it appears to be in real life. Implicit in this idea is the concept that even when left to its own devices, the growing organism has a trend toward righting itself, provided conditions are supportive of that, even without intervention. The context is not just important to determine phenotype; it also provides a protective envelope while the disordered person is regaining equilibrium.

As mentioned, also implicit in the concept of internally driven change toward health is the idea that in order for people to become what they are, there is a necessary interaction with the environment. Contextual variables shape the internal program. Psychosocial experiences interact with genetic programs to produce phenotypes. This is true for normal progress, and for the development of psychopathology as well. The model is inherently interactionist, weighing internal, intra-individual variables as much as external forces in the causation, precipitation, and maintenance of psychopathology, as well as in its conceptualization of treatment. This is an important consideration that has implications for the design of interventions aimed at children and adolescents who suffer from psychopathology. The context in which they operate is crucial to their functioning and determines not only how they become disordered, but also how they become well. The psychosocial environment in all its own richness is not a coincidental nuisance variable, but a necessary ingredient for the cause of psychopathology and its treatment. Maybe cancer can be treated in an interpersonal vacuum, but very few mental disorders, especially those in children and adolescents, can.

Change in response to intervention is also understood in more complex ways than in a simple disease model. Because we expect that there will be internal organization and internal forces, which drive the person in a certain direction, we would not expect that response to intervention would always be rapid and uncomplicated. The individual has its own internal set of processes, which equilibrate his or her adjustment. These processes can be cognitive, emotional, neuronal,

interpersonal, or otherwise. They are to be taken into account when we apply treatment. Progress in treatment will usually not be linear, but more like a series of equilibrating steps. Repeated applications of interventions are required to move an individual along the path toward healing.

The concept of developmental *phases* is helpful for scientists and practitioners to track what counts for normal and abnormal, and it also occupies a central position in the definition of psychopathology from the developmental perspective (see below, and Mash & Barkley, 1996; Wakefield, 1992). Different phases of development have been described throughout the literature. Unlike in lower level organisms, these phases are often less well defined and contain much overlap. They are helpful though, from a clinical point of view, in constructing multivariate templates for associations to be expected, competencies present or absent. They, therefore, determine the kind of targets present and types of interventions to be planned.

Table 1.1 outlines one possible array of developmental phases we have found quite useful in our practice. It lists the stages of psychosocial development and the critical tasks to be achieved in that particular period. There are many other tasks

TABLE 1.1. CLINICALLY RELEVANT STAGES OF NORMAL DEVELOPMENT.

Stage of Psychosocial Development	Critical Tasks to Be Achieved
Infancy	Attachment, control over bodily functions, locomotion, speech
Preschool	Separation and expansion of interpersonal orbit, exploration, play as an instrument of knowledge
School-age	Acquisition of skills and knowledge, book-based learning, extra-familial relationships with authority and same-sex peers
Early Adolescence	Pubertal maturation, independence, establishing diverse peer group, exploring sexual components of peer relationships
Late Adolescence	Exit from family, advanced learning, search for partners, managing sexual component of relationships, establishing health habits
Young Adulthood	Establishment of partnerships and family, early career development, refining health habits
Middle Adulthood	Establishing and maintaining a career, growth of own family, mentoring and generativity, maintaining health habits

not listed, pertinent to other domains of functioning, which cannot be listed. The reader is referred to a more complete rendition of this model in Steiner and Hayward (2004).

Implicit in the notion of developmental change is the idea of building *complexity* (Werner, 1948). This means that the model explicitly embraces the concept that our mental functioning becomes more diverse and complicated as we age. Our thinking, emotional life, actions, abilities, and competencies change under the impact of us interacting with our environment. We are learning. We become also more differentiated, specialized, and capable. Psychopathology can manifest itself therefore not only in simple deficits (as a disease model would postulate), but it also can show up as lack of progression and differentiation (a developmental arrest, a “fixation” in analytical language), or as a precocious assumption of complex functions that are not commensurate with what is expected from us, and finally, a loss of complexity of a domain under duress, which can be reversed by appropriate intervention (for example, removal of what impedes progress).

Let us elaborate on a theme which we have briefly touched on above, the importance of *contextual variables* in determining normality, psychopathology, and treatment. Intervening with youth quickly teaches us one thing: humans (especially immature humans) are extraordinarily dependent on their psychosocial context to achieve adaptive functioning and mental health. This has been elegantly discussed and described by Bronfenbrenner and Ceci (1994) and multiple others since then. Social learning theory (Bandura, 1997) deserves perhaps the most credit in calling our attention to the fact that maladaptive behavior not only is sometimes caused by factors in the psychosocial environment, but also precipitated and maintained by the environment. It is not just the individual that needs to be changed in order to allow a child to return to normal functioning. These environmental influences can be so powerful that interventions at the individual level are weak and ineffective despite all best intentions. Even more importantly, in some forms of psychopathology, the adaptive value of the particular problematic mental state or behavior in the repertoire of a given person is such that they will not change it despite the most dire consequences, but rather seek out psychosocial contexts, which more easily accommodate the characteristics in question. A good example: the psychology of the antisocial child, who associates with antisocial peers rather than giving up antisocial exploits. Humans not only carry the results of their deficient interactions with certain environments within them (the definition of psychopathology), but they also appetitively seek out new environments which are in accord with their internal expectancies. Psychopathology has a self-perpetuating quality, which makes it unique in the realm of medicine and the healing arts. Unless one is prepared to encounter this quality and deal with it appropriately, the best laid foundations for recovery will be in vain.

The developmental model also looks for *continuities* across time. As the model deals with rapid change, it cannot be primarily phenomenologically based. Because things can be so different between two ages, yet represent the same phenomenon, the developmental model has to look beneath the surface, and has to attempt to describe what the causal process is that ties together different manifestations of the same psychopathology. The developmental model is intrinsically process oriented, and in that sense pragmatic and close to treatment. Treatment focuses on causal processes that need to be changed to restore normal adjustment. The model's basis in *emergent complexities* admits a range of causal processes in psychopathology, a position that is well suited for our current level of understanding how children become mentally disordered, and how they recover, and what they need to do in order to get well. We will see below that criteria for evidence-based practice demand that one identify processes to be targeted and that such processes play a central role in establishing the efficacy of an intervention.

The model, in looking for continuities, will also acknowledge discontinuities and incomplete manifestations of pre- or post-syndromal psychopathology. This is an important link to other sciences, basic to the mental health domain: epidemiology (Steiner & Hayward, 2004). The study of populations is necessary and important to define the abnormal and the normal. It also defines specific risks and protective factors, which hinder normal development and hinder normal adaptation. Such interests also focus the developmental mental health clinician on early intervention, prevention, community practices, assisting other systems that deal with youth in identifying those who are about to become ill, and helping the system avert the problem by using community-based interventions. The link to public health and systems, which routinely deal with juveniles, is an important one. The mechanisms and strategies, which are effective in influencing these systems either as a consultant or as a participant, are a vital part of developmentally based practice.

Because the model is interested in continuity and phases of growth and influence, the system is also *historical*. The developmental model is interested in pathways and trajectories. This attitude goes well beyond the "history of present illness" found in mainstream medicine, in that it goes beyond a mere symptom tally. It also goes beyond the customary exploration of familial clusters of disorders. Of importance are the special attributes and characteristics of social environments encountered on one's way to maturity, their ability to facilitate growth, and their tendency to impede it. The person's pathways through time are examined in great detail, as they also offer information about the individual and his or her past adaptive success. We chart progress achieved in various domains of functioning (interpersonal, academic-vocational, recreational, and basic biological, just to name a few). We note the successful achievements of each phase

and the critical tasks associated with it. And we chart failure of progression even in the absence of symptoms, because such failure is included in the expanded developmental definition of what counts as psychopathology. We will discuss this further.

Because the model is historical, it concerns how a particular individual reconstructs the pathways under discussion. The model is oriented toward personal *narratives*, in that it pays a great deal of attention to how a particular patient describes his or her life and understands it. Our narrations are full of metaphors we live by (Lakoff & Johnson, 1980). In turn, these metaphors determine our reactions and actions. This reconstruction is important for several reasons. On the most basic level, we get a feeling for the more extended and habitual level of functioning. As patients describe these pathways, we appreciate their way of understanding how things go right or wrong in their lives, and what they see as their strengths and weaknesses as people—not just their deficiencies as patients. Building an array of strengths in addition to tallying symptoms is an important preparatory step in setting up interventions. Secondly, the narrative of their lives helps us delineate to what extent patients see themselves as responsible for what is happening to them. Such beliefs often play a central role in the perpetuation of psychopathology, such as in post-traumatic stress disorder (PTSD) (Steiner, 1996). We also get a measure of how the patient assigns blame and culpability to others. This may be particularly important in cases of externalizing disorders. Such an understanding also will help us anticipate roadblocks to treatment in the form of non-compliance and resistance to suggested interventions. Finally, from a pragmatic point of view, their understanding of what happened and what life events intervened, how they relate to what went wrong and what factors hindered and helped their adjustment become a cornerstone for the formulation of a particular case, which subsequently drives the composition of their treatment package. Such a formulation has to take into account an empathic understanding of the patient's perception of the situation in order to maximize the chances that the recommended interventions will be followed and carried out appropriately. This narrative will prepare us for the formulation, including the synthesis of all relevant material in a particular case to a person-specific scenario of how this patient became ill now, what forces put and keep him or her there, and what it will take to get his or her life back on track again. The model forces this synthesis across domains, thus preparing us well for a treatment plan and interventions that stand the greatest chance to be effective.

Having outlined the specific features of the model, we now can turn to a new definition of psychopathology from this particular perspective. Psychopathology is a “harmful dysfunction” (Wakefield, 1992), that is, a “condition that causes harm or deprivation of benefit as judged by social norms (and evolutionary purpose).”

Psychopathology is a process (a dysfunction, not just a tumor) that is harmful to an individual. The process is not just defective; it is dysfunctional, maladaptive. Psychopathology results from the failure of some internal mechanism to perform its natural function—an effect that is part of the evolutionary explanation of the mechanism. The person is symptomatic, but the precise appearance and phenomenology of the pathology is left open, and the dysfunctional process is emphasized. The process leads also to attendant adaptive failure (Mash & Barkley, 1996); it goes beyond symptoms and stalls normal expected progression. This harmful dysfunction has become predominantly internalized, which means it has become highly independent of the context in which it occurs, or it even results in the individual seeking out contexts that fit the harmful dysfunction and that perpetuate its presence (Steiner, 1996). That is, the child will persist doing whatever is maladaptive, regardless of where he or she is or how old he or she is. The net outcome of psychopathology is symptoms (and syndromes) and adaptive failure, which is defined as lack of progression in stage-salient tasks and competencies.

The strong emphasis on process, away from phenomenology, is one of the hallmark features of the model. Most recently, critics of the current diagnostic practices in psychiatry (McHugh, 1998) have also suggested a reorientation of our taxonomies toward causal process. Four pathways are described that might be useful for conceptualizing mental disorders:

- Disease or damage to the brain (for example, mental retardation)
- Weak constitution (bipolar, ADHD)
- Behavior or habits that become a way of life (anorexia nervosa, SAD)
- Reactions induced by external events (PTSD).

We consider this conceptualization a major advance over the current descriptive approach. Whether or not these pathways are sufficient or all necessary remains an issue for empirical research. Viewed from a developmental perspective, though, we must applaud the effort.

The Basic Sciences for Developmental Approaches to Psychopathology

From what we have discussed so far, it should be clear that there is not one science that forms the basis for the developmental clinician. Reaching across the whole span, from culture to molecule, we have to be cognizant that organization at each one of those levels can influence what we see in the consulting room. To represent the entire range of science underpinning mental health would be beyond the scope

of this volume. Therefore, we have selected four contributions from sciences that are either revolutionizing our thinking about mental health problems or have great promise to lead to revolutionary changes in our practice over the next five years. Each one of the following chapters describes the contributions of a particular field that might have implications for the causation of psychopathology and, therefore, treatment. Although the gap between these fields and clinical practice is considerable, we would like to give the clinician the opportunity to get acquainted with lines of investigation, which are extremely promising and important.

We first chose a contribution from ethology. We discuss this contribution first, as in some ways this science can be used to study all three therapeutically important spheres (biology, psychology, and sociology) systematically and with fewer constraints than in humans. Furthermore, we can manipulate all these spheres, control their influences, and study effects on the other spheres and specific domains of functioning. By changing patterns of rearing, we can examine influences on stress hormones at the time and in the animal's future (Levine, 1985). By pairing aggressive and anxious animals with calming older ones in ideal habitats, we can examine the influence of "psychotherapy" on temperament and neurophysiology. By controlling breeding behavior, we can examine relative influences of genes and environment (Suomi, 1996) on personality and emotional regulation.

Ethology is the science of observing animals in their natural environment. We can use this intrinsically naturalistic and organism-oriented paradigm to examine specific forces in carefully designed experiments done to gain insights about specific behavior patterns developing, reactions to stressors, and neurophysiological underpinnings of disease states and therapeutic modalities. One of the main advantages of this science for developmentalists is that the methods allow us to collapse time and shrink it to manageable units. By observing animals whose life span is much shorter than ours, we can rapidly gain insight into how certain traits unfold, which variables foster such growth, and which hinder growth. Since the animal, that is, the organism, is usually the unit of observation, the field allows for holistic approaches to diverse sets of very complex phenomena, such as aggression and attachment.

The field has a long and distinguished history of bringing insights to the mental health sciences. The work of Bowlby (1969), McKinney and Bunney (1969), and Suomi (1996) has taught us much about attachment, temperament, stress reactivity, and even therapist models of animals. More recently, ethology is contributing to our understanding of psychopharmacology and neuroscience. Another valuable insight derived from ethology is one regarding methodology: animal behavior, like human behavior, is quite flexible and multivalent, not rigid, hardwired, and completely predictable. It is rare that single behavioral patterns map onto single neural states—animal behavior too is complex, and can relate to many

different situations. Animal behavior, like human behavior, must be interpreted in the social context in which it occurs. Insofar as animals do so themselves (and depending on the level of animal, they certainly have to in order to survive), they possess a “mind” and a “psychology” that also allows for detailed studies of particular aspects of psychological functioning. But the methods of this science also have limitations, which must be carefully heeded: animal models often are very distinct from humans and do not lend themselves readily to human comparisons. The chapter by Dr. Lyons addresses the core concepts of ethology, the validity of animal models of behavior, and briefly reviews the utility and limitations of animal research in developmental psychiatry. The following four chapters address content areas that are rapidly becoming vital to our attempts to understand normal development and psychopathology in children and youth: sociology, the study of influences on brain organ formation, molecular neurobiology, and genetics all are accumulating knowledge at an unprecedented pace which shortly should lead to new approaches to treatment.

Dr. Niranjan Karnik discusses the social environment, how we understand it, and how it influences children in health and disorder. Sociology is the study of wide arrays of social, cultural, and environmental factors. Family and friends, in order of immediate relevance, to institutions such as schools and welfare agencies, can be either a precipitant or maintaining factor of psychopathology, and have great potential as treatment or ameliorator. Because children are inherently social beings, and their genetic potential is highly dependent on growth inducing influences, the social environment is essential to the understanding of mental disorder and normal development in humans.

After discussing appropriate current models for conceptualizing the social environment for the work of Uri Bronfenbrenner (Bronfenbrenner & Ceci, 1994), Dr. Karnik divides the social environment into the domains of the family, peer groups and subcultures, the school, mass media, and welfare institutions, selecting these areas because they have an intimate relationship to the clinical sphere, and the high probability that practitioners will have to address questions regarding these social spheres and their relationship to the child or adolescent. These subdivisions also lend themselves to links with the specific methods of treatment that are informed by the sociological base, which are presented in the third section of interventions, as edited by Dr. Wilson.

The chapter introduces us to the methods by which social sciences study cultures, subcultures, and families. Ranging from surveys to experiments and qualitative methods, each has its own contribution to make, and has its limitation. As so often is the case, we need all of these methods to form a comprehensive picture, and each method will shape what we see. Subsequently, we discuss in detail the family, peer groups subcultures, and mass media and their influence on youth, schools

and educational systems, welfare institutions, and the juvenile justice system. All of these inherently social institutions have powerful shaping and causal influences on how the lives of young people unfold and how they become disordered.

As children and adolescents are exquisitely dependent on social influences throughout their development in health and disorder, practitioners will ignore the influence of these forces at their own peril. We quickly appreciate how powerful social influences will be when patients describe as the source of their dropping out of treatment or non-compliance with their medication information they have gathered on the Internet, advice they have gotten from their peers, and prohibitions they have received from their families. As Dr. Karnik argues, social history taking must go far beyond the immediate sphere of influence if we want to be accurate in our assessments and maximally beneficial in our interventions. It is a rare practitioner in developmental psychopathology that will not be in contact with schools, welfare systems, and juvenile justice. Skills to consult with these systems in advocating for our patients are core to our professions. And some of the most powerful interventions in psychiatry are socially based, by far exceeding the impact of medications and psychotherapy in terms of immediate impact and net result. Consider the impact of placement in secure custody and seclusion in this regard.

The next chapter by Drs. Remmel and Falvell, two well-known experts in developmental psychology, addresses advances in *cognitive science* pertinent to our field. Connections to clinical practice are immediately apparent. In mental health, psychotherapy always has, and most likely always will, occupy a central place. Pioneered by Freud (1949), we have seen an immense diversification and enrichment in technique and sophistication. Cognitive behavior therapy is among the best-studied and supported intervention techniques in our field, and rivals medications in efficacy. This chapter lays the foundation for our psychotherapy section.

Since its founding days of Piaget (Flavell, Miller, & Miller, 2002) and Vygotsky (1978), the field of cognitive development has moved rapidly during the past ten years. We are increasingly able to study competencies at a younger and younger age by innovative techniques of investigation, which have more closely molded to children's capacity for understanding. This has led to an increasingly fine-grained picture of children's varying levels of knowledge, understanding, and how mental processes change with age. The chapter studies three content areas: core conceptual developments, developments in social cognition, and children's knowledge about the mind. Our appreciation of the newly gained knowledge in all of these is key to matching our interventions to what a young person can handle and utilize. Nobody can doubt anymore that children and adolescents have their own patterns of structuring information that sometimes is highly distinct from that in adults. Their understanding actively shapes what they understand. Much of the therapeutic enterprise is based on our appeal to rational forces, and to appreciate

the diversity of the children's capacities is key to our helping them. In order to firmly conclude that we are dealing with resistance to change, defensive behavior, or non-compliance, we first need to make sure that our patients understand what we ask and are able to do what we recommend. These points hardly need elaboration. Another core concept to intervention is the understanding of causality, which varies considerably across the age spans we encounter. Much of therapy appeals to understanding why one thing will lead to another. Variations in the understanding of these chains of events are a core piece of information to consider as we intervene. Children and adolescents also differ from adults in the way they understand hypothetical situations, especially those with multiple facets, which are so often used in treatment. Ever since the pioneering work of Piaget (Flavell, Miller & Miller, 2002), we have come to understand the changes in logical reasoning, which occur as a function of age. While there is some debate as to whether these sequences hold for everyone, we must appreciate the fact that differential logic can be present when different domains of functioning and competencies are discussed. Children also differ from adults in terms of their ability to control, focus, restrain, and inhibit their behavior. Our understanding of this sequence is highly pertinent to behavioral interventions: not all who are restless have ADHD. Finally, the chapter presents a series of discussions on social cognitions highly relevant to the clinical enterprise: self concept, stability of behavior across time, children's understanding of ability, variability in attribution biases, capacity for perspective taking, and awareness that the stream of consciousness has obvious implications for psychotherapeutic technique, especially for those types that seek to work reflectively, rationally, analytically, and introspectively. Many of these insights have accumulated in the past decade, and this chapter will bring them closer to the practitioner, pointing to their current and future relevance. Clearly, the development of cognition is a strong basis for our essential practices.

In the next three chapters, we turn to a discussion of what it is that is in the "black box," and the types of processes that make us able to reflect and be social. As we better understand them, we also will be in a much better position to see the limits of socio- and psychotherapeutic interventions. For many decades, mental health practitioners, especially those from a behaviorist background, thought the contents of our heads to be irrelevant to their science and practice. With the advent of pediatric psychopharmacology, we have learned that this is clearly not so, from a pragmatic perspective. Additionally, we hope that these following three chapters will show us very exciting and new directions in which basic biological research will contribute to our understanding of the causes of and the interventions for psychopathology. These three chapters describe developments in the neurosciences, which directly will relate to our pharmacological treatments as well, and point to some possible interventions that were unimaginable even ten years ago.

The pace of the acquisition of knowledge in the biological neurosciences has quickened in the past ten years and the excitement of the progress has sometimes led to some excessively simplistic demands on our professions. I have heard some colleagues say things like, "If you do not study the genetics of slime molds you are wasting your time in psychiatry," or, "In just a few years we are all going to be geneticists anyway," and mean it. It should be quite obvious for all the foregoing material that this group of authors does not share this perspective. In fact, it is difficult to see at the present time how we can replace our social and cognitive understanding of psychopathology and treatment with that of biology. Only those in the grip of a powerful ideology (Wilson & Herrnstein, 1985) could expect such simplistic reductionism to be valid. Unfortunately, the pressure on medicine and the healing sciences to become increasingly cost effective has combined with such simple-minded solutions to produce such gems as the "med eval" and the "med check," procedures that masquerade as mental health interventions, which in fact are ineffective and sometimes outright dangerous. Our book, as we mentioned, should be an antidote to this attitude and a challenge to managed care and the insurance industry to support not only what is cheap, but also what works.

Drs. Rubenstein and Puelles discuss what we have learned regarding the development of the brain. Employing a series of extremely sophisticated techniques, neuroscientists are now able to map out the wiring diagrams of the brain as it matures. The brain has been called "the most complex organ in the universe," and rightly so (Edelman, 1994). This chapter provides excellent support for this proposition. The connections formed between different brain areas of functioning are the preconditions for developing circuits, which support higher-level functions to be executed. Faulty connections seem to come about through a variety of interferences at the level of neurons in the originating area, the targeted area, and the neuron's dendrites itself. This process is clearly genetically driven, but additionally requires the concerted and integrated action of neural systems. Typically, neural systems are distributed over multiple regions. Some systems are pathways that include predominantly sensory inputs, sensory perception and integration centers, regions that translate sensory stimuli into potential responses, and motor output regions (regions that we would typically associate with stimulus-motor response type sequences). Other systems consist of entirely internal loops, such as the cortex-basal ganglia-thalamus-cortex circuit. These circuits would have, as a predominant task, the internal information processing associated with memory formation, emotional coloring of incoming sensations, and internal thoughts. Loops and systems are usually redundant, a kind of "fail-safe" wiring which helps us survive, but poses problems for scientists who attempt to study processes underlying the formation of these loops. Multiple influences on similar functions are the norm and not the exception, which probably also relates to the fact that our

medications are usually not as potent as we would like them to be. If one pathway in the brain is influenced by medication, then there are several alternatives available to bypass whatever influence was externally imposed.

All these brain structures are influenced by other systems that can affect the quality or intensity of the process, such as serotonin, norepinephrine, dopamine, and acetylcholine inputs, and a theme that we will return to in the next chapter on neuronal plasticity. As Rubenstein and Puelles point out, it is highly unlikely that neuropsychiatric disorders will ever be understood solely at the cellular level. Clearly we must ask questions regarding the organization and connection of neurons to understand the development of certain psychopathologies. Another message to be taken from this important summary is that we have barely begun to understand the normal developmental processes that make the brain the organ that it is. We know that multiple factors can interfere with the internally directed organization of the brain as a functional unit, but at the present time we do not know which ones they are and which critical periods they operate in. The challenge for the next five to ten years is to establish them as pathogenetic factors in a variety of psychopathologies. The syndromes for which such information will most likely be relevant are certain pervasive developmental disorders, such as autism and mental retardation, but also more complex psychopathologies such as Asperger's syndrome and psychopathy.

Because of the complexity of the processes that control the normal development of brain function, Rubenstein and Puelles advocate that perhaps one should consider neuropsychiatric disorders in a non-traditional pathophysiological context. Rather than focusing on a disease process affecting a single cell type or organ, one should view the disorder as affecting a neural pathway or a neural circuit. Genetic and other influences should be considered in the pathogenesis of disorders. Our understanding of how perinatal and postnatal influences (such as maternal nutrition, drug exposure, trauma, stress, depression, and subsequent life events) influence the formation of the brain as an organ is almost completely absent at this point. But having arrived at an understanding of how the brain forms under normal conditions, we are now in a much better position to take these next steps.

Two additional chapters then take us to suborganic levels of abstraction. Drs. Post and Post examine molecular and cellular developmental vulnerabilities to the onset of affective disorders in children and adolescents and make some suggestions for several levels of treatment, ranging from disease progression to prevention.

They begin by pointing out that we are just beginning to have an understanding of the cellular-related processes pertinent to emotion, learning, and memory—the kinds of processes that profoundly influence us as we grow and gain experiences, and reorganize ourselves according to what has been learned. The

seminal work of Kandel, which earned him the Nobel Prize in medicine, has paved the way for our understanding of these processes (Kandel and Hawkins, 1994). At this point, we assume that the basic wiring of emotional regulation would be extremely plastic and evolve over the course of a child's development. Rather than regarding the brain as a fixed organic entity following a rigidly predetermined pattern of development, it is exquisitely responsive to external input, presumably over one's whole lifetime. This whole line of inquiry is especially pertinent for the study of emotional development. Nerve cells can form new connections and relinquish old ones, dependent on learning and memory. By mapping cellular components, which influence preferential connections in response to exposure to external influences, we begin to appreciate the shaping that takes place—for better or worse—after we are born. The Posts apply this paradigm to the development of affective disorders and anxiety disorders. Discussing pre-clinical data pertinent to affect and affiliation in animals and clinical data in patients with affective and anxiety disorders, this chapter links events at the neuronal level to clinical syndromes. The Posts apply their model to illness progression, as we find in stress responsivity and episode sensitization in bipolar illness. The clinical data are then linked to subhuman data on stimulant-induced behavioral sensitization and electrophysiological kindling in animals, an experimental paradigm, which provides convergent support for the postulated model from the background of experimental science. By using the external drug-use paradigm again, the Posts discuss the impact of such “environmental” factors on gene expression and neuronal development. As work by Beurrier and Malenka (2002) has shown in the case of substance use cravings, we are now in a position to outline the long lasting impact of exogenous substances on neuronal action patterns. The Posts extend these findings to the case of affective regulation and dysregulation, showing that experience can change something as basic as gene expression in the brain. Applying the findings from neurology to the study of affective processes and their disorders, the Posts then show that the phenomenon of *kindling*, which is the stimulation of full-blown nerve discharge after a series of subthreshold stimuli, can also help us understand clinical phenomena such as bipolar disorder and chronic traumatization. Finally, they discuss exciting findings regarding the connections between neonatal stressors and lasting effects on behavior, neuroendocrinology, and substance abuse vulnerability.

In many ways, the findings from this wide range of studies have immediate implications for clinical practice as far as the causes of bipolar illness is concerned. Additionally, there are also implications for therapeutic interventions in this disorder and others, which potentially respond to anticonvulsants and mood stabilizers. There is the exciting possibility that a therapeutic compound may in fact have curative or protective effects, a topic recently summarized by Ketter (2002).

Potentially, psychiatry would be in a position of possessing not just therapeutic or palliative medications (such as stimulants for ADHD or antipsychotics for schizophrenia), but preventive or even curative ones. Posts' discussion also links with the importance of pharmacoprophylaxis in vulnerable individuals with bipolar disorder, a form of tertiary prevention. They are advocating in early and targeted prevention for those at high risk, such as for instance symptomatic, but not syndromally ill bipolar offspring. And they conclude by indicating promising directions for primary prevention in high-risk populations, such as unaffected bipolar offspring. Preventive principles also can be applied to patients with bipolar illness and their offspring when we seek to protect them from substance abuse and its especially deleterious consequences in bipolar illness and interventions for maltreated children. They conclude with a neurobiological overview and integration of the findings in the clinically diverse data sets discussed.

Finally, we examine the role of the gene in the formation of psychopathology. With the mapping of genome, there was much excitement regarding the potential of genetic factors playing a role in much (or even most) of the origin of psychopathology. In a balanced and erudite discussion, Dr. Joachim Hallmayer tells us where we stand in regard to the role of genes in psychopathology and treatment. Although the overall message is cautious and complex, we are beginning to appreciate the contributions of genetics to mental health practices. Changes in available techniques have given us the opportunity to examine gene-expression data that complement gene-sequence data. At the present time, there are many efforts seeking to characterize specific genes, in hopes that such understanding will vastly expand our understanding of health and disease and the practice of medicine.

While there are some examples of interventions, which are based on genetic techniques and successfully studied and marketed, in the area of psychopathology, we have mostly promissory notes. Ultimately, Dr. Hallmayer suggests, the major impacts of this molecular revolution are envisaged to be:

- Using specific genetic indicators to target treatment and to identify individuals at risk for a certain disorder to be able to prevent onset of disorder
- Using genetic studies to predict efficacy of certain medications in individuals (pharmacogenetics)
- Using genes as therapeutic agents to implant corrective genetic material
- Using animal models for studying the genetic contributions to the development of certain disorders.

Introducing us to the types of genetic changes, which are pertinent to our understanding, the chapter leads us to a discussion of single gene disorders, such as Rett syndrome, along with other forms of mental retardation. Dr. Hallmayer then

turns to a discussion of genetically complex disorders, such as autism, where we have good reason to believe that multiple genes are involved in creating the problem. Many vexing questions are raised by this discussion, which shows us in impressive detail how far the distance is between the gene and higher levels of complexity affected in a disorder, which is not particularly subtle in its manifestations. Abandoning single gene models leads to questions about how many genes are involved, and how they interact to produce the adverse outcome. On the clinical side, the findings in autism also lead to the expansion of the original diagnostic category into a disease spectrum to account for differential influences of combinations of genes. The chapter continues with a discussion of the implications for therapy for autism. While it is clear from the discussion that direct therapeutic implications will depend on many other unknown factors, we are beginning to see how genetics may play a role in the therapeutics of some disorders in the not so distant future. One such example is the exciting new area of investigation, pharmacogenetics. Clinicians have struggled for many years with the fact that there is a high inter-individual variability in the response to pharmacological drugs, both in terms of side effects and treatment response. Pharmacogenetics seeks to improve our knowledge in regards to who will respond to a particular intervention, in what way and why, based on genetic profiles of relevant nervous system components, such as, for instance, receptors. Other plausible targets for pharmacogenetic studies are variations in enzymes controlling drug absorption and elimination. As in the study of psychopathology and its causes, studies so far have focused on single genes. The technological advance of gene array technology allows characterization of gene expression of thousands of genes in parallel. More multi-targeted studies such as this have considerable promise to improve our currently poor level of understanding of variables that affect efficacious medication treatment.

The chapter then turns to a discussion of gene therapy, where genetic material is inserted into a defective cell in order to treat the problem. For many complex reasons discussed in the chapter, this is not a simple enterprise, but most definitely worthy of pursuing, as once again it offers the chance of curative interventions. Most of the clinical studies in this area are in oncology, and most of those are still in early stages of development (stage I and II), establishing safety, side effects, and tolerability criteria.

In all these chapters, we begin to appreciate the distance between basic science and current clinical practice. However, we can see several important touch points, which show great promise to influence how we will be able to help children and adolescents with mental health problems in new and exciting ways. But, in order to evaluate how adequate and efficacious current techniques of intervention are, we need further tools. The last chapter in this section will provide those. But first we

need to briefly establish the current standards for evidence-based practice in medicine. Evidence-based practice has become the standard of the healing sciences in this millennium. Several criteria have been proposed for a practice to be evidence based: we find the ones suggested by Kazdin (2000) to be most helpful. He lists the four requirements for practice to be evidence based, and these criteria show the intrinsically necessary close relationship between basic science and clinical practice.

First, there has to be a *theory linking a particular process to a particular problem*. These theories can originate either in the consulting room or in the laboratory. They can be derived from a series of experiments or a series of patients. An example of a clinically generated theory regarding the origin of anorexia nervosa would be the idea that anorexia nervosa represents an unconscious avoidance of the demands of maturation and adolescence (Steiner & Lock, 1998). An example for a laboratory-generated theory would be that impulsive aggression is caused by a serotonergic neurotransmission deficit. (Swann, et al., 2002). I would characterize the first theory as one of *top down* causation (an attitude, a belief, or a distortion of thinking originates from a high level of complexity in the human mind/brain setup and causes the psychopathological state). The serotonergic theory would be labeled as a *bottom up* process. Both theories lead to testable hypotheses related to intervention and treatment. Given our current state of knowledge, either theory would deserve attention but would lead to quite different interventions. In general, bottom up theories would be easier to test (in animals, for instance) and thus probably lead more rapidly to data. Conversely, theories, which involve highly complex mental states, which occur only in humans, would be more difficult to test for many reasons, some of them ethical. On the other hand, the top down theories might be much more readily applicable to humans, while the external validity of bottom up hypotheses would be at times questionable.

Secondly, there has to be *some research assessing the validity of the proposed mechanism*. In this regard, the serotonergic hypothesis fares much better than the adolescence-avoidance hypothesis. More data in animals and humans confirm the validity of the proposed mechanism (Swann, et al., 2002), although the identity challenge theory is not completely without backing (Steiner & Lock, 1998). It is clear, though, that in both cases, strictly controlled laboratory experiments would be necessary to examine the validity of the particular process.

Thirdly, there should be, according to Kazdin (2000), some preliminary *outcome evidence that treatment along the lines suggested by the hypothesis changes the pathological status* of the patient. In other words, we need to show that by targeting the fears of growing up (presumably by some reflective psychotherapy or perhaps even a desensitization paradigm), we in fact alter the outcome of anorexia nervosa or bulimia. Evidence along these lines exists (Steiner & Lock, 1998). By the same

token, we need to show that giving mood stabilizers or SSRIs, thus theoretically enhancing serotonergic neurotransmission, leads to reduction in maladaptive aggression. This is easier and more powerfully shown in animals (Swan, et al., 2002) than in humans (Steiner, et al., 2003), but outcomes are still in line with this theory.

Finally, we need to demonstrate that the *causal process we have postulated for a particular problem is influenced by the treatment we use to target it and changes in that process are connected to the desired outcome*. An elegant body of work in this regard is the work by Patterson's group on coercive behaviors in the genesis and persistence of maladaptive aggression. In this research, it has been convincingly shown that a parent's response to a child's coercive behavior has direct and indirect implications for their antisocial conduct. Altering this causal loop of the parent unwittingly reinforcing coercion and non-compliance changes the child's chance to persist in maladaptive aggression (Patterson, 1986).

A complex series of steps establishes a treatment as evidence based. The proper labels for how treatments are in fact supported would be as follows (*Clinical Evidence*, 2002):

“Beneficial”	Clearly supported effectiveness by evidence from Randomized Controlled Trials (RCT), low risk, high benefit to be expected
“Likely to be beneficial”	Less well supported by RCTs
“Trade off between benefits and harms”	Clinicians and patients should carefully weigh these before applying the treatment
“Unknown effectiveness”	Either insufficient or poor quality empirical support
“Unlikely to be beneficial”	Even less support than in the previous category
“Likely to be ineffective or harmful”	Both of these have been demonstrated clearly in studies

As we will see in the body of this volume, for most interventions we have a long way to go. In order to be able to evaluate how adequately all our available interventions have been tested, we need to become familiar with methods of scientific evaluation, which are particularly pertinent to developmental-based research. The final chapter by Drs. Saxena and Blasey addresses this need. The chapter examines the various designs used in clinical and basic research, illustrating the particular design through a study from the extant literature. Ranging from the “gold standard procedure” (the randomized controlled study to cohort studies), to case control studies, case series, and case reports, the chapter steps us through the advantages and disadvantages of each design. Review papers and meta-analytic

techniques used to summarize extensive data from divergent sources are carefully outlined. Special design features, such as longitudinal designs, follow-up studies, and prospective versus retrospective studies are elaborated. Pilot studies are briefly discussed. Multicenter designs and various specific terms we encounter when describing measurements and studies are summarized. The chapter concludes with a careful stepwise dissection of the features of one of the author's favorite studies to illustrate the approach to the scientific literature on a personal level. A useful glossary of commonly encountered terms is appended.

Where Are We, and Where Are We Going?

We have traversed a great distance in this chapter, going from culture to molecule in our search for better ways to help children and adolescents with mental health problems. To summarize all that we will be introduced to in this section is almost impossible. So rich and diverse are the findings in the fields supporting clinicians' endeavors that we have to modestly list them and wait for the near future to provide us with further integration. From my perspective, the diversity, richness, and complexity of facts we have encountered may be overwhelming to those practicing in the consulting room. They certainly are to our trainees and I might add, most of us in academia. From another perspective, we live in very exciting times. Not so long ago, this section in a handbook on treatment would have been either very slender indeed, or very parochial in character. We clearly have departed from the old days of school-driven treatment and thinking. Our continued challenge is not to become prematurely reductionistic while avoiding being confusedly biopsychosocial.

The developmental model we discussed at the beginning of this chapter will hopefully prevent us from doing either. From its perspective, I see that the evidence supports three major pathways, corresponding to the three major sections in this handbook: psychotherapy, pharmacotherapy, and sociotherapy. At the current state of knowledge in our field, it is impossible to dismiss any of these. As our empirical data base grows, we will hopefully simplify our approaches, but at the same time, we most likely also will stand to gain a whole new array of techniques that are just beginning to be defined and studied, all offering the prospect of reducing human suffering and restoring mental health.

Many questions remain at this stage of knowledge in treatment based on the developmental model. What must be fairly acknowledged is that the empirical data backing up the interventions to be discussed are unevenly distributed. However, at the very least, all of the interventions presented in this volume have a rationale, and sometimes even a more or less sophisticated theory behind them, which allows for the generation of testable hypotheses. Some interventions are

standardized, even manualized, in their mode of application; many of them show efficacy, or even effectiveness. As of today, we must keep an open mind regarding all of them and not assume that all can be reduced to one or the other.

The past fifty years have seen a monumental change in the way we think about our mental health and its causes. We have gone from a quasi-disease model of psychic determinism (psychoanalysis) to a black box, environmentalist, continuous trait model (learning theory) back to a phenomenological, typologically based disease model (the *Diagnostic and Statistical Manual of Mental Disorders [DSM]* and the *International Clinical Diagnosis [ICD]*) to a neuroscience-driven reductionism. Each one of these has left its impact on the field, and each has had its modicum of successes; however, none of them have produced any impressive cures or massively effective interventions. Any parochial insistence that one model is superior to the others is ill placed. Pragmatically, we need to track all these models and interventions in their further development and testing in the laboratory and the real world of the consulting room. To uphold our end of the bargain in the dialectical progress of mental health sciences and practice, we need to understand causal processes as best we can, apply the interventions best suited to change them, and show that our approaches work. It is then up to the colleagues in the lab to study why and how exactly they work and, in turn, help us refine how we approach problems in the consulting room.

We (Steiner & Hayward, 2004) conceptualize the progress in solving the mental health problems in youth as dialectic between practitioners and scientists. We approach these problems by studying them scientifically and treating them clinically. At the present time, we are called upon to masterfully select interventions according to problems, blend according to need, and execute to the best of our abilities. This we will discuss in our final chapter on integrating treatment.

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