

CHAPTER 1

Child Psychopathology

A Developmental–Systems Perspective

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This volume provides a comprehensive account of the diagnosis, phenomenology, developmental pathways, correlates, causes, and outcomes of psychopathology in children.¹ Our understanding of developmental psychopathology has grown exponentially over the past several decades (Beauchaine & Hinshaw, 2013; Cicchetti, 2006; Cicchetti & Toth, 2009; Mash & Wolfe, 2013; Rutter, 2005). New conceptual frameworks and findings, as well as advances in knowledge and methods, continue to further our understanding of childhood disorders (Granic, 2005; Iacono & Malone, 2011; Moffitt, 2005; Roth & Sweatt, 2011; Rutter & Sroufe, 2000; Sameroff & Mackenzie, 2003), as well as our ability to assess and treat children with problems (Gunnar, Fisher, & The Early Experience, Stress, and Prevention Network, 2006; Kraemer et al., 2003; March, 2009; Mash & Barkley, 2006; Weisz, Sandler, Durlak, & Anton, 2005). However, this understanding is tempered by the often unsystematic and fragmented fashion in which research findings in child psychopathology have accrued, and by the conceptual and research complexities inherent in the study of such a rapidly changing and socially embedded organism as the child (Hinshaw, 2001; Sameroff & Mackenzie, 2003). In this introductory chapter, we address several central themes and issues related to conceptualizing childhood dysfunction and its many determinants. In doing so, we provide a

developmental–systems framework for understanding child psychopathology—one that emphasizes the role of developmental processes, the importance of context, and the influence of multiple and interacting events and processes in shaping adaptive and maladaptive development.

FACTORS THAT COMPLICATE THE STUDY OF CHILD PSYCHOPATHOLOGY

Since modern views of mental illness began to emerge in the late 18th and early 19th centuries, the study of psychopathology in children has lagged behind that of adults (Silk, Nath, Siegel, & Kendall, 2000). For example, in 1812, Benjamin Rush, the first American psychiatrist, suggested that children were less likely to suffer from mental illness than adults because the immaturity of their developing brains would prevent them from retaining the mental events that caused insanity (Silk et al., 2000). However, it is now well established that many childhood disorders are common, early-occurring, and chronic, and that they exact a high toll from children, their families, and society (Costello, Egger, & Angold, 2006; Costello, Foley, & Angold, 2006). Furthermore, disorders of childhood often show significant homo- and heterotypic continuity with later

child and adult psychopathology (Bufferd, Dougherty, Carlson, Rose, & Klein, 2012; Copeland, Shanahan, Costello, & Angold, 2009; Reef, Diamantopoulou, van Meurs, Verhulst, & van der Ende, 2009), further supporting the relevance of childhood psychopathology for long-term adjustment. Looking backward from adulthood, epidemiological researchers have found that many adults with a mental disorder first developed psychopathology as children (Kessler et al., 2005). Thus interest in the study of child psychopathology has rightly increased dramatically.

However, an array of unresolved issues hampers progress in the investigation of psychopathology in children. Critically, issues concerning the conceptualization and definition of psychopathology in children continue to be vigorously debated (Rutter & Uher, 2012). Until fairly recently, much of the field's accumulated knowledge about the phenomenology of disorders of childhood was extrapolated from work with adults. For example, only in recent decades have child-focused models of depressive disorders emerged (e.g., Abela & Hankin, 2008). While it is well established that children can and do meet criteria for depression derived largely from research with adults, it is also clear that there are key differences in the presentation of the disorder across development (Rohde, Lewinsohn, Klein, Seeley, & Gau, 2013; Weiss & Garber, 2003). Furthermore, in contrast to adult forms of the disorder, evidence for the genetic basis of childhood depression is decidedly mixed (Rice, 2010), suggesting that aspects of models of adult depression may not extrapolate well to earlier manifestations of the disorder. This is but one example of the complexities regarding continuities and discontinuities of disorders across development.

Even in studies conducted with children, much of our knowledge is based on findings obtained at a single point in a child's development and in a single context. Although useful, such findings provide still photographs of moving targets and fail to capture the dynamic changes over time that characterize most forms of child psychopathology (Achenbach & Dumenci, 2001; Lewis & Granic, 2000). Although contextual models (e.g., Bronfenbrenner, 1977) and longitudinal approaches (e.g., Robins, 1966) have been applied to the field of child study for decades, researchers have only fairly recently begun to use developmentally sensitive systems-oriented models to account for the emergence of psychopathology in children (Granic, 2005; Sameroff, 2000). While longitudinal studies have become much more common, such studies are complicated by

multiple issues including the question of how to best implement developmentally sensitive measures that can differentiate between true change and stability across a broad span of development from change in measurement strategy (Singer & Willett, 2003, pp. 13–14). In addition, many prior studies have not consistently attended to the broader familial, social, and cultural contexts in which atypical child development occurs (Davies & Cummings, 2006; Marks, Patton, & García Coll, 2011; Serafica & Vargas, 2006), often focusing solely on intrinsic characteristics of the child to the neglect of the broader context in which development unfolds.

The study of child psychopathology is further complicated by the fact that many childhood problems are not narrow in scope or expression, and that most forms of psychopathology in children are known to overlap and/or coexist with other disorders (Angold, Costello, & Erkanli, 1999; Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Drabick & Kendall, 2010; Lilienfeld, 2003). For example, it has been established for some time that there is pervasive overlap among such problems as child maltreatment, violence, emotional and behavioral disorders, substance abuse, delinquency, and learning difficulties, between childhood anxiety and depression and between reading disabilities and anxiety and depression (e.g., Garber & Weersing, 2010; Oshri, Rogosch, & Cicchetti, 2013; Seligman & Ollendick, 1998; Willcutt & Pennington, 2000b). Many behavioral and emotional disturbances in youth are also associated with specific physical symptoms and/or medical conditions and poor health outcomes (Costello, Egger, & Angold, 2006; Nigg, 2013; Pinquart & Shen, 2010; Reynolds & Helgeson, 2011; Spady, Schopflocher, Svenson, & Thompson, 2005).

It is also the case that distinct boundaries between many commonly occurring childhood behaviors (e.g., noncompliance, defiance) and those problems that come to be labeled as "disorders" (e.g., oppositional defiant disorder) are not easily drawn (e.g., Loeber, Burke, Lahey, Winters, & Zera, 2000). There is mounting evidence that most forms of psychopathology differ *in degree* from normative behavior, rather than *in kind* (i.e., distinctions between normal and abnormal behavior are typically quantitative, rather than qualitative; see Coghill & Sonuga-Barke, 2012, for a review of this issue in child psychopathology). Furthermore, judgments of deviancy often depend as much on other child characteristics (e.g., age, sex, intelligence), the situational appropriateness of a child's behavior, the social and cultural context in which judgments are made,

and the characteristics and decision rules of adults who make these judgments as they do on any specific behaviors displayed by the child (Achenbach, 2000; De Los Reyes & Kazdin, 2005; Mash & Barkley, 2007).

It has become increasingly evident that most forms of child psychopathology are etiologically heterogeneous and cannot be attributed to a single unitary cause. Although a handful of rare disorders (e.g., phenylketonuria, fragile-X intellectual disability, Rett's disorder) may be caused by single genes, behavioral and molecular genetics research indicate that more common and complex disorders are likely the result of multiple genes (Goldsmith, Gottesman, & Lemery, 1997; McGuffin, Riley, & Plomin, 2001; O'Conner & Plomin, 2000), and that most forms of child psychopathology are likely to have an oligo- or polygenic basis, involving susceptibility genes that interact with one another and with environmental influences to result in observed levels of impairment (Dodge & Rutter, 2011; Dodge & Sherrill, 2007; Moffitt, Caspi, & Rutter, 2006; State, Lombroso, Pauls, & Leckman, 2000). Child and family disturbances are likely to result from multiple, frequently co-occurring, reciprocal, and interacting risk factors, causal events, and processes (e.g., El-Sheikh, Keiley, Erath, & Dyer, 2013; Jaffee & Price, 2007; Rutter, 2007a). Contextual events exert considerable influence in producing child and adolescent disorders—an influence that is almost always equivalent to, or greater than, those factors usually thought of as residing “within” the child (Davies & Cummings, 2006; Reiss & Neiderhiser, 2000; Rutter, 2000). Furthermore, it has become increasingly clear that genetic influences on disorder risk can no longer be assumed to be static in their effects, as the functional impact of polymorphisms is further moderated by an array of regulatory processes known as “epigenetic effects” (Mill, 2011; Zhang & Meaney, 2010), some of which unfold in response to environment conditions. For example, animal models indicate that epigenetic effects may account for the influence of early caregiver behavior on offspring outcomes via its impact on the expression of specific genes (Weaver, Meaney, & Szyf, 2006). Life experiences that alter gene expression may also account for monozygotic twin discordance on highly heritable psychiatric phenotypes (e.g., Petronis et al., 2003). The best way to capture this dynamic interplay between genetic and environmental risks with respect to psychopathology processes has yet to be determined.

Numerous risk markers for child psychopathology have been identified, including genetic influences (e.g.,

Goodyer, Bacon, Ban, Croudace, & Herbert, 2009; Gotlib, Joormann, Minor, & Hallmayer, 2008; Sheikh et al., 2013); temperament (e.g., Hayden, Klein, Durbin, & Olino, 2006; Olino, Klein, Dyson, Rose, & Durbin, 2010); insecure child–parent attachments (e.g., Lee & Hankin, 2009; Priddis & Howieson, 2012); social-cognitive deficits (e.g., Luebke, Bell, Allwood, Swenson, & Early, 2010; Zadeh, Im-Bolter, & Cohen, 2007); deficits in social learning (e.g., Arsenio & Lemerise, 2010; Lansford, Malone, Dodge, Pettit, & Bates, 2010); emotion regulation and dysregulation (e.g., Feng et al., 2009; Tortella-Feliu, Balle, & Sesé, 2010); effortful control and related constructs (Eisenberg et al., 2005; Gusdorf, Karreman, van Aken, Dekovic, & van Tuijl, 2011); neuropsychological and/or neurobiological dysfunction (e.g., Cicchetti & Cannon, 1999; Lopez-Duran, Kovacs, & George, 2009); maladaptive patterns of parenting and maltreatment (e.g., Beauchaine, Neuhaus, Zalewski, Crowell, & Potapova, 2011; Cicchetti & Toth, 2005; Harkness, Stewart, & Wynne-Edwards, 2011; Lovejoy, Graczyk, O'Hare, & Neuman, 2000); parental psychopathology (e.g., Goodman & Gotlib, 1999; Pettit, Olino, Roberts, Seeley, & Lewinsohn, 2008); parental discord (e.g., Fear et al., 2009; Pagani, Japel, Vaillancourt, Côté, & Tremblay, 2008; Shelton & Harold, 2008); limited family resources and other poverty-related life stressors (e.g., Dupéré, Leventhal, & Lacourse, 2009; Najman et al., 2010; Schreier & Chen, 2013; Tracy, Zimmerman, Galea, McCauley, & Vander Stoep, 2008); institutional deprivation (e.g., Ellis, Fisher, & Zaharie, 2004); and a host of other factors. However, these factors cannot be understood in isolation, and for most disorders, research does not support granting central etiological status to any single risk or causal factor (e.g., Sameroff, 2010).

Since the many causes and outcomes of child psychopathology are often interrelated and operate in dynamic and interactive ways over time, they are not easy to disentangle. The designation of a specific factor as a cause or an outcome of child psychopathology usually reflects (1) the point in an ongoing developmental process at which the child is observed, and (2) the perspective of the observer. For example, a language deficit may be viewed as a disorder in its own right (e.g., language disorder), the cause of other difficulties (e.g., impulsivity), or the outcome of some other condition or disorder (e.g., autism spectrum disorder). In addition, biological and environmental determinants interact at all periods of development. For example, Belsky and de Haan (2011) recently noted that the characteristic

styles parents use influence critical patterns of cortical and subcortical development across childhood and well into adolescence. Consistent with this, Dougherty, Klein, Rose, and Laptook (2011) reported that familial depression and parental hostility interacted to predict heightened cortisol reactivity to stress in a sample of community-dwelling preschoolers—a finding that suggests altered activity of the stress-regulating hypothalamic–pituitary–adrenocortical (HPA) system among children with multiple facets of risk for psychopathology. The majority of this work has focused on the impact of severe early adversity (e.g., maltreatment), so that far less is known about the impact of more normative experiences on children’s brain development. Still, these and many other findings indicate that early experiences may shape neural structure and function, which may then create dispositions that direct and shape a child’s later experiences and behavior (Cicchetti & Walker, 2001; Fox, Zeanah, & Nelson, 2012; Glaser, 2000; Kaufman & Charney, 2001).

In a volume covering child psychopathology, it is also worth noting that there may be issues related to the stigma of mental illness with particular relevance to children. Although definitions of stigma have varied across studies, it appears to be a multidimensional construct that is not well characterized with respect to disorders of childhood, relative to adulthood. Stigma can be experienced across different contexts and targets (Mukolo, Heflinger, & Wallston, 2010), and appears to play a role in decreasing the likelihood that services are sought for children with a mental disorder, particularly in minority groups and cultures (e.g., Yeh, McCabe, Hough, Dupuis, & Hazen, 2003). Differentiating the consequences of mental health stigma from those related to the symptoms of disorder can be difficult and has not always been closely attended to in research designs (e.g., caregiver strain could stem from both children’s symptoms of disorder and parents’ own symptoms, as well as perceived negative responses to the children’s status as patients; Brannan & Heflinger, 2006). Additional work on the origins and role of stigma, especially as it pertains to the willingness of families to seek care or to participate in basic science on the etiology of disorder, is therefore critical.

As will be discussed throughout this volume, current models of child psychopathology seek to incorporate the roles of evolved mechanisms; neurobiological factors; early parent–child relationships; attachment processes; a long-term memory store that develops with age and experience; micro- and macrosocial influ-

ences; cultural factors; age and gender; and reactions from the social environment as variables and processes that interact and transform one another over time. In short, then, current approaches view the roots of developmental and psychological disturbances in children as the result of complex interactions over the course of development between the biology of brain maturation and the multidimensional nature of experience (Belsky & de Haan, 2011; D’Onofrio, Rathouz, & Lahey, 2011; Reiss & Neiderhiser, 2000; Rutter et al., 1997).

The experience and the expression of psychopathology in children have cognitive, affective, physiological, and behavioral components; in light of this, many differing descriptions and definitions of dysfunctionality in children have been proposed. As we discuss in a later section, a common theme in defining child psychopathology has been “adaptational failure” in one or more of these components or in the ways in which these components are organized and integrated (Rutter & Sroufe, 2000; Sameroff, 2000). Adaptational failure may involve deviation from age-appropriate norms (Achenbach, 2001); exaggeration or diminishment of normal developmental expressions; interference in normal developmental progress; failure to master developmental tasks; failure to develop a specific function or regulatory mechanism; and/or the use of non-normative skills (e.g., rituals, dissociation) as a way of adapting to regulatory problems or traumatic experiences (Sroufe, 1997).

A multitude of etiological models and treatment approaches have been proposed to explain and remediate psychopathology in children. Unfortunately, most of these have yet to be substantiated or even adequately tested (Kazdin, 2000, 2001). These models and approaches have differed in their relative emphasis on certain causal mechanisms and constructs, often using very different terminology and concepts to describe seemingly similar child characteristics and behaviors. Although useful, many of these models have been based on what seem to be faulty premises concerning singular pathways of causal influence that do not capture the complexities of child psychopathology (Kazdin & Kagan, 1994).

In this regard, evolutionary models have emphasized the role of selection pressures operating on the human species over millions of years; biological paradigms have emphasized genetic mutations, neuroanatomy, and neurobiological mechanisms as factors contributing to psychopathology; psychodynamic models have focused on intrapsychic mechanisms, conflicts, and defenses;

attachment models have emphasized the importance of early relationships and the ways in which internal representations of these relationships provide the foundation for constructing working models of self, others, and relationships more generally; behavioral/reinforcement models have emphasized excessive, inadequate, or maladaptive reinforcement and/or learning histories; social learning models have emphasized the importance of observational learning, vicarious experience, and reciprocal social interactions; cognitive models generally focus on the child's distorted or deficient cognitive structures and processes; affective models have emphasized dysfunctional emotion-regulating mechanisms; and family systems models have conceptualized child psychopathology within a framework of intra- and intergenerational family systems and subsystems and have emphasized the structural and/or functional elements surrounding family relational difficulties.

The distinctiveness of each model mentioned above is in the relative importance it attaches to certain events and processes. However, it should be emphasized that despite these variations in the relative emphasis given to certain causes versus others, most models recognize the role of multiple interacting influences. For example, although they differ in emphasis, social learning and affective models both place importance on the role of symbolic representational processes in explaining childhood dysfunction.

There is a growing recognition of the need to integrate currently available models through intra- and interdisciplinary research efforts. Such integration generally requires looking beyond the emphasis of each single-cause theory to see what can be learned from other approaches, as well as a general openness to relating concepts and findings from diverse theories (cf. Arkowitz, 1992). Studies suggest that theoretical integration is becoming more common in psychopathology research (e.g., Beauchaine, 2001). Attachment theory has, for instance, been increasingly integrated with cognitive models (e.g., Ingram & Ritter, 2000). Theoretical integration is also apparent in studies combining proximal cognitive and interpersonal factors with distal variables, such as genetic markers of risk, the early home environment, and patterns of attachment (e.g., Caspi et al., 2003; Gibb, Beevers, & McGeary, 2013; Hayden, Klein, et al., 2010; Lara, Klein, & Kasch, 2000). The link between cognitive and neuropsychological functioning is likewise being tested more frequently (e.g., Nigg, Blaskey, Huang-Pollack, & Rappley, 2002). Thus researchers increasingly recognize the importance of

combining theoretical approaches, and are accepting the monumental task of incorporating increased complexity into their research designs. The need for such integrative research approaches has important implications for training future developmental psychopathologists to be conversant in a broad array of research approaches and theories.

On a related note, interdisciplinary perspectives on child psychopathology mirror the considerable investment in children on the part of many different disciplines and professions. The study of the etiology and maintenance of psychopathology in children has been and continues to be the subject matter of psychology, medicine, psychiatry, education, and numerous other disciplines. Clearly, no one discipline has proprietary rights to the study of childhood disturbances, and each has tended to formulate child psychopathology in terms of its own unique perspective. Particularly relevant, in the context of this chapter, is that child psychopathology and normality in medicine and psychiatry have traditionally been conceptualized and defined categorically in terms of the presence or absence of a particular disorder or syndrome that is believed to exist “within the child.” In contrast, psychology has more often conceptualized psychopathology–normality as representing extremes on a continuum or dimension of characteristics, and has also focused on the role of environmental influences that operate “outside the child.” However, the boundaries between categories and dimensions, or between inner and outer conditions and causes, are arbitrarily drawn, and there is a continuing need to find workable ways of integrating the two different world views of psychiatry/medicine and psychology (Pickles & Angold, 2003; Richters & Cicchetti, 1993; Scotti & Morris, 2000; Shaffer, Lucas, & Richters, 1999).

Despite these ongoing issues in the field, the subsequent chapters in this volume attest to the substantial and rapid accrual of research on child psychopathology. This in turn has resulted in a rapidly expanding and changing knowledge base. Each chapter in this volume provides a comprehensive review of current research and theory for a specific form of child psychopathology, and a discussion of new developments and directions related to this disorder. In the remainder of this introductory chapter, we provide a discussion of the following: an overview of the significance and implications of child psychopathology; epidemiological considerations; key concepts in the field; approaches to the definition and conceptualization of childhood disorders; an overview of the developmental psycho-

pathology framework; predominant theories regarding etiology; and prevalent and recurrent conceptual and methodological issues that cut across the wide spectrum of disorders represented in this volume. Particular emphasis is given to concepts, methods, and strategies capturing the complexities, reciprocal influences, and divergent pathways that current models and research have identified as crucial for understanding child psychopathology.

SIGNIFICANCE OF CHILD PSYCHOPATHOLOGY

There has been and continues to be a great deal of misinformation and folklore concerning disorders of childhood. Many unsubstantiated theories have emerged in both the popular and scientific literatures. These have ranged from mid-19th-century views that overstimulation in the classroom causes insanity (see Makari, 1993), to mid-20th-century views that inadequate parenting causes autism (Bettelheim, 1967) or that chemical food additives are the primary cause of hyperactivity (Feingold, 1975). In addition, many of the constructs used to describe the characteristics and conditions of psychopathology in children have been globally and/or poorly defined (e.g., “adjustment problem,” “emotional disturbance”). Despite the limitations, uncertainties, and definitional ambiguities that exist in the field, it is also evident that psychopathology during childhood represents a frequently occurring and significant societal concern that is gradually coming to the forefront of the political agenda.

Increasingly, researchers in the fields of child development, developmental psychopathology, child psychiatry, and clinical child psychology are considering the social policy implications of their work and striving to effect improvements in the identification of and services for youth with mental health needs (Cicchetti & Toth, 2000; Kazdin & Blase, 2011; Shonkoff, 2010; Shonkoff & Bales, 2011). For example, such work contributed to a recent report of the Surgeon General’s office on suicide prevention, part of which focused on prevention of suicide in youth (U.S. Public Health Service, 2012). Such efforts are critical, given that public policies that promote early socioemotional well-being and reduce the conditions that lead to early child maltreatment may provide the foundation needed for later school success and positive peer and teacher relationships. Policy makers are generally not well acquainted with children’s mental health concerns, or with the se-

rious ramifications of early maladjustment (Nelson & Mann, 2011). Furthermore, public policy has not kept pace with advances in the field of child psychopathology (Zero to Three, 2012), especially with regard to recognizing how common and pervasive disorders of childhood are, or having an awareness of the benefits of early screening and intervention (Sices, 2007). Strategies to promote positive early development, as well as to prevent and treat early mental health problems, will require not only significant investment on the part of federal and local governments but an increased recognition that public policy should be shaped by empirical research. The need for policy to (1) support the training of individuals with the necessary expertise in children’s mental health, and to (2) address the significant, ongoing obstacle faced by many parents of how to afford such expertise, has also been noted (Zero to Three, 2012).

The growing attention to children’s mental health problems and competencies arises from a number of sources. First, many young people experience significant mental health problems that interfere with normal development and functioning. As many as one-third of children in the United States experience some type of difficulty (Costello, Mustillo, et al., 2003); this longitudinal study indicated that the risk of experiencing a psychiatric disorder by age 16 was much higher than previous estimates, derived from cross-sectional data, had indicated. Furthermore, this estimate probably underestimates the impact of psychopathology in youth, since it does not capture subclinical or undiagnosed disturbances that nevertheless place children at high risk for the later development of more severe clinical problems (e.g., Keenan et al., 2008). In addition, although not meeting formal diagnostic criteria, many subclinical conditions (e.g., depressed mood, eating problems) are associated with significant impairment in functioning (e.g., Angold, Costello, Farmer, Burns, & Erkanli, 1999; Lewinsohn, Striegel-Moore, & Seeley, 2000). Evidence gathered by the World Health Organization (WHO) suggests that by the year 2020, childhood neuropsychiatric disorders will rise by over 50% internationally to become one of the five most common causes of morbidity, mortality, and disability among children (U.S. Public Health Service, 2001b).

Second, a significant proportion of children do not grow out of their childhood difficulties, although the ways in which these difficulties are expressed change in dynamic ways over time (Masten & Cicchetti, 2010). Even when diagnosable psychopathology is not

evident at later ages, a child's failure to adjust during earlier developmental periods may still have a lasting negative impact on later family, occupational, and social adjustment. Furthermore, some forms of child psychopathology—for example, an early onset of antisocial patterns of behavior—are highly predictive of a host of negative outcomes later in life (e.g., Kim-Cohen et al., 2005).

Third, recent social changes and conditions may place children at increasing risk for the development of disorders, and also for the development of more severe problems at younger ages (Dupéré et al., 2009; Masten & Narayan, 2012). These social changes and conditions include multigenerational adversity in inner cities; chronic poverty in women and children; pressures of family breakup, single parenting, and homelessness; problems of the rural poor; direct and indirect exposure to traumatic events (e.g., terrorist attacks or school shootings); adjustment problems of children in immigrant families; difficulties of Native American children; and conditions associated with the impact of prematurity, HIV, cocaine, and alcohol on children's growth and development (McCall & Groark, 2000; Shonkoff & Phillips, 2000). In addition to sociocultural changes, medical advances associated with higher rates of fetal survival may also contribute to a greater number of children's showing serious behavior problems and learning disorders at a younger age.

Fourth, for a majority of children who experience mental health problems, these problems go untreated: Kataoka, Zhang, and Wells (2002) reported that of children identified as needing mental health services, only about 20% received such assistance. Rates of unmet need were even higher in ethnic minority groups and in children without insurance. Even when children are identified and receive help for their problems, this help may be less than optimal. For example, only about half of children with identified attention-deficit/hyperactivity disorder (ADHD) seen in real-world practice settings receive care that conforms to recommended treatment guidelines (Hoagwood, Kelleher, Feil, & Comer, 2000). The fact that so few children with mental health problems receive appropriate help is probably related to such factors as a lack of screening, inaccessibility, cost, a lack of perceived need on the part of parents, parental dissatisfaction with services, and the stigmatization and exclusion often experienced by these children and their families (Hinshaw, 2007; Hinshaw & Cicchetti, 2000). These and other factors have stimulated recent initiatives to identify children with

unmet mental health needs (e.g., Jensen et al., 2011). Although empirically supported prevention and treatment programs for many childhood disorders have become increasingly established in recent decades (Chorpita et al., 2011; Kazak et al., 2010), a pressing need remains for additional research on normative child development, developmental psychopathology, and the continued development and evaluation of prevention and intervention programs that are grounded in empirical evidence (Greenberg, Domitrovich, & Bumbarger, 2001; Kazdin, 2001; Rapport, 2001; Silverman & Hinshaw, 2008).²

Fifth, a majority of children with mental health problems who go unidentified and unassisted often end up in the criminal justice or mental health systems as young adults (Loeber & Farrington, 2000). They are at much greater risk for dropping out of school and of not being fully functional members of society in adulthood; this adds further to the costs of childhood disorders in terms of human suffering and financial burdens. For example, average costs of medical care for youngsters with ADHD are estimated to be double those for youngsters without ADHD (Leibson, Katusic, Barbaresi, Ransom, & O'Brien, 2001). Moreover, allowing just *one* youth to leave high school for a life of crime and drug abuse is estimated to cost society from \$1.7 to \$2.3 million or more (Cohen, 1998; Cohen & Piquero, 2009).

Finally, a significant number of children in North America experience maltreatment, and chronic maltreatment during childhood is associated with psychopathology in children and later in adults (Fergusson, Borden, & Horwood, 2008; Gunnar et al., 2006). Based on a review of the evidence, De Bellis (2001) has proposed that the psychobiological outcomes of abuse be viewed as "an environmentally induced complex developmental disorder" (p. 539). Although precise estimates of the rates of occurrence of maltreatment are difficult to obtain, due to the covert nature of the problem and other sampling and reporting biases (Cicchetti & Manly, 2001; Wekerle, Wolfe, Dunston, & Alldred, Chapter 16, this volume), the numbers appear to be large. Over 3.5 million suspected cases of child abuse and neglect are investigated each year by child protective service agencies, and about 1 million children in the United States were confirmed as victims of child maltreatment in 2010 (U.S. Department of Health and Human Services [USDHHS], 2011). It has been estimated that each year over 2,000 infants and young children die from abuse or neglect at the hands of their par-

ents or caregivers (USDHHS, 2011). Moreover, many reports of “accidental” injuries in children may be the result of unreported mistreatment by parents or siblings (Peterson & Brown, 1994). It would appear, then, that the total number of children who show adverse psychological and physical effects of maltreatment in North American society is staggering.

EPIDEMIOLOGICAL CONSIDERATIONS

Prevalence

Epidemiological studies seek to determine the prevalence and distribution of disorders and their correlates in particular populations of children who vary in age, sex, socioeconomic status (SES), ethnicity, or other characteristics (Costello & Angold, 2000). Although epidemiological studies of child psychopathology of the same scope as those of adult psychopathology (e.g., Kessler et al., 2005) have not been conducted, disorders of childhood appear to be common. Although reported rates vary widely from study to study, current best estimates are that 20–40% of all children worldwide have a clinically diagnosable disorder, and that many more children exhibit specific symptoms or subclinical problems (Belfer, 2008; Kessler et al., 2012; Merikangas, He, Brody, et al., 2010). Overall lifetime prevalence rates for childhood problems are on the order of 36% of all children (Costello, Mustillo, et al., 2003). Earlier studies also reported high rates of disorder; for example, Rutter, Tizard, and Whitmore (1970), in the classic Isle of Wight Study, found the overall rate of child psychiatric disorders to be 6–8% in 9- to 11-year-old children. Richman, Stevenson, and Graham (1975), in the London Epidemiological Study, found moderate to severe behavior problems for 7% of the population, with an additional 15% of children having mild problems. Boyle and colleagues (1987) and Offord and colleagues (1987), in the Ontario Child Health Study, reported that 19% of boys and 17% of girls had one or more disorders. Many other epidemiological studies have reported similar rates of prevalence (e.g., Brandenburg, Friedman, & Silver, 1990; Costello, Farmer, Angold, Burns, & Erkanli, 1997; Earls, 1980; Hewitt et al., 1997; Lapouse & Monk, 1958; MacFarlane, Allen, & Honzik, 1954; Shaffer et al., 1996; Verhulst & Koot, 1992; Werner, Bierman, & French, 1971). Perhaps the most consistent general conclusions to be drawn from these studies are that prevalence rates for childhood

disorders are generally high, but that rates may vary with the nature of the disorder; the age, sex, SES, and ethnicity of the children; the criteria used to define the problem both concurrently and over time, the method used to gather information (e.g., interview, questionnaire); the informants (e.g., children, parents, teachers); sampling methods; and a host of other factors.

Age Differences

Bird, Gould, Yager, Staghezza, and Camino (1989) reported no significant age differences for children ages 4–16 years in the total number of *Diagnostic and Statistical Manual of Mental Disorders*, third edition (DSM-III) disorders diagnosed at each age. However, some studies have reported interactions among child age, number or type of problems, child sex, clinical status, and source of information (e.g., Simonoff et al., 1997). For example, Achenbach, Howell, Quay, and Conners (1991) found that externalizing problems showed a decline with age relative to internalizing problems, but only for those children who had been referred for treatment. More recently, using structured clinical interview data in a large sample of youth, Costello, Mustillo, and colleagues (2003) reported that the highest prevalence of disorder was found in children ages 9–10, with levels gradually falling through age 12 and then rising again throughout the adolescent years. The authors noted that this was likely due to the fact that the prevalence of many disorders of childhood (e.g., ADHD, separation anxiety disorder) decreases by age 12, while disorders of adolescence and adulthood (e.g., major depression) have not yet emerged. Merikangas, He, Burstein, and colleagues (2010) recently reported that 22% of adolescents had a disorder associated with severe impairment and/or distress in a nationally representative survey of adolescents ages 13–18.

These and other findings raise numerous questions concerning age differences in children’s problem behaviors. Answers to even a seemingly simple question such as “Do problem behaviors decrease (or increase) with age?” are complicated by (1) a lack of uniform measures of behavior that can be used across a wide range of ages; (2) qualitative changes in the expression of behavior with development; (3) interactions between child age and sex; (4) the use of different informants across development; (5) the specific problem behavior(s) of interest; (6) the clinical status of the children being assessed; and (7) the use of different diagnostic criteria for children of different ages. Notwith-

standing these difficulties, both longitudinal and cross-sectional general population surveys are informative in depicting changes in the proportions of specific parent-, teacher-, or child-reported problem behaviors with age (e.g., “hyperactive,” “argues,” “cries”), as well as the manner in which the age changes vary as a function of problem type, child sex, and child clinical status. However, it should be emphasized that general age trends are based on group statistics, which may obscure the nonlinear and non-normative changes that often occur for individual children. In addition, general surveys do not provide information concerning the processes underlying age changes. Studies of change in individual children over time, and of the context in which this change occurs, are needed if such processes are to be understood.

Socioeconomic Status

Although most children treated for mental health problems are from the middle class, mental health problems are overrepresented among the very poor. It is estimated that 20% or more of children in North America are poor, and that as many children growing up in poverty are impaired to some degree in their social, behavioral, and academic functioning (McLeod & Nonnemaker, 2000). Lower-SES children have been reported to display more psychopathology and other problems than upper-SES children (e.g., McMahon & Luthar, 2007; Samaan, 2000). However, although the reported relationships between SES and child psychopathology are statistically significant, the effects are small and should be interpreted cautiously (Achenbach et al., 1991), as global estimates of SES tell us little about the multifarious processes through which SES and children’s adaptive and maladaptive development are related (Schreier & Chen, 2013). Knowledge of such processes is needed to inform our understanding of disorders and to develop preventative efforts that target the appropriate mechanisms. For example, the effects of SES on aggression can be explained partly by stressful life events and by beliefs that reflect a tolerance or acceptance of aggression (Guerra, Tolan, Huesmann, Van Acker, & Eron, 1995). Other work suggests that the impact of SES on broader externalizing problems may be related to the reduced ability of impoverished parents to monitor their children (Costello, Compton, Keeler, & Angold, 2003). Further illustrating the complex interplay between risks, the environment that parents provide is also related to parental psychopathology, as in

the case of adult ADHD, which is associated with low SES (Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1998); in such a case, parents confer both genetic and contextual risk on offspring, and this contextual risk emerges at least in part through gene–environment correlation (with the potential for “downward drift,” such that disorder reduces economic opportunity, in the present case).

Thus associations between socioeconomic disadvantage and children’s mental health derive from the fact that SES is a marker of many potential sources of negative influence (Bradley, Corwyn, McAdoo, & García Coll, 2001). Low SES is often characterized by low maternal education, a low level of employment, single-parent status, parental psychopathology, limited resources, and both chronic and acute negative life events (e.g., poor nutrition, exposure to violence), in addition to low income. Since overall indices of SES may include one or more of these variables in any given study, the relationship that is reported between SES and child psychopathology may vary as a function of the particular index used, as well as ethnic factors (McLeod & Nonnemaker, 2000). In short, SES is a marker of many factors that influence risk for child psychopathology, and the way in which this indicator is operationalized has an impact on its associations with childhood disorder.

Some research findings in child psychopathology are confounded by a failure to include SES in models. For example, although physically abused children show higher levels of externalizing problems than nonabused children (Mash, Johnston, & Kovitz, 1983), it is not clear that physical abuse and externalizing problems are associated when the effects of SES are controlled for (Cummings, Hennessy, Rabideau, & Cicchetti, 1994; Fergusson et al., 2008). The relationships among SES, maltreatment, and behavior disorders are further complicated by other findings that the effects of physical abuse on internalizing disorders may be independent of SES, whereas the effects of abuse on externalizing disorders may be dependent on SES-related conditions (Okun, Parker, & Levendosky, 1994).

Sex Differences

Although sex differences in the expression of psychopathology have been formally recognized since Freud’s writings at the beginning of the 20th century, psychopathology in girls has historically received far less research attention than psychopathology in boys (Bell-

Dolan, Foster, & Mash, 2005; Rose & Rudolph, 2006). Until recently, many studies either have excluded girls from their samples entirely or have failed to examine whether relevant effects differed across the two sexes. For example, until fairly recently, there were relatively few studies of disruptive behavior disorders in girls (e.g., Moffitt, Caspi, Rutter & Silva, 2001; Silverthorn & Frick, 1999), probably because such disorders are more common in boys than in girls during childhood. Also contributing to this may be sampling biases (in which boys, who are more severely disruptive, are more likely to be referred and studied), as well as the fact that the inclusionary diagnostic criteria most commonly used are derived and validated largely from studies with boys (Frick & Nigg, 2012; Spitzer, Davies, & Barkley, 1990).

Research has confirmed that there are important differences in the prevalence, expression, accompanying disorders, underlying processes, outcomes, and developmental course of psychopathology in boys versus girls (Willcutt & Pennington, 2000a; Zahn-Waxler, Shirlcliff, & Marceau, 2008). ADHD, autism spectrum disorder, childhood conduct and oppositional disorders, and learning and communication disorders are all more common in boys than girls, whereas the opposite is true for most anxiety disorders, adolescent depression, and eating disorders (Copeland et al., 2011; Rutter et al., 2004). Relatedly, boys exhibit higher levels of externalizing symptoms than girls do throughout childhood and early adolescence, whereas girls and boys are comparable in terms of internalizing symptoms in early childhood, with girls' levels of these symptoms increasing more rapidly than boys during adolescence (e.g., Bongers, Koot, van der Ende, & Verhulst, 2003). Although these sex differences are well established, their meaning is poorly understood (Martel, 2013). For example, it is difficult to determine whether observed sex differences are functions of referral or reporting biases, the way in which disorders are currently defined, differences in the expression of a disorder (e.g., direct vs. indirect aggressive behavior), sex differences in the genetic penetrance of disorders, sexual selection effects/evolutionary processes, or sex differences in biological characteristics and environmental susceptibilities. All are possible, and there is a need for research into the processes underlying observed differences. Clearly the mechanisms and causes of sex differences may vary for different disorders (e.g., ADHD vs. depression), or for the same disorder at different ages (e.g., child vs. adolescent obsessive-compulsive

disorder or early- vs. late-onset conduct disorder). For example, Moffitt and Caspi (2001) found that sex differences in life-course-persistent antisocial behavior were attributable to differences in rates of risk factors for early-onset, persistent forms of such behaviors, such as hyperactivity, poor parenting, and neuropsychological dysfunction, which may disproportionately affect boys compared to girls.

Early research into sex differences focused mainly on descriptive comparisons of the frequencies of different problems for boys versus girls at different ages. In general, differences in problem behaviors between the sexes are small in children of preschool age or younger (e.g., Briggs-Gowan, Carter, Skuban, & Horwitz, 2001; Gadow, Sprafkin, & Nolan, 2001), but become increasingly common with age. For example, Weisz and Suwanlert (1989) studied children in the United States and Thailand, and found that boys were rated higher than girls on every problem for which there was a significant sex difference—including total problems, undercontrolled problems, overcontrolled problems, and culture-specific problems. Across cultures, boys have been found to display more fighting, impulsivity, and other uncontrolled behaviors than girls (Olweus, 1979). It has been found that boys show greater difficulties than girls during early and middle childhood, particularly with respect to ADHD and disruptive behavior disorders (Costello, Mustillo, et al., 2003). Girls' problems may increase during adolescence, with higher prevalence rates for depression and dysphoric mood from midadolescence through adulthood. For example, conduct disorder and ADHD have been found to be more frequent in 12- to 16-year-old boys than girls, whereas emotional problems have been found to be more frequent for girls than boys in this age group (Boyle et al., 1987; Offord et al., 1987).

However, not all studies have reported significant sex differences in overall rates of problem behavior (e.g., Achenbach & Edelbrock, 1981; Velez, Johnson, & Cohen, 1989), and even when significant overall sex differences have been found, they tend to be small and to account for only a small proportion of the variance. It has also been found that although there is a much larger predominance of externalizing problems in boys and of internalizing problems in adolescent girls in samples of children who are referred for treatment, sex differences in externalizing versus internalizing problems are minimal in nonreferred samples of children (Achenbach et al., 1991). Furthermore, there may be cohort effects on sex differences in some forms of psychopathology.

For example, the sex difference in substance use disorders, which historically consisted of higher rates of these disorders in boys compared to girls, appears to be disappearing in more recent cohorts due to increased substance use by girls (Johnston, O'Malley, Bachman, & Schulenberg, 2011).

Comparisons of the behavioral and emotional problems in boys versus girls over time can provide useful information about sex-related characteristics. However, taken in isolation, such global comparisons do not address possible qualitative differences in (1) expressions of psychopathology in boys versus girls; (2) the processes underlying these expressions; (3) the long-term consequences of certain behaviors for boys versus girls; and/or (4) the impact of certain environmental events on boys versus girls (Zahn-Waxler et al., 2008). As noted by Hops (1995), it seems likely that “the pathways from childhood to adolescence and adult pathology are age and gender specific and that these differences may be the result of different social contexts that nurture the development of health or pathology for female and male individuals” (p. 428). In addition to differential socialization practices, there are likely to be differences in the expression and outcome of psychopathology in boys versus girls as a function of biologically based differences. For example, in a study of the psychophysiology of disruptive behavior in boys versus girls, Zahn-Waxler, Cole, Welsh, and Fox (1995) found that disruptive girls showed high electrodermal responding relative to disruptive boys and were also highly activated by a sadness mood induction. These investigators suggested that girls' disruptive behavior may be more closely connected than boys' disruptive behavior to experiences of anxiety. Other research has found that increases in depression in females during adolescence are related mostly to accompanying changes in levels of estrogen and testosterone (Angold, Costello, Erkanli, & Worthman, 1999). It is also possible that for some disorders (e.g., ADHD), girls may require a higher genetic loading for the disorders than boys before the disorders are likely to express themselves (Rhee, Waldman, Hay, & Levy, 1999).

There may also be differences in the processes underlying the expression of psychopathology and distress in boys versus girls (Chaplin & Aldao, 2013; Kistner, 2009; Rutter, Caspi, & Moffitt, 2003). For example, a slower rate of biological maturation (Zahn-Waxler, Crick, Shirtcliff, & Woods, 2006; Zahn-Waxler et al., 2008), as well as sex differences in temperamental variables (Else-Quest, Hyde, & Goldsmith, 2006;

Frick & Morris, 2004; Olino, Durbin, Klein, Hayden, & Dyson, 2013) may provide explanatory mechanisms for the higher rates of conduct problems in boys versus girls. In addition, depression in adolescent females has been found to be strongly associated with maternal depression, whereas a lack of supportive early care appears to be more strongly associated with depression in adolescent males (Duggal, Carlson, Sroufe, & Egeland, 2001). It has also been found that the types of child-rearing environments predicting resilience to adversity may differ for boys and girls. Resilience in boys is associated with households in which there is a male model (e.g., father, grandfather, older sibling), structure, rules, and some encouragement of emotional expressiveness. In contrast, resilient girls come from households that combine risk taking and independence with support from a female caregiver (e.g., mother, grandmother, older sister) (Werner, 1995). With respect to future goals for this specific aspect of research, the role of paternal psychopathology in offspring psychopathology risk, and whether its impact differs for boys versus girls, has not been explored to the extent it should (Connell & Goodman, 2002), given its known impact on other factors that shape child outcomes (e.g., paternal caregiving; Wilson & Durbin, 2010).

Zahn-Waxler and colleagues (2008) refer to the “gender paradox of comorbidities,” which is that although the prevalence of disruptive behavior is lower in females than in males, the risk of comorbid conditions such as anxiety is higher in female samples. In explaining this paradox, these authors suggest that girls' heightened level of interpersonal sensitivity, caring, and empathy may be a protective factor with respect to the development of antisocial behavior. At the same time, girls' heightened sensitivity to the plight of others, and their reluctance to assert their own needs in situations involving conflict and distress, may elevate their risk for the development of internalizing problems. However, the relations between gender and comorbidity are likely to vary with the disorders under consideration, the age of a child, the source of referral, and other factors. For example, in contrast to Zahn-Waxler and colleagues (1995), Biederman and colleagues (2002) found that girls with ADHD had a significantly *lower* rate of comorbid major depression than did boys with ADHD. Martel (2013) has posited that sex differences such as these may have emerged via sexual selection processes related to the enhanced survival value or impact on mating opportunities linked to the biological substrates of these conditions.

Although findings relating to sex differences and child psychopathology are complex, inconsistent, and frequently difficult to interpret, the cumulative findings from research strongly indicate that the effects of gender are critical to understanding the expression and course of most childhood disorders (Bell-Dolan et al., 2005; Zahn-Waxler et al., 2008). It is particularly important to understand the processes and mechanisms underlying these gender effects, and to recognize that biological influences and differential socialization practices are likely to interact throughout development in accounting for any differences between the sexes that are found.

Rural versus Urban Differences

Although there is a general belief that rates of child behavior disorders are higher in urban than in rural areas, research findings in support of this view are weak and/or inconsistent. Findings from older studies of the Isle of Wight, Inner London Borough, and Ontario Child Health Studies reveal prevalence rates of problem behavior that were higher for urban than rural children (Offord et al., 1987; Rutter, 1981). On the other hand, in a cross-cultural investigation, Weisz and Suwanlert (1991) found few differences in parent or teacher ratings of child problems as a function of rural versus urban status in either of the cultures that were studied (United States and Thailand). In a detailed analysis that controlled for the effects of SES and ethnicity and also looked at gradations of urbanization, Achenbach and colleagues (1991) found few differences in children's behavior problems or competencies as a function of rural-versus-urban status, although there was a significant but very small effect indicating higher delinquency scores for children in urban environments. These investigators concluded that earlier findings of higher rates of problem behavior in urban than in rural areas "may have reflected the tendency to combine areas of intermediate urbanization with large urban areas for comparison with rural areas as well as a possible lack of control for demographic differences" (p. 86). Even in studies in which rural versus urban differences have been found, for the most part these differences were associated with economic and cultural differences between sites, and not with urbanization per se (Zahner, Jacobs, Freeman, & Trainor, 1993). Further complicating this issue is the possibility that the effects of urbanicity on psychopathology likely vary depending on disorder. For example, van Son, van Hoeken, Bartelds,

van Furth, and Hoek (2006) found that rates of bulimia nervosa were higher in urban areas, whereas rates of anorexia nervosa did not differ depending on urbanization. Intriguingly, some of the effects of urbanicity on psychopathology may operate via gene-environment interaction; for example, environmental conditions appear to moderate the relative contribution of genetic effects on externalizing forms of psychopathology (LeGrand, Keys, McGue, Iacono, & Krueger, 2008).

Ethnicity and Culture

Ethnicity

Numerous terms have been used to describe ethnic influences. These include "ethnicity," "race," "ethnic identity," "ethnic orientation," "acculturation," "bicultural orientation," and "culture." As Foster and Martinez (1995) have pointed out, there is a need to recognize the diversity of terminology that has been used in describing ethnicity, and the fact that these terms refer to related but different things. Despite the growing ethnic diversity of the North American population, ethnic representation in research studies and the study of ethnicity-related issues more generally have received less attention in studies of child psychopathology (García Coll, Akerman, & Cicchetti, 2000; U.S. Public Health Service, 2001a). Until recently, research into child psychopathology has generally been insensitive to possible differences in prevalence, age of onset, developmental course, and risk factors related to ethnicity (Yasui & Dishion, 2007), as well as to the considerable heterogeneity within specific ethnic groups (Murry, Bynum, Brody, Willert, & Stephens, 2001; Serafica & Vargas, 2006). In addition, few studies have compared ethnic groups while controlling for other important variables, such as SES, sex, age, and geographic region. Some recent studies suggest that children from minority groups are overrepresented in certain disorders, such as substance use disorders (Nguyen, Huang, Arganza, & Liao, 2007). Overall, studies with much larger national samples that included European American, African American, and Hispanic American children have reported either no or very small differences related to race or ethnicity when SES, sex, age, and referral status were controlled for (Achenbach & Edelbrock, 1981; Achenbach et al., 1991; Lahey et al., 1995). Thus, although externalizing problems have been reported more frequently among African American children (McLaughlin, Hilt, & Nolen-Hoeksema,

2007), this finding is probably an artifact related to SES. Unfortunately, African American and Hispanic American children are much less likely to receive specialty mental health services or psychotropic medications (García Coll & Garrido, 2000). Native American youth appear to have elevated rates of problem behaviors, including substance abuse and suicide (Whitbeck, Yu, Johnson, Hoyt, & Walls, 2008). Ethnicity has not been found to be strongly associated with risk for eating disorders (Leon, Fulkerson, Perry, & Early-Zald, 1995), although differences between European Americans and other groups have been reported for such sub-clinical eating disturbances as dietary restraint, ideal body shape, and body dissatisfaction (Wildes & Emery, 2001). More research is needed in which potentially important third variables (e.g., SES) are adequately addressed, but these and other findings suggest that the effects of ethnicity are likely to vary with the problem under consideration and its severity.

As is the case for SES and sex differences, global comparisons of the prevalence of different types of problems for different ethnic groups are not likely to be very revealing. On the other hand, studies into the processes affecting the form, associated factors, and outcomes of different disorders for various ethnic groups hold promise for increasing our understanding of the relationship between ethnicity and child psychopathology (e.g., Bird et al., 2001; Bradley, Corwyn, Burchinal, McAdoo, & García Coll, 2001).

Culture

The values, beliefs, and practices that characterize a particular ethnocultural group contribute to the development and expression of childhood distress and dysfunction, which in turn are organized into categories through cultural processes that further influence their development and expression (Achenbach & Rescorla, 2007; Harkness & Super, 2000; Wong & Ollendick, 2001). Through shared views about causality and intervention, culture also structures the way in which people and institutions react to a child's problems. Since the meaning of children's social behavior is influenced by cultural beliefs and values, it is not surprising that the form, frequency, and predictive significance of different forms of child psychopathology vary across cultures, or that cultural attitudes influence diagnostic and referral practices (Lambert et al., 1992). For example, shyness and oversensitivity in children have been found to be associated with peer rejection and social

maladjustment in Western cultures, but with leadership, school competence, and academic achievement in Chinese children in Shanghai (Chen, Rubin, & Li, 1995). Similarly, Lambert, Weisz, and Knight (1989) found that overcontrolled problems were reported significantly more often for Jamaican than for American youngsters—a finding consistent with Afro-British Jamaican cultural attitudes and practices that discourage child aggression and other undercontrolled behavior, and that foster inhibition and other overcontrolled behavior.

Weisz and Sigman (1993), using parent reports of behavioral and emotional problems in 11- to 15-year-old children from Kenya, Thailand, and the United States, found that Kenyan children were rated particularly high on overcontrolled problems (e.g., fears, feelings of guilt, somatic concerns), due primarily to numerous reports of somatic problems. In this mixed-race sample, whites were rated particularly high on undercontrolled problems (e.g., “arguing,” “disobedient at home,” “cruel to others”). Weisz and Suwanlert (1987) compared 6- to 11-year-old children in the Buddhist-oriented, emotionally controlled culture of Thailand with American 6- to 11-year-olds. Parent reports revealed Thai–U.S. differences in 54 problem behaviors, most of which were modest in magnitude. Thai children were rated higher than American children on problems involving overcontrolled behaviors such as anxiety and depression, whereas American children were rated higher than Thai children on undercontrolled behaviors such as disobedience and fighting.

Weisz and Suwanlert (1991) compared ratings of behavior and emotional problems of 2- to 9-year-old children in Thailand and the United States. Parents and teachers in Thailand rated both overcontrolled and undercontrolled problems as less serious, less worrisome, less likely to reflect personality traits, and more likely to improve with time. These findings suggest that there may be cultural differences in the meanings ascribed to problem behaviors across cultures.

Findings from these and other studies suggest that the expression of, and tolerance for, many child behavioral and emotional disturbances are related to social and cultural values. The processes that mediate this relationship are in need of further investigation. In this regard, it is important that the results of research on child psychopathology not be generalized from one culture to another, unless there is support for doing so. There is some support for the notion that some processes—for example, those involved in emotion regulation and its

relation to social competence—may be similar across diverse cultures (Eisenberg, Pidada, & Liew, 2001). The rates of expression of some disorders, particularly those with a strong neurobiological basis (e.g., ADHD, autism spectrum disorder), may be less susceptible to cultural influences than others. However, even so, social and cultural beliefs and values are likely to influence the meaning given to these behaviors, the ways in which they are responded to, their forms of expression, their outcomes, and responses to intervention (Castro, Barrera, & Holleran-Steiker, 2010).

An important distinction to be made with respect to cross-cultural comparisons is whether there are substantive differences in the rates of a disorder, or differences in the raters' perceptions of these problems. For example, Weisz and Suwanlert (1989) compared the teacher-reported behavioral/emotional problems of Thai and U.S. children (ages 6–11 years). It was found that Thai teachers were confronted with students who were more prone to behavioral and emotional problems at school than were teachers in the United States, but that they applied different judgments to the behaviors they observed. Similarly, cultural factors are known to influence not only informal labeling processes but formal diagnostic practices as well. For example, reported prevalence rates of ADHD in Great Britain are much lower than in the United States because of differences in the way in which diagnostic criteria for ADHD are applied in the two countries. Such differences in diagnostic practices may lead to spurious differences in reported prevalence rates for different forms of child psychopathology across cultures.

Cross-cultural research on child psychopathology would suggest that the expression and experience of mental disorders in children is not universal (Fisman & Fisman, 1999). Patterns of onset and duration of illness and the nature and relationship among specific symptoms vary from culture to culture, and across ethnic groups within cultures (Achenbach, 2001; Hoagwood & Jensen, 1997; Yasui & Dishion, 2007). However, few studies have compared the attitudes, behaviors, and biological and psychological processes of children with mental disorders across different cultures. Such information is needed to understand how varying social experiences and contexts influence the expression, course, and outcome of different disorders across cultures. Greater social connectedness and support in more traditional cultures and greater access to resources and opportunities in industrialized societies are examples of mechanisms that may alter outcomes across

cultures. Sensitivity to the role of cultural influences in child psychopathology has increased (Evans & Lee, 1998; Lopez & Guarnaccia, 2000), and is likely to continue to do so as globalization and rapid cultural change become increasingly more common (García Coll et al., 2000).

KEY CONCEPTS IN CHILD PSYCHOPATHOLOGY

Several recurrent and overlapping issues have characterized the study of psychopathology in children (Cicchetti & Toth, 2009; Rutter & Sroufe, 2000). A number of these are highlighted in this section, including (1) difficulties in conceptualizing psychopathology and normality; (2) the need to consider healthy functioning and adjustment; (3) questions concerning developmental continuities and discontinuities; (4) the concept of developmental pathways; (5) the notions of risk and resilience; (6) the identification of protective and vulnerability factors; and (7) the role of contextual influences.

Psychopathology versus Normality

The attempt to establish boundaries between what constitutes abnormal and normal functioning is an arbitrary process at best (see Achenbach, 1997), although this does not necessarily imply that such boundaries are meaningless, if they are informative with respect to impairment and other clinically significant factors. Traditional approaches to mental disorders in children have emphasized concepts such as symptoms, diagnosis, illness, and treatment; by doing so, they have strongly influenced the way we think about child psychopathology and related questions (Richters & Cicchetti, 1993). Childhood disorders have most commonly been conceptualized in terms of deviancies involving breakdowns in adaptive functioning, statistical deviation, unexpected distress or disability, and/or biological impairment.

Wakefield (1992, 1997, 1999b, 2010) has proposed an overarching concept of mental disorder as “harmful dysfunction.” This concept encompasses a child’s physical and mental functioning, and includes both value- and science-based criteria. In the context of child psychopathology, a child’s condition is viewed as a disorder only if (1) it causes harm or deprivation of benefit to the child, as judged by social norms; and (2) it results from the failure of some internal mechanism to perform its natural function (e.g., “an effect

that is part of the evolutionary explanation of the existence and structure of the mechanism”; Wakefield, 1992, p. 384). This view of mental disorder focuses attention on evolved adaptations or internal functional mechanisms—for example, executive functions in the context of self-regulation (Barkley, 2001). Nevertheless, as Richters and Cicchetti (1993) have pointed out, this view only identifies the decisions that need to be made in defining mental disorders; it does not specify *how* such decisions are to be made.

As is the case for most definitions of mental disorder that have been proposed, questions related to defining the boundaries between normal and abnormal, understanding the differences between normal variability and dysfunction, defining what constitute “harmful conditions,” linking dysfunctions causally with these conditions, and circumscribing the domain of “natural” or of other proposed mechanisms are matters of considerable controversy (Hudziak, Achenbach, Althoff, & Pine, 2007; Lilienfeld & Marino, 1995).³ Categories of mental disorder stem from human-made linguistic distinctions and abstractions, and boundaries between what constitutes normal and abnormal conditions, or between different abnormal conditions, are not easily drawn. Although it may sometimes appear that efforts to categorize mental disorders are “carving nature at its joints,” whether or not such “joints” actually exist is open to debate (e.g., Angold & Costello, 2009; Cantor, Smith, French, & Mezzich, 1980; Lilienfeld & Marino, 1995). However, clear distinctions do not necessarily need to exist for categorical distinctions to have utility. For instance, there is no joint at which one can carve day from night, although distinguishing the two has proven incredibly useful to humans in going about their social discourse and engagements. Likewise, although the threshold for determining disorder from high levels of symptoms may be fuzzy, it could be stipulated as being at that point along a dimension where impairment in a major, culturally universal life activity befalls the majority of people at or exceeding that point. Thus, despite the lack of clear boundaries between what is normal and abnormal, categorical distinctions are still useful as long as they adequately predict which children will be most likely to benefit from access to special education, treatment, or disability status.

Healthy Functioning

The study of psychopathology in children requires concomitant attention to adaptive developmental processes

for several reasons. First, judgments of deviancy require knowledge of normative developmental functioning, both with respect to a child’s performance relative to same-age peers and with respect to the child’s own baseline of development. Second, maladaptation and adaptation often represent two sides of the same coin, in that dysfunction in a particular domain of development (e.g., the occurrence of inappropriate behaviors) is usually accompanied by a failure to meet developmental tasks and expectations in the same domain (e.g., the nonoccurrence of appropriate behaviors). It is important to point out, however, that adaptation should not be equated with the mere absence of psychopathology, nor should the converse be assumed (i.e., that symptoms can be equated with maladaptation). With respect to the former, Kendall and colleagues (Kendall, Marrs-Garcia, Nath, & Sheldrick, 1999; Kendall & Sheldrick, 2000), contend that it is important to use normative comparisons to evaluate treatment outcome; they suggest that improvement involves falling within a certain range of healthy functioning, in addition to decreased symptoms. Moreover, adaptation involves the presence and development of psychological, physical, interpersonal, and intellectual resources (see Fredrickson, 2001). With respect to the latter point, symptoms and impairment tend to be only moderately correlated, suggesting that for some children, symptoms do not have a pervasive negative impact on important life domains (Barkley, 2012a; Gordon et al., 2006).

Third, in addition to the specific problems that lead to referral and diagnosis, disturbed children are likely to show impairments in other areas of adaptive functioning. For example, in addition to their core symptoms of hyperactivity/impulsivity and inattention, children with ADHD typically show lower-than-average levels of functioning in their socialization, communication, and activities of daily living (e.g., Stein, Szumowski, Blondis, & Roizen, 1995). Fourth, most children with specific disorders are known to cope effectively in some areas of their lives. Understanding a child’s strengths informs our knowledge of the child’s disorder and provides a basis for the development of effective treatment strategies. Fifth, children move between pathological and nonpathological forms of functioning over the course of their development. Individual children may have their “ups and downs” in problem type and frequency over time. Sixth, many child behaviors that are not classifiable as deviant at a particular point in time may nevertheless represent less extreme expressions or compensations of an already existing disorder

or early expressions of a later progression to deviant extremes as development continues (Adelman, 1995). Finally, no theory of a childhood disorder is complete if it cannot be linked with a theory of how the underlying normal abilities develop and what factors go awry to produce the disordered state. Therefore, understanding child psychopathology requires that we also attend to these less extreme forms of difficulty and develop more complete models of the normal developmental processes underlying the psychopathology.

For these and other reasons to be discussed, the study of child psychopathology requires an understanding of both abnormal and healthy functioning (Cicchetti, 2006). As noted by Cicchetti and Richters (1993), "it is only through the joint consideration of adaptive and maladaptive processes within the individual that it becomes possible to speak in meaningful terms about the existence, nature, and boundaries of the underlying psychopathology" (p. 335). To date, far greater attention has been devoted to the description and classification of psychopathology in children than to healthy child functioning; to nonpathological psychosocial problems related to emotional upset, misbehavior, and learning; or to factors that promote the successful resolution of developmental tasks (Adelman, 1995; Sonuga-Barke, 1998). In light of this imbalance, there is a need for studies of normal developmental processes (Lewis, 2000), for investigations of normative and representative community samples of children (Ialongo, Kellam, & Poduska, 2000; Kazdin, 1989), and for studies of "resilient" children who show normal development in the face of adversity (Masten & Cicchetti, 2010).

Developmental Continuities and Discontinuities

A central issue for theory and research in child psychopathology concerns the continuity of disorders identified from one time to another and the relationship between child, adolescent, and adult disorders (Caspi, 2000; Rutter, Kim-Cohen, & Maughan, 2006; Schulenberg, Sameroff, & Cicchetti, 2004). Some childhood disorders, such as intellectual disability and autism spectrum disorder, are typically chronic conditions that will persist throughout childhood and into adulthood. Other disorders, such as functional enuresis and encopresis, occur during childhood and only rarely manifest themselves in adults (Walker, 2003). However, most disorders (e.g., mood disorders, schizophrenia, generalized anxiety disorder) are expressed, albeit in modified forms, in both childhood and adulthood and exhib-

it varying degrees of continuity over time. Evidence in support of the continuity between child and adult disorders is equivocal and depends on a number of methodological factors related to research design, assessment instruments, the nature of the study sample, and the type and severity of the disorder (Garber, 1984). In general, the literature suggests that child psychopathology is continuous with adult disorders for some, but not all, problems. As we discuss below, there is evidence that appears to favor the stability of externalizing problems over internalizing problems. However, previous findings may reflect the severity and pervasiveness of the disorders assessed, referral biases, and the fact that longitudinal investigations of children with internalizing and other disorders are just beginning to emerge. For example, recent longitudinal studies have found that anxiety disorders in childhood predict a range of psychiatric disorders in adolescence (e.g., Bittner et al., 2007). In another report, early-onset bulimia nervosa was associated with a 9-fold increase in risk for late-adolescent bulimia nervosa and a 20-fold increase in risk for adult bulimia nervosa (Kotler, Cohen, Davies, Pine, & Walsh, 2001).

The possible mechanisms underlying the relationships between early maladaptation and later disordered behavior are numerous and can operate in both direct and indirect ways (Garber, 1984; Rutter, 1994a; Sroufe & Rutter, 1984). Some examples of *direct* relationships between early and later difficulties include (1) the development of a disorder during infancy or childhood, which then persists over time; (2) experiences that alter an infant's or child's physical status (e.g., neural plasticity), which in turn influences later functioning (Courchesne, Chisum, & Townsend, 1994; Johnson, 1999; Nelson, 2000); and (3) the acquisition of early patterns of responding (e.g., compulsive compliance, dissociation) that may be adaptive in light of a child's current developmental level and circumstances, but may result in later psychopathology when circumstances change and new developmental challenges arise.

Some examples of *indirect* associations between child and adult psychopathology may involve early predispositions that eventually interact with environmental experiences (e.g., stressors), the combination of which leads to dysfunction. For example, Egeland and Hiester (1995) found that the impact of day care on disadvantaged high-risk children at 42 months of age was related to the children's attachment quality at 12 months of age, with securely attached children more likely to be negatively affected by early out-of-home

care. Other examples of indirect links between child and adult disturbance include (1) experiences (e.g., peer rejection) that contribute to an altered sense of self-esteem (DuBois & Tevendale, 1999), or that create a negative cognitive set, which then leads to later difficulties; and (2) experiences providing various opportunities or obstacles that then lead to the selection of particular environmental conditions, and by doing so guide a child's course of development (Rutter, 1987; Sroufe & Rutter, 1984).

Research efforts have focused not only on the continuities and discontinuities in childhood disorders, but also on the identification of factors that predict them. One factor that has been studied in the context of conduct disorder is age of onset, with early onset usually viewed as the occurrence of conduct disorder symptoms prior to age 10 years (American Psychiatric Association [APA], 2013b). It has been found that early onset of conduct disorder symptoms is associated with higher rates and more serious antisocial acts over a longer period of time for both boys and girls (Lavigne et al., 2001). However, there may be different subgroups of children with an early onset, and dispositional and psychosocial variables that are present prior to and following onset may influence the seriousness and chronicity more than age of onset per se does (Frick & Viding, 2009; Tolan & Thomas, 1995). A question that needs to be addressed is this: Does early age of onset operate in a causal fashion for later problems, and if so, how? Another issue is whether the causal processes that are associated with an early onset of a disorder (e.g., depression) are different from those that serve to maintain the disorder. Even then, the specification of an age of onset need not be made so precisely that it creates a false distinction that only valid cases meet this precise threshold, as may have happened with ADHD (see Nigg & Barkley, Chapter 2, this volume). Such efforts to impose precision where none exists may have backfired by hampering studies of teens and adults having the same disorder who cannot adequately recall such a precise onset, and by presuming that cases having qualitatively identical symptoms and impairments but later onsets are invalid instances of a disorder.

Although research supports the notion of continuity of disorders, it does not support the continuity of identical symptoms over time (i.e., “homotypic correspondence”). Continuity over time for patterns of behavior rather than for specific symptoms is the norm. For example, although externalizing disorders in boys are stable over time, the ways in which these behavioral

patterns are expressed are likely to change dramatically over the course of development (Olweus, 1979). Even with wide fluctuations in the expression of behavior over time, “children may show consistency in their general adaptive or maladaptive pattern of organizing their experiences and interacting with the environment” (Garber, 1984, p. 34). Several research findings can be used to illustrate this notion of consistent “patterns of organization.” For example, early, heightened levels of behavioral inhibition may affect later adjustment by influencing the way in which a child adapts to new and unfamiliar situations and the ensuing person–environment interactions over time (Kagan, 1994a). Another example of a consistent pattern of organization involves early attachment quality and the development of internal working models that children carry with them into their later relationships (Bowlby, 1988; Goldberg, 1991). Internal working models of self and relationships may remain relatively stable over time, at the same time that the behavioral expressions of these internal models change with development. From a neuroscientific perspective, Pennington and Ozonoff (1991) argue that certain genes and neural systems also play a significant predisposing role in influencing the continuity of psychopathology, and that the “discontinuities at one level of analyses—that of observable behavior—may mask continuities at deeper levels of analysis; those concerned with the mechanisms underlying observable behavior” (p. 117).

Given that developmental continuity is reflected in general patterns of organization over time rather than in isolated behaviors or symptoms, the relationships between early adaptation and later psychopathology are not likely to be direct or uncomplicated. The connections between psychopathology in children and adults are marked by *both* continuities and discontinuities. The degree of continuity–discontinuity will vary as a function of changing environmental circumstances and transactions between a child and the environment that affect the child's developmental trajectory.

Developmental Pathways

The concept of “developmental pathways” is crucial for understanding continuities and discontinuities in psychopathology. Such pathways are not directly observable, but function as metaphors that are inferred from repeated assessments of individual children over time and used as a framework for synthesis and integration (Loeber, 1991; Pickles & Hill, 2006). A pathway, ac-

According to Loeber (1991), “defines the sequence and timing of behavioral continuities and transformations and, ideally, summarizes the probabilistic relationships between successive behaviors” (p. 98). In attempting to identify developmental pathways as either “deviant” or “normal,” it is important to recognize that (1) different pathways may lead to similar expressions of psychopathology (i.e., “equifinality”); and (2) similar initial pathways may result in different forms of dysfunction (i.e., “multifinality”), depending on the organization of the larger system in which they occur (Cicchetti & Rogosch, 1996; Lewis, 2000; Loeber, 1991).

Research findings related to child maltreatment provide an example of a developmental pathway to maladaptive outcomes, with the important qualification that most children who are abused do not exhibit these negative outcomes. The question of why some children seem particularly susceptible to the impact of abuse has led to the search for susceptibility factors, such as child genetic markers (see Wekerle et al., Chapter 16, this volume). However, it is known that physically abused children are more likely to develop insecure attachments, to view interpersonal relationships as coercive and threatening, to become vigilant and selectively attend to hostile cues, to classify others instantly as threatening or nonthreatening, and to acquire aggressive behavioral strategies for solving interpersonal problems (see Cicchetti & Manly, 2001). These children bring representational models to peer relationships that are negative, conflictual, and unpredictable. They process social information in a biased and deviant manner, and develop problems with peer relationships that involve social withdrawal, unpopularity, and overt social rejection by peers (Dodge, Pettit, & Bates, 1994). In another example of a developmental pathway, the diagnosis of conduct disorder typically precedes the initiation of use of various substances, and this use in turn precedes the diagnosis of alcohol dependence in adolescents (Kuperman et al., 2001). Tragically, this can, in turn, exacerbate risk for persistent antisocial behavior by virtue of the reciprocal influences of alcohol dependence on antisocial behavior and vice versa (Barkley, Fischer, Smallish, & Fletcher, 2004).

The systematic delineation of developmental pathways not only offers several advantages for the study of the etiology and outcomes of childhood disorders, but may also suggest strategies for intervention. Loeber (1991, p. 99) describes these advantages as “attempts to capture the changing manifestations and variable phenotype of a given disorder” over time. In this way, the

study of developmental pathways includes etiological considerations, the assessment of comorbidities as they accrue over time, and a sensitivity to diverse outcomes (e.g., White, Bates, & Buyske, 2001).

Risk and Resilience

Previous studies of child psychopathology focused on elucidating the developmental pathways for deviancy and maladjustment, to the relative exclusion of those for competency and adjustment (but see, for exceptions, Luthar, 1993; Rutter, 1985, 1987, 1994b; Rutter & Rutter, 1993). However, a significant number of children who are at risk do *not* develop later problems. There is a growing recognition of the need to examine not only risk factors, but also those conditions that protect vulnerable children from dysfunction and lead to successful adaptations despite adversity (Cicchetti & Garnezy, 1993; Masten & Wright, 2010).

“Resilience,” which refers to successful adaptation in children who experience significant adversity, has now received a good deal of attention (Luthar, Cicchetti, & Becker, 2000). Early patterns of adaptation influence later adjustment in complex and reciprocal ways. Adverse conditions, early struggles to adapt, and failure to meet developmental tasks do not inevitably lead to negative outcomes. Rather, many factors can provide turning points whereby success in a particular developmental task (e.g., educational advances, peer relationships) shifts a child’s course onto a more adaptive trajectory. Conversely, numerous events and circumstances, and underlying dynamic biological systems, may shape a child’s developmental trajectory toward maladaptation (e.g., a dysfunctional home environment, peer rejection, difficulties in school, parental psychopathology, intergenerational conflict, and genetic effects).

Although the term “resilience” has not been clearly operationalized, it is generally used to describe children who (1) manage to avoid negative outcomes and/or to achieve positive outcomes despite being at significant risk for the development of psychopathology; (2) display sustained competence under stress; or (3) show recovery from trauma (Werner, 1995). Risk is usually defined in terms of child characteristics that are known to be associated with negative outcomes—for example, difficult temperament (Ingram & Price, 2001; Rothbart, Ahadi, & Evans, 2000)—and/or in terms of a child’s exposure to extreme or disadvantaged environmental conditions (e.g., poverty or abuse). Individual children who are predisposed to develop psychopathology, and

who show a susceptibility to negative developmental outcomes under high-risk conditions, are referred to as “vulnerable.” Genetic makeup and temperament are two factors that are presumed to contribute to susceptibility for children who are exposed to high-risk environments (Rutter, 1985; Seifer, 2000).

Further complicating such models are recent findings suggesting that certain genetic variants and temperament traits may serve not simply as markers of vulnerability to high-risk environments, but as markers of differential susceptibility to an array of positive and negative contexts (Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011). The notion of differential susceptibility to the environment means that some individual-difference factors will be linked to both especially negative *and* positive outcomes for children, depending on whether the early environment is harsh or one of nurturing support. In contrast, other children lacking such markers of plasticity (Ellis & Boyce, 2008) will tend to have intermediate outcomes regardless of the quality of the early environment. These two types of children (i.e., those highly responsive to their environments vs. those more resistant to environmental influence) have been compared to the delicate orchid and the hardy dandelion (Ellis & Boyce, 2008), with the so-called “dandelion” children exhibiting resilience in the context of early adversity.

Research on resilience has lacked a consistent vocabulary, conceptual framework, and methodological approach (Luthar et al., 2000; Masten, 2011; Rutter, 2000). It is particularly important to note that resilience is not defined as a universal, categorical, or fixed attribute of a child, but rather as a number of different types of dynamic processes that operate over time. Individual children may be resilient in relation to some specific stressors but not others, and resilience may vary over time and across contexts (Rutter, 2012). Models of resilience have increasingly begun to address the complex and dynamic relationships between the child and his or her environment, to incorporate the theoretical and empirical contributions of developmental psychology, and to acknowledge the multiple factors related to normal and deviant behavior (Rutter, 2006; Shiner & Masten, 2012).

One problem in research on resilience has been an absence of agreed-upon criteria for defining positive developmental outcomes (see Kaufman, Cook, Arny, Jones, & Pittinsky, 1994, for a review of the ways in which positive outcomes in studies of resilience have been operationalized). For example, there is currently

debate as to whether the criteria for defining resilience and adaptation should be based on evidence from external criteria (e.g., academic performance), internal criteria (e.g., subjective well-being), or some combination of these (see Masten, 2001). Variations across studies in the source of information (e.g., parent or teacher); the type of assessment method (e.g., interview, questionnaire, observation); the adaptational criteria used; and the number and timing of assessments can easily influence the proportion of children who are designated as resilient or not in any particular investigation (Kaufman et al., 1994; Masten, 2001). In addition, there is also some confusion about, and circularity in, how the term “resilience” has been used, in that it has been used to refer both to an outcome and to the cause of an outcome. Furthermore, in instances in which resilience is used to refer to qualities of children that are putative markers of the capacity for positive adaptation despite adversity, it is important that such markers reflect capture more than the simple absence of vulnerability in order for them to have unique incremental validity for child outcomes beyond models of risk (see next section).

Several different models of resilience have also been proposed, the most common ones being a compensatory model, a challenge model (e.g., stress inoculation), and a protective-factors model (Garmezy, Masten, & Tellegen, 1984). Years of research suggest that resilience is not indicative of any rare or special qualities of a child per se (as implied by the term “the invulnerable child”), but rather is the result of the interplay of normal developmental processes such as brain development, cognition, personality development, caregiver–child relationships, regulation of emotion and behavior, and the motivation for learning (Masten, 2001). Some researchers have argued that resilience may be more ubiquitous than previously thought, and that this phenomenon is part of the “ordinary magic” and makeup of basic human adaptation (Masten, 2001; Sheldon & King, 2001). It is when these adaptational systems are impaired, usually through prolonged or repeated adversity, that the risk for childhood psychopathology increases.

Finally, the possibility that children may actually benefit from exposure to mild to moderate levels of stress has been proposed (e.g., Rutter, 2012; Taleb, 2012), but is not well understood from the standpoint of empirical research. In brief, the notion behind this hypothesis is that the experience of stress enables children to develop coping and other skills that permit them to

manage future stressors more successfully; if so, overly protective, hypervigilant parenting styles would have a negative impact on children, in part by preventing them from having such experiences. While this idea has common-sense appeal and complements exposure-based approaches to treating anxiety and other psychological problems, it has yet to accrue much in the way of research attention; it thus represents an important future direction in work on how stress influences child development.

Protective and Vulnerability Factors

Various protective and vulnerability factors have been found to influence children's reactions to potential risk factors or stressors (Kim-Cohen & Gold, 2009; Luthar, 2006). These include factors within the child, the family, and the community (Osofsky & Thompson, 2000; Werner & Smith, 1992). An example of a within-child risk factor would be cases in which individual differences in genetic risk moderate associations between adversity and negative outcomes (e.g., Brody et al., 2014). Common risk factors that have been found to have adverse effects on a child encompass both acute stressful situations and chronic adversity; they include such events as chronic poverty, poor caregiving, parental psychopathology, death of a parent, community disasters, homelessness, reduced social support, decreased financial resources, family breakup, parental marital/couple conflict, and perinatal stress (Brennan et al., 2008; Deater-Deckard & Dunn, 1999; Luecken & Lemery, 2004; Repetti, Taylor, & Seeman, 2002; Rutter, 1999; Tebes, Kaufman, Adnopoz, & Racusin, 2001).

Protective factors within a child that have been identified include an "easy" temperament (i.e., a child who is energetic, affectionate, cuddly, good-natured, and/or easy to deal with), which makes the child engaging to other people; early coping strategies that combine autonomy with help seeking when needed; high intelligence and scholastic competence; effective communication and problem-solving skills; positive self-esteem and emotions; high self-efficacy; genetic factors (i.e., Dodge & Sherrill, 2007); and the will to be or do something (Fredrickson, 2001; Gilgun, 1999). An example of possible protective factors within the child is seen in findings that high respiratory sinus arrhythmia in conjunction with high skin conductance—taken as indices of a child's ability to self-regulate via self-soothing, focused attention, and organized and goal-directed behavior—can buffer children from the

increases in internalizing symptoms associated with exposure to parental marital conflict (El-Sheikh et al., 2013).

At a family level, protective factors that have been identified include the opportunity to establish a close relationship with at least one person who is attuned to the child's needs; positive parenting; availability of resources (e.g., child care); a talent or hobby that is valued by adults or peers; and family religious beliefs that provide stability and meaning during times of hardship or adversity (Werner & Smith, 1992). Protective factors in the community include extrafamilial relationships with caring neighbors, community elders, or peers; an effective school environment, with teachers who serve as positive role models and sources of support; and opening of opportunities at major life transitions (e.g., adult education, voluntary military service, church or community participation, a supportive friend or marital/relationship partner).

In summary, early patterns of adaptation influence later adjustment in complex and reciprocal ways. Adverse conditions, early adaptational struggles, and failure to meet developmental tasks do not inevitably lead to a fixed and unmalleable dysfunctional path (Rutter, 2007a). Rather, as noted earlier, many different factors can act to alter a child's developmental course for the better. Conversely, numerous events and circumstances may serve to alter this course for the worse.

The interrelated issues of developmental continuities—discontinuities; of developmental pathways; of risk, resilience, and antifragility; and of vulnerability and protective factors are far from being resolved or clearly understood. The multitude of interdependent and reciprocal influences, mechanisms, and processes involved in the etiology and course of child psychopathology clearly suggest a need for more complex theories (e.g., chaos theory, nonlinear dynamic models) (Granic, 2005; Glantz & Johnson, 1999), research designs, and data-analytic strategies (Rutter, 2007b; Singer & Willett, 2003).

Contextual Influences

Messick (1983) cogently argued that any consideration of child psychopathology must consider and account for three sets of contextual variables: (1) the child *as* context—the idea that unique child characteristics, predispositions, and traits influence the course of development; (2) the child *of* context—the notion that the child comes from a background of interrelated family, peer,

classroom, teacher, school, community, and cultural influences; and (3) the child *in* context—the understanding that the child is a dynamic and rapidly changing entity, and that descriptions taken at different points in time or in different situations may yield very different information.

Research has increasingly come to recognize the reciprocal transactions between the developing child and the multiple social and environmental contexts in which development and psychopathological symptoms occur (Deater-Deckard, 2001; Dirks, De Los Reyes, Briggs-Gowan, Cella, & Wakschlag, 2012). Understanding context requires a consideration of events that impinge directly on the child in a particular situation at a particular point in time; extrasituational events that affect the child indirectly (e.g., a parent’s work-related stress); and temporally remote events that continue to affect the child through their representation in the child’s current cognitive–affective database.

Certainly, relatively straightforward aspects of the physical context are known to affect child development (e.g., diet, lead; Chandramouli, Steer, Ellis, & Emond, 2009; Grantham-McGregor & Baker-Henningham, 2005). However, defining context has been, and continues to be, a matter of some complexity. The context of maltreatment provides an illustration of difficulties in definition. Maltreatment can be defined in terms of its type, timing, frequency, severity, and chronicity in the family (e.g., Manly, Kim, Rogosch, & Cicchetti, 2001). Each of these parameters and their interaction may contribute to child outcomes, but in different ways. For example, Manly, Cicchetti, and Barnett (1994) studied different types of maltreatment and found that outcomes generally did not differ for children who were categorized as neglected versus abused. However, a regression analysis indicated that neglect accounted for more of the variance in child problems than other types of abuse did. In this study, sexually abused children were also found to be more socially competent than children exposed to other forms of maltreatment. This may reflect a lack of chronicity associated with sexual abuse, or it may suggest that problems related to sexual abuse may not reveal themselves until later periods in a child’s development, when issues concerning sexuality become more salient. Other studies have found that psychological maltreatment and emotional abuse account for most of the distortions in development attributed to maltreatment in general, and have the most negative consequences for a child (Crittenden, Claussen, & Sugarman, 1994).

The example of maltreatment illustrates how contexts for development encompass heterogeneous sets of circumstances, and how child outcomes may vary as a function of (1) the configuration of these circumstances over time, (2) when and where outcomes are assessed, and (3) the specific aspects of development that are affected. More precise definitions are needed if the impact of maltreatment—or, for that matter, any contextual event (e.g., parent disciplinary styles, family support, intellectual stimulation, nutrition)—is to be understood.

Even for those forms of child psychopathology for which there are strong neurobiological influences, the expression of the disorder is likely to interact with contextual demands. For example, Iaboni, Douglas, and Baker (1995) found that although the overall pattern of responding shown by children with ADHD was indicative of a generalized inhibitory deficit, the self-regulatory problems of these children became more evident with continuing task demands for inhibition and/or deployment of effort. Likewise, tasks having high interest value or high external incentives may moderate these children’s typically deficient performance on less interesting or low-incentive tasks (Carlson & Tamm, 2000; Slusarek, Velling, Bunk, & Eggers, 2001).

Child psychopathology research has increasingly focused on the role of the family system, the complex relationships within families, and the reciprocal influences among various family subsystems (Fiese, Wilder, & Bickham, 2000). There is a need to consider not only the processes occurring within disturbed families, but the common and unique ways in which these processes affect both individual family members and subsystems. Within the family, the roles of the mother–child and marital/couple subsystems have received the most research attention to date, with less attention given to the roles of siblings (Hetherington, Reiss, & Plomin, 1994) and fathers (Phares, Rojas, Thurston, & Hankinson, 2010). For the most part, research into family processes and child psychopathology has not kept pace with family theory and practice, and there is a need for the development of sophisticated methodologies and valid measures that will capture the complex relationships hypothesized to be operative in disturbed and normal family systems (Bray, 1995; Bray, Maxwell, & Cole, 1995). This task is complicated by a lack of consensus concerning how healthy family functioning or family dysfunction should be defined; what specific family processes are important to assess (Mash & Johnston, 1995); or the extent to which such measures of fam-

ily environment reflect true environmental effect or shared genetic influences between parent and child (Plomin, 1995).

DEFINING CHILD PSYCHOPATHOLOGY

There has been, and continues to be, a lack of consensus concerning how psychopathology in children should be defined (Angold & Costello, 2009; Rutter, 2011). Despite ongoing debate, for pragmatic purposes, researchers and clinicians typically define child psychopathology using standardized diagnostic systems such as the most recent revision of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association [APA], 2013a) and the *International Classification of Diseases*, 10th revision (ICD-10; WHO, 2010). The diagnostic criteria utilized in DSM-5 are the ones most commonly used in North America, and these are presented for the individual disorders described in each subsequent chapter of this volume. However, the increased use and acceptance of DSM-5 and its predecessors should not be taken as an indication of widespread agreement regarding the fundamental nature of what constitutes psychopathology in children or the specific criteria used to define it (cf. Coghill & Sonuga-Barke, 2012; Hudziak et al., 2007; Rutter, 2011). In many ways, the acceptance and use of DSM-5 seems to reflect a degree of resignation on the part of many researchers and clinicians concerning the prospects for developing a widely agreed-upon alternative approach. Nevertheless, alternative approaches are being advanced that apply current research findings toward the development of classification frameworks for psychopathology (Insel et al., 2010; Sanislow et al., 2010), as discussed later in this chapter.

Several fundamental questions have characterized most discussions concerning how child psychopathology should be defined:

1. Should child psychopathology be viewed as a disorder that occurs within the individual child (e.g., disorder of the brain, psychological disturbance), as a relational disturbance, as a reaction to environmental circumstances, or (as is likely) some combination of all these?

2. Does child psychopathology constitute a condition qualitatively different from normality (aberration),

an extreme point on a continuous trait, a delay in the rate at which a normal trait would typically emerge, or some combination of the three? How are “subthreshold” problems to be handled?

3. Can homogeneous disorders be identified? Or is child psychopathology best defined as a configuration of co-occurring disorders or as a profile of traits and characteristics?

4. Can child psychopathology be defined as a static entity at a particular point in time, or do the realities of development necessitate that it be defined as a dynamic and ongoing process that expresses itself in different ways over time and across contexts?

5. Is child psychopathology best defined in terms of its current expression, or do definitions also need to incorporate nonpathological conditions that may constitute risk factors for later problems? This question is especially relevant when considering disorder and risk for disorder in infants and toddlers (see Lyons-Ruth, Zeanah, Benoit, Madigan, & Mills-Koonce, Chapter 15, this volume).

There are currently no definitive answers to these questions. More often, the way in which they are answered reflects theoretical or disciplinary preferences and utility, such as specific purposes and goals (e.g., defining samples for research studies, or determining program or insurance eligibility).

Psychopathology as Adaptational Difficulty

As we have noted earlier, a common theme in defining child psychopathology has been that of adaptational difficulty or failure (Garber, 1984; Mash, 1998). Sroufe and Rutter (1984) note that regardless of whether “particular patterns of early adaptation are to a greater or lesser extent influenced by inherent dispositions or by early experience, they are nonetheless patterns of adaptation” (p. 23). Developmental competence is reflected in a child’s ability to use internal and external resources to achieve a successful adaptation (Masten, Burt, & Coatsworth, 2006; Waters & Sroufe, 1983), and problems occur when the child fails to adapt successfully. Even with wide variations in terminology and proposed explanatory mechanisms across theories, there is general agreement that maladaptation represents a pause, a regression, or a deviation in development (Garber, 1984; Simeonsson & Rosenthal, 1992).

In conceptualizing and defining psychopathology as adaptational difficulty, it is also essential to conceptualize and identify the specific developmental tasks and challenges that are important for children at various ages and periods of development, and the many contextual variables that derive from and surround the child (Garber, 1984; Luthar, Burack, Cicchetti, & Weisz, 1997; Mash, 1998). In this regard, the study of psychopathology in children and the study of development and context are for all intents and purposes inseparable (Cicchetti & Aber, 1998).

In determining whether a given behavior should be considered to be deviant in relation to stage-salient developmental issues, Garber (1984) stresses the need to understand several important parameters. The first, “intensity,” refers to the magnitude of behavior as excessive or deficient. The second, “frequency,” refers to the severity of the problem behavior, or how often it does or does not occur. Third, the “duration” of behavior must be considered. Some difficulties are transient and spontaneously remit, whereas others persist over time. To these parameters, we would add a qualitative parameter reflecting how grossly atypical the behavior may be (e.g., some of the complex compulsions seen in Tourette’s disorder), such that even low-intensity, low-frequency, and short-duration behavior may be so bizarre as to constitute “psychopathology.” It is crucial that the intensity, frequency, duration, and atypicality of the child’s behavior be appraised with respect to what is considered normative for a given age (e.g., the developmental appropriateness of a behavior). The final parameter of deviance concerns the “number of different symptoms” and their “configuration.” Each of these parameters is central to research and theory, and to one’s specific definition of adaptational failure, regression, stagnation, or deviation.

Social Judgment

The diagnosis of psychopathology in children is almost always a reflection of both the characteristics and behavior of the child *and* of significant adults and professionals (Lewis, 2000). Research findings utilizing behavior problem checklists and interviews indicate that there can be considerable disagreement across informants (e.g., parents, teachers, professionals) concerning problem behaviors in children (Achenbach, McConaughy, & Howell, 1987; Feiring & Lewis, 1996; Youngstrom, 2013). Mothers typically report more

problems than do fathers (e.g., Achenbach et al., 1991), and across a range of domains, teachers identify more problems than other informants do in assessing the same domains. For example, in a study with maltreated children, only 21% were classified as resilient by teachers, whereas 64% were so classified based on reports from other sources (Kaufman et al., 1994).

Issues regarding disagreement–agreement among informants are complicated by the fact that the amount of agreement will vary with the age and sex of the child (Offord, Boyle, & Racine, 1989), the nature of the problem being reported on (e.g., internalizing vs. externalizing; De Los Reyes & Kazdin, 2005), the method used to gather information (e.g., interview vs. questionnaire), and the informants being compared. For example, Tarullo, Richardson, Radke-Yarrow, and Martinez (1995) found that both mother–child and father–child agreement was higher for preadolescent than for adolescent children; in a meta-analysis, Duhig, Renk, Epstein, and Phares (2000) reported higher mother–father agreement for externalizing than for internalizing problems. Disagreements among informants create methodological difficulties in interpreting epidemiological data when such data are obtained from different sources, and also in how specific diagnoses are arrived at in research and practice. For most research studies, the practice tends to be to consider a symptom present if any informant endorses it as such (e.g., Costello, Mustillo, et al., 2003).

Also of importance is how disagreements among informants are interpreted (De Los Reyes, 2011). For example, disagreements may be viewed as (1) reflections of bias or error on the part of one informant; (2) evidence for the variability of children’s behavior across the situations in which they are observed by others; (3) lack of access to certain types of behavior (i.e., private events) on the part of one informant; (4) denial of the problem; or (5) active distortion of information in the service of some other goal (e.g., defensive exclusion, treatment eligibility).

Parental psychopathology may “color” descriptions of child problems—as may occur when abusive or depressed mothers provide negative or exaggerated descriptions of their children (Gotlib & Hammen, 1992; Mash et al., 1983; Richters, 1992; Youngstrom, Izard, & Ackerman, 1999), or when dismissive/avoidant adult informants deny the presence of emotional problems at the same time that professionals observe a high level of symptoms (Dozier & Lee, 1995). These latter types of problems in reporting may be especially likely, given

the frequent lack of correspondence between the expression and the experience of distress for many child and adult disturbances. Hypothesized relationships between parental psychopathology and reports of exaggerated child symptoms have received mixed support. For example, some studies have failed to find evidence for distorted reports by depressed mothers (Tarullo et al., 1995). However, recent work (Durbin & Wilson, 2012) examining maternal ratings of child behavior also coded by objective raters found that mothers' lifetime psychiatric diagnoses and personality traits were associated with their reports of child emotional behavior, and that for some emotions, mothers' mental health and dispositional variables were more strongly related to their reports of their children's emotions than were objective indices of the children's observable emotional behavior. Related work (Hayden, Durbin, Klein, & Olino, 2010) indicates that maternal characteristics, such as mothers' own personality traits, influence the extent to which they successfully encode and/or report on analogous child behaviors. Intriguingly, the extent to which informant discrepancies are present regarding child behavior may predict poor child outcomes, above and beyond individual informants' reports of children (De Los Reyes, 2011). Thus, while it is well known that informant discrepancies exist, the meaning of these discrepancies and their implications for child outcomes requires further study.

APPROACHES TO CONCEPTUALIZING CHILD PSYCHOPATHOLOGY

The types of problems for which children are referred for treatment are reflected in the different approaches that have been used to conceptualize and classify these problems. Among the more common of these approaches are the following:

1. General and specific behavior problem checklists, which enumerate individual child symptoms—for example, the Child Behavior Checklist (Achenbach & Rescorla, 2001) and the Children's Depression Inventory 2 (Kovacs, 2010).
2. Dimensional approaches, which focus on symptom clusters or syndromes, derived from behavior problem checklists—for example, the Child Behavior Checklist and Profile (Achenbach, 1993; Achenbach & Rescorla, 2001).
3. Categorical approaches, which use predetermined diagnostic criteria to define the presence or absence of particular disorders—for example, DSM-5 (APA, 2013a) and ICD-10 (WHO, 2010).⁵
4. A multiple-pathway, developmental approach, which emphasizes developmental antecedents and competencies both within the child and the environment that contribute to (mal)adjustment and (mal)adaptation (Sroufe, 1997).

Issues related to the use of these different classification approaches are discussed in a later section of this chapter. What follows is a brief overview of the types of problem behaviors, dimensions, and disorders that occur during childhood and that are the topics of this volume's other chapters.

Individual Symptoms

For the most part, individual behavioral and emotional problems (i.e., symptoms) that characterize most forms of child psychopathology occur in almost all children at one time or another during their development (e.g., Achenbach & Edelbrock, 1981; Achenbach et al., 1991; MacFarlane et al., 1954). When taken in isolation, specific symptoms have generally shown little correspondence to a child's overall current adjustment or to later outcomes. This is the case even for many symptoms hypothesized to be significant indicators of psychopathology in children in earlier decades—for example, thumbsucking after 4 years of age (Friman, Larzelere, & Finney, 1994). Usually the age-appropriateness, clustering, and patterning of symptoms are what serve to define child psychopathology, rather than the presence of individual symptoms.

Many of the individual behavior problems displayed by children referred for treatment are similar to those that occur in less extreme forms in the general population or in children of younger ages. For example, Achenbach and colleagues (1991) found that although referred children scored higher than nonreferred children on 209 of 216 parent-rated problems, only 9 of the 209 items showed effects related to clinical status that were considered to be large (accounting for more than 13.8% of the variance), according to criteria specified by Cohen (1988). Examples of parent-reported individual symptoms that were more common in referred than in nonreferred children and that accounted for 10% or more of the variance in clinical status included “sad or

depressed,” “uncooperative,” “nervous,” “high-strung, or tense,” “feels he/she can’t succeed,” “feels worthless or inferior,” “disobedient at school,” “easily distracted,” “lies,” “fails to finish things he/she starts,” “defiant,” and “doesn’t get along with other kids” (Achenbach et al., 1991). As can be seen, even the problems that best discriminated between referred and nonreferred children are relatively common behaviors that occur to some extent in all children; they are not particularly strange or unusual behaviors. In addition, most *individual* problem behaviors (approximately 90% of those on behavior problem checklists) do not, by themselves, discriminate between groups of clinic-referred and nonreferred children. Nondiscriminating items include some problems for children in both groups that are relatively common (e.g., “brags,” “screams”) and others that occur less frequently (e.g., “sets fires,” “bowel movements outside the toilet”).

Dimensions of Child Psychopathology

A second approach to describing child psychopathology identifies symptom clusters or “syndromes” derived through the use of multivariate statistical procedures, such as factor analysis or cluster analysis (e.g., Achenbach, 1993, 1997; McDermott, 1993; McDermott & Weiss, 1995). Research has identified two broad dimensions of child psychopathology—one reflecting “externalizing” or “undercontrolled” problems, and the other reflecting “internalizing” or “overcontrolled” problems (Reynolds, 1992). The externalizing dimension encompasses behaviors often thought of as directed at others, whereas the internalizing dimension describes feelings or states that are commonly viewed as “inner-directed.” The presence of these two dimensions may account for the pervasive comorbidity found between internalizing (e.g., depression and anxiety) and externalizing (e.g., oppositional and conduct problems) disorders; moreover, extensions of this research applied to adults suggest that a similar structure may characterize adult psychopathology (Krueger & Markon, 2006; although see Kotov et al., 2011), thus supporting the lifespan continuity of this dimensional structure. Within the two broad dimensions of externalizing and internalizing disorders, there may be further subdivisions or syndromes, including anxious/depressed (e.g., “crying,” “fearful of multiple situations”), withdrawn/depressed (e.g., “enjoys little,” “withdrawn”), somatic complaints (e.g., “feels dizzy,” “tired”), social prob-

lems (e.g., “lonely,” “gets teased”), thought problems (e.g., “hears or sees things”), attention problems (e.g., “problems sitting still or attending”), rule-breaking behavior (e.g., “steals,” “swears”), and aggressive behavior (e.g., “argumentative,” “physically aggressive”) (Achenbach & Rescorla, 2001).

Categories of Child Psychopathology

The DSM-5 diagnostic system (APA, 2013a) provides comprehensive coverage of the general types of symptom clusters displayed by children characterized as having mental disorders. To illustrate, DSM-5 categories that apply to children are listed in Tables 1.1–1.3. These tables are not intended to be exhaustive of all DSM-5 diagnoses that may apply to children. Rather, they are intended to provide an overview of the range and variety of disorders that typically occur during childhood. Specific DSM-5 disorders and their subtypes are discussed in detail in the subsequent chapters of this volume.

Table 1.1 lists the DSM-5 categories for neurodevelopmental disorders, including intellectual disability, communication disorders (e.g., language disorder), autism spectrum disorder, ADHD, specific learning disorder, and motor disorders. Most of these disorders are early-emerging, often co-occurring conditions characterized by deficits and delays in attaining developmental milestones, and are associated with a range of impairments in multiple domains of functioning (e.g., social, academic). An array of specifiers, such as age of onset and severity, can be applied to provide further detail to the clinical description of individual patients and to aid prediction of the disorder’s course. Whether the disorder is accompanied by a medical or genetic condition or an environmental factor with potential etiological significance (e.g., fetal alcohol exposure) can also be noted as part of the diagnosis.

Table 1.2 is a noncomprehensive list of DSM-5 categories for other disorders that can be diagnosed in children or adolescents (e.g., schizophrenia, depressive disorders, bipolar and related disorders, anxiety disorders). It is noteworthy that, unlike its immediate predecessors, the DSM-5 does not contain a separate section on disorders of infancy and childhood; instead, disorders previously located in this section in DSM-IV are now found in the section for neurodevelopmental disorders (e.g., ADHD) or are integrated in other sections throughout the manual (e.g., separation anxiety

TABLE 1.1. DSM-5 Categories for Neurodevelopmental Disorders

<u>Intellectual disabilities</u>
Intellectual disability (intellectual developmental disorder)
Global developmental delay
Unspecified intellectual disability
<u>Communication disorders</u>
Language disorder
Speech sound disorder
Childhood-onset fluency disorder (stuttering)
Social (pragmatic) communication disorder
Unspecified communication disorder
<u>Autism spectrum disorder</u>
Autism spectrum disorder
<u>Attention-deficit/hyperactivity disorder</u>
Attention-deficit/hyperactivity disorder
Other specified attention-deficit/hyperactivity disorder
Unspecified attention-deficit/hyperactivity disorder
<u>Specific learning disorder</u>
Specific learning disorder
<u>Motor disorders</u>
Developmental coordination disorder
Stereotypic movement disorder
Tourette's disorder
Persistent (chronic) motor or vocal tic disorder
Provisional tic disorder
Other specified tic disorder
Unspecified tic disorder
<u>Other neurodevelopmental disorders</u>
Other specified neurodevelopmental disorder
Unspecified neurodevelopmental disorder

TABLE 1.2. Select DSM-5 Categories for Other Disorders Diagnosed in Infancy, Childhood, or Adolescence

<u>Schizophrenia spectrum and other psychotic disorders</u>
Schizotypal personality disorder; schizophrenia; schizoaffective disorder; schizophreniform disorder; delusional disorder; brief psychotic disorder
<u>Bipolar and related disorders</u>
Bipolar I disorder; bipolar II disorder; cyclothymic disorder
<u>Depressive disorders</u>
Disruptive mood dysregulation disorder; major depressive disorder, single episode or recurrent episodes; persistent depressive disorder (dysthymia)
<u>Anxiety disorders</u>
Separation anxiety disorder; selective mutism; specific phobia; social anxiety disorder (social phobia); panic disorder; agoraphobia
<u>Obsessive-compulsive and related disorders</u>
Obsessive-compulsive disorder; body dysmorphic disorder; hoarding disorder; trichotillomania (hair-pulling disorder); excoriation (skin-picking) disorder
<u>Trauma- and stressor-related disorders</u>
Reactive attachment disorder; disinhibited social engagement disorder; posttraumatic stress disorder; acute stress disorder; adjustment disorders
<u>Feeding and eating disorders</u>
Pica; Rumination disorder; avoidant/restrictive food intake disorder; anorexia nervosa; bulimia nervosa; binge-eating disorder
<u>Elimination disorders</u>
Enuresis; encopresis
<u>Disruptive, impulse-control, and conduct disorders</u>
Oppositional defiant disorder; intermittent explosive disorder; conduct disorder; antisocial personality disorder; pyromania; kleptomania
<u>Substance-related and addictive disorders</u>
Substance use disorders; substance-induced disorders

disorder). This change was made with the goal of emphasizing a lifespan approach to conceptualizing mental disorders, and in recognition of the fact that many disorders can and do manifest themselves across the lifespan (APA, 2013b). Although it is true that boundaries drawn between disorders of childhood and other age groups are arbitrary, and potentially hamper tests of psychopathology continuity over time, the long-term implications of this significant change to DSM organization are unclear. The addition of a specific section dedicated to disorders of childhood to DSM-III is widely regarded as having played a critical role in increasing research interest in childhood disorders; whether removing this distinction will result in a decrease in the level of attention being paid to disorders of children remains to be seen.

Finally, Table 1.3 is a noncomprehensive list of DSM-5 categories for other conditions that are not defined as mental disorders, but may be a focus of clinical attention. We have focused on those with the greatest relevance for childhood or adolescence, in that they emphasize relational problems, maltreatment, and academic and adjustment difficulties.

TABLE 1.3. DSM-5 Categories for Other Conditions That May Be a Focus of Clinical Attention

Relational problems

Problems related to family upbringing (e.g., parent–child relationship problem; child affected by parental relationship distress)

Other problems related to primary support group (e.g., disruption of family by separation or divorce; uncomplicated bereavement)

Abuse and neglect

Child maltreatment and neglect problems (e.g., confirmed and suspected physical and sexual abuse; confirmed and suspected neglect; encounters for mental health services for these problems)

Educational and occupational problems

Educational problems (e.g., academic problems)

Housing and economic problems

Housing problems (e.g., homelessness; inadequate housing)

Economic problems (e.g., lack of adequate food or safe drinking water; extreme poverty; low income)

APPROACHES TO THE CLASSIFICATION AND DIAGNOSIS OF CHILD PSYCHOPATHOLOGY

There is general agreement in medicine, psychiatry, and psychology regarding the need for a system of classifying childhood disorders. However, major areas of contention have arisen around such issues as which disorders should be included in the system, what the optimal strategies are for organizing and grouping disorders, and which specific criteria should be used to define a particular disorder (Achenbach, 1985; Achenbach & Edelbrock, 1989; Mash & Barkley, 2007; Sonuga-Barke, 1998).

The two most common approaches to the diagnosis and classification of child psychopathology involve the use of (1) “categorical” classification systems that are based primarily on informed clinical consensus, an approach that has dominated and continues to dominate the field (APA, 1994, 2000, 2013a); and (2) empirically based “dimensional” classification schemes derived through the use of multivariate statistical techniques (Achenbach, 1993, 1997; Achenbach & Rescorla, 2001). In addition, alternative and/or derivative approaches to classification have been proposed to address perceived deficiencies associated with the use of categorical and dimensional approaches. These have included developmentally based measures (Garber, 1984; Mohr & Regan-Kubinski, 1999; Sroufe, 1997), laboratory and performance-based measures (Frick, 2000), prototype classification (Cantor et al., 1980; Shaffner, 2012), and behavioral classification/functional analysis based on behavioral excesses, deficits, and faulty stimulus control (Mash & Hunsley, 1990; Ringdahl & Falcomata, 2009). Although each of these alternative approaches has something to offer to the classification of childhood disorders, they are generally underdeveloped and unstandardized, and have not been widely accepted or used in either research or practice.

In addition to these alternatives, the limitations of diagnostic systems derived from expert consensus (e.g., DSM-5) have led to both a call for greater emphasis on the underlying neurobiological substrates of psychopathology in classification, and a response, by virtue of the development of the Research Domain Criteria (RDoC; Insel et al., 2010; Sanislow et al., 2010). The RDoC initiative, which was spearheaded by the National Institute of Mental Health, aims to generate research on the biological substrates of psychopathology, with

the goal of developing future classification schemes that map more clearly onto the underlying pathophysiology of disorder. Multiple workshops were held in 2010–2012 with the goal of defining various domains of functioning (e.g., cognitive systems, arousal/regulatory systems), which were further broken down into constructs (e.g., attention, circadian rhythms) that have units of analysis with genetic, molecular, neural, and behavioral levels (Morris & Cuthbert, 2012), although the primary focus of RDoC is on neural circuitry (Insel et al., 2010). It is already known that many if not all of these levels of analysis will cut across disorders as they are traditionally defined, which can be taken as evidence for the failure of current diagnostic systems to “carve nature at its joints” and for the need for the RDoC framework.

One long-term goal of the RDoC initiative is that genetic sequencing, brain imaging, and other laboratory-based approaches will supplant diagnostic systems based on clinical consensus, play a central role in clinical assessments, and directly inform treatment (Insel, 2013; Insel et al., 2010). This is clearly a highly ambitious goal, given the currently limited ability of genetic and neuroimaging findings to predict treatment response or other important clinical outcomes; at present, most constructs with the capacity to predict clinical outcomes (e.g., age of onset, negative life events) would be considered “psychological” or “behavioral” rather than biological. Furthermore, embedded within the RDoC initiative is the notion that mental disorders are disorders of the brain, and can be best understood, and ultimately treated, through the application of clinical neuroscience methodologies and genomics. This viewpoint could be considered reductionistic; at the least, it is an empirical stance that may or may not ultimately be supported by data. Thus, while the core premise behind RDoC (i.e., that contemporary diagnostic systems do not map closely onto etiology, although it would be desirable for them to do so) is not especially controversial, the perceived preeminence of biological approaches to disorder may be to some in the field.

To date, no single classification scheme for childhood disorders has established adequate validity (Cantwell, 1996; Mash & Barkley, 2007; Rutter & Uher, 2012). Many researchers and clinicians have expressed and continue to express concerns that current diagnostic and classification systems (1) underrepresent disorders of infancy and childhood; (2) are inadequate in representing the interrelationships and overlap that exist among many childhood disorders; (3) are not suffi-

ciently sensitive to the developmental, contextual, and relational parameters that are known to characterize most forms of psychopathology in children; and (4) are heterogeneous with respect to etiology (Jensen & Hoagwood, 1997; Kagan, 1997; Rice, 2010).

Categorical Approaches

Categorical approaches to the classification of childhood disorders have included systems developed by the Group for the Advancement of Psychiatry (1974), the WHO (2010), the APA (2013a), and the Zero to Three/National Center for Clinical Infant Programs (2005a). Although a detailed review of all these systems is beyond the scope of this chapter, a brief history of the APA’s development of the DSM approach is presented to illustrate the issues associated with categorical approaches, the growing concern for more reliable classification schemes for childhood disorders, and the evolving conceptualizations of childhood disorders over the past 60 years. Discussion of the DSM approach in relation to specific child and adolescent disorders appears in the chapters that follow. Also, the *Diagnostic Classification of Mental Health and Developmental Disorders of Infancy and Early Childhood-Revised*, or *Diagnostic Classification: 0–3R* (DC:0–3R; Zero to Three/National Center for Clinical Infant Programs, 2005a), is described to illustrate a categorical approach that attempts to integrate developmental and contextual information into the diagnosis of infants’ and young children’s problems.

Development of the DSM Approach

One of the first efforts to collect data on mental illness was in the U.S. census of 1840, which recorded the frequency of a single category of “idiocy/insanity.” Forty years later, seven categories of mental illness were identified: dementia, dipsomania, epilepsy, mania, melancholia, monomania, and paresis (APA, 1994). Much later (in the 1940s), the WHO classification system emerged with the manuals of the ICD, whose sixth revision included, for the first time, a section for mental disorders (APA, 1994; Cantwell, 1996).

In response to perceived inadequacies of the ICD system for classifying mental disorders, the APA’s Committee on Nomenclature and Statistics developed DSM-I in 1952 (APA, 1952). There were three major categories of dysfunction in DSM-I—“organic brain syndromes,” “functional disorder,” and “mental defi-

ciency” (Kessler, 1971)—under which were subsumed 106 categories. The term “reaction” was used throughout the text, which reflected Adolf Meyer’s psychobiological view that mental illness involves reactions of the personality to psychological, social, and biological factors (APA, 1987). Children were largely neglected in the early versions of DSM (Cass & Thomas, 1979; Silk et al., 2000). In fact, DSM-I included only one child category of “adjustment reactions of childhood and of adolescence,” which was included under the heading of “transient situational disorders.”

As reflected in the use of the term “reaction,” psychoanalytic theory had a substantial influence on the classification of both child and adult psychopathology (Clementz & Iacono, 1993). In part, this was due to the fact that the first classification system to focus on childhood psychopathology was developed by Anna Freud in 1965 (see Cantwell, 1996). Although the term “reaction” was eliminated from DSM-II (APA, 1968), a separate section was reserved for classifying neuroses, and diagnoses could be based on either an assessment of the client’s presenting symptomatology or inferences about his or her unconscious processes (Clementz & Iacono, 1993). Once again, apart from conditions subsumed under the adult categories, DSM-II gave little recognition to childhood difficulties except for mental retardation and schizophrenia—childhood type (Cass & Thomas, 1979).

As a formal taxonomy, DSM-III (APA, 1980) represented a substantial departure from, and advance over, earlier editions of the DSM. The first and second editions contained only narrative descriptions of symptoms, and clinicians had to draw on their own definitions for making a diagnosis (APA, 1980); thus interrater reliability of psychiatric diagnoses was quite poor. DSM-III, in which explicit inclusion, exclusion, and duration criteria for each disorder were included, represented a landmark shift of the field aimed at achieving greater diagnostic reliability (Achenbach, 1985; APA, 1980). Moreover, unsubstantiated etiological inferences that were heavily embedded in psychoanalytic theory were dropped, more child categories were included, and a greater emphasis was placed on empirical data (Achenbach, 1985). These changes reflected the beginnings of a conceptual shift in both diagnostic systems and etiological models away from an isolated focus of psychopathology as existing within the child alone, and toward an increased emphasis on his or her surrounding context. DSM-III was revised in 1987 (DSM-III-R) to help clarify the inconsistencies and ambiguities that arose in its use. For

example, empirical data at that time did not support the category of attention deficit disorder *without* hyperactivity as a unique symptom cluster (Routh, 1990), and this category was removed from DSM-III-R. DSM-III-R was also developed to be polythetic, in that a child could be diagnosed with a certain subset of symptoms without having to meet all criteria. This was an important change, especially in light of the heterogeneity and rapidly changing nature of most childhood disorders (Mash & Barkley, 2007). Relative to its predecessors, far greater emphasis was also placed on empirical findings in the development of DSM-IV, particularly for the child diagnostic categories.

In order to bridge the planned 12-year span between DSM-IV and DSM-5, a revision (DSM-IV-TR) of DSM-IV was published in 2000 (APA, 2000). DSM-IV-TR was limited to text revisions (e.g., associated features and disorders, prevalence) and was designed mainly to correct any factual errors in DSM-IV, make sure that information was still current, and incorporate new information since the time the original DSM-IV literature reviews were completed in 1992. In 2013, after a considerable delay, DSM-5 was released.

Although contemporary versions of DSM have included numerous improvements over previous DSMs—with their greater emphasis on empirical research, and more explicit diagnostic criteria sets and algorithms—criticisms have also been raised (e.g., Hyman, 2010; Rutter, 2011; Uher & Rutter, 2012). First, although DSM-5 incorporates greater dimensional representation of disorders than its predecessors, it still relies largely on a categorical scheme that may not always optimally serve children’s needs. For example, it may be necessary for a child to meet specific diagnostic criteria for specific learning disorder in order to qualify for a special education class. However, if the child’s problems are subclinical, or the child’s problems relate to more than one DSM category, then he or she may be denied services (Achenbach, 2000). Useful approaches to the goal of incorporating the strengths of dimensional operationalizations of disorder (e.g., increased information) with those of categorical approaches (e.g., ease of communication) have been proposed (e.g., Kamphuis & Noordhof, 2009) and should be applied more frequently in the field.

Another problem with DSM-5 relates to the wording and the lack of empirical adequacy for certain criterion sets. For example, the words “often” in the criteria for ADHD and conduct disorder, and “persistent” and “excessive” in the criteria for separation anxiety disorder,

are not clearly defined. This ambiguity poses a particular problem when one considers that the primary sources of assessment information are often a child's parents, whose perception and understanding of these terms may be idiosyncratic or inaccurate. This ambiguity and other factors may contribute to the unreliability or unsuitability of the DSM for diagnosing certain childhood disorders (e.g., Nicholls, Chater, & Lask, 2000).

A further difficulty with DSM-5 diagnostic criteria is the lack of emphasis on the situational or contextual factors surrounding and contributing to various disorders. This is a reflection of the fact that DSM-5 continues to view mental disorder as individual psychopathology or risk for psychopathology, rather than in terms of problems in psychosocial adjustment. One problem with respect to the atheoretical nature of DSM is that it has perhaps mistakenly fostered the assumption that a description of symptoms is sufficient for diagnosis, without taking into account natural history, psychosocial correlates, biological factors, or response to treatment (Cantwell, 1996). However, the consideration in DSM-5 of such factors as culture, age, and gender associated with the expression of each disorder is laudable, as is the increased recognition of the importance of family problems and extrafamilial relational difficulties.

Other concerns exist, including the extent to which comorbidity is an artifact of the DSM's polythetic criteria (Angold, Costello, & Erkanli, 1999; Nottelmann & Jensen, 1995), or whether the pendulum has swung too far from not identifying psychopathological conditions in children to overly liberal diagnostic practices that label relatively healthy children as disordered (Silk et al., 2000). It is also the case that ongoing changes in diagnostic criteria based on new findings and other considerations (e.g., eligibility for services) are likely to influence prevalence estimates for many childhood disorders. For example, current estimates of the prevalence of autism spectrum disorder (e.g., Kogan et al., 2009) are substantially higher than even fairly recent, previous ones (e.g., Fombonne, 1999; Tanguay, 2000); this increase is primarily due to a broadening of the criteria used to diagnose autism spectrum disorder, as well as increased recognition of milder forms of the disorder and changes in case-finding approaches (Costello, Foley, & Angold, 2006).

Development of the DC:0–3R System

In addition to the limitations noted above, DSM-5 does not provide in-depth coverage of the mental health and

developmental problems of infants and young children, for whom such problems are frequently nested within the context of the family. To address this perceived deficiency, DC:0–3 and DC:0–3R, the current version, were developed by the Diagnostic Classification Task Force of the Zero to Three/National Center for Clinical Infant Programs (1994, 2005a). The revised version, developed after a decade's use of the original, primarily differs from the DC:0–3 in terms of its increased use of specific criteria to operationalize disorders, and thus increase interrater reliability (Zero to Three/National Center for Clinical Infant Programs, 2005b; Postert, Averbeck-Holocher, Beyer, Müller, & Furniss, 2009), although few data are available to speak to whether this aim was achieved. The DC:0–3R is intended to provide a comprehensive system for classifying problems during the first 3–4 years of life (Zero to Three/National Center for Clinical Infant Programs, 2005b). Unlike DSM-5, DC:0–3R is based on the explicit premise that diagnosis must be guided by the principle that infants and young children are active participants in relationships within their families. Hence descriptions of infant–caregiver interaction patterns, and of the links between these interaction patterns and adaptive and maladaptive patterns of infant and child development, constitute an essential part of the diagnostic process.

In explicitly recognizing the significance of relational functioning, DC:0–3R includes a relationship classification as a separate axis (Axis II) in its multiaxial approach (Axis I, clinical disorders; Axis III, medical and developmental disorders and conditions; Axis IV, psychosocial stressors; Axis V, emotional and social functioning). The formal classification of relationships is based on observations of parent–child interaction and information about the parent's and child's subjective experience. In classifying DC:0–3R Axis II, evidence for parental over-/underinvolvement, anxiety/tension, and anger/hostility are rated, and the clinician assesses the intensity, frequency, and duration of difficulties in the relationship, classifying these as either perturbation, disturbance, or disorder. Axis V of DC:0–3R, emotional and social functioning, includes the ways in which infants or young children organize their affective, interactive, and communicative experiences. Axis V assessment is based in large part on direct observations of parent–child interaction. The various levels include social processes such as mutual attention, mutual engagement or joint emotional involvement, reciprocal interaction, and affective/ symbolic communication. Problems may reflect constrictions in range of affect

within levels or under stress, or failure to reach expected levels of emotional development.

DC:0–3R differs significantly from other classification systems in recognizing the significance of early relational difficulties and the need to integrate diagnostic and relational approaches in classifying child psychopathology. In addition, the dimensions and specific processes that are used for classification (e.g., negative affect, unresponsivity, uninvolved, lack of mutual engagement, lack of reciprocity in interaction) include those that have been identified as important in many developmental and clinical research studies on early relationships, and the system is decidedly more sensitive to developmental and contextual parameters than DSM-5. However, although promising, DC:0–3R is still relatively untested and suffers from many of the same criticisms that have been noted for DSM-5 (Eppright, Bradley, & Sanfacon, 1998). Nevertheless, the scheme provides a rich descriptive base for exploring the ways in which psychopathology is expressed during the first few years of life, and it calls attention to the need to examine potential continuities between early problems and later individual and/or family disorders (Postert et al., 2009).

Dimensional Approaches

Dimensional approaches to classification assume that a number of relatively independent dimensions or traits of behavior exist, and that all children possess these to varying degrees. These traits or dimensions are typically derived through the use of multivariate statistical methods, such as factor analysis or cluster analysis (Achenbach, 1993). Empirically derived schemes are more objective, usually more reliable, and more informative than clinically derived classification systems. However, several problems are also associated with their use, including their complexity, as well as the dependency of the derived dimensions on sampling, method, and informant characteristics, and on the age and sex of the children (Mash & Barkley, 2007). As a result, there can be difficulties in integrating information obtained from different methods, from different informants, over time, or across situations. Dimensional approaches have also shown a lack of sensitivity to contextual influences, although there have been efforts to develop dimensional classification schemes based on item pools that include situational content (e.g., McDermott, 1993). Moreover, in many applied contexts, “categorical” decisions regarding treatment must be made,

such whether to engage in treatment. Thus most dimensional measures typically provide thresholds to indicate points at which symptoms are clinically significant to facilitate decisions regarding whether treatment should be implemented, effectively reducing such measures to categorical approaches. Nevertheless, dimensional measures of severity and/or chronicity can provide important clues regarding how intensive treatment should be (e.g., watchful monitoring vs. psychotherapy vs. combined medication and psychotherapy, in the case of depressive symptoms; Klein, 2008).

The growth in the use of multivariate classification approaches in child and family assessment has been fueled by the extensive work of Thomas Achenbach and his colleagues (see the website for the Achenbach System of Empirically Based Assessment, www.aseba.org) with the various parent, teacher, youth, observer, and interview versions of the Child Behavior Checklist and Profile (Achenbach, 1993; Achenbach & Rescorla, 2001), and by the development of similar assessment batteries (e.g., the Behavior Assessment System for Children, Second Edition; Reynolds & Kamphaus, 2004). For a comprehensive discussion of these approaches and the use of empirically derived classification schemes more generally, the reader is referred to Achenbach (1993), Hart and Lahey (1999), and Mash and Barkley (2007).

It should also be noted that there has been a trend toward greater convergence of the categorical and dimensional approaches to classification. Many of the items that were retained in DSM-IV child categories were derived from findings from multivariate studies, and the process that led to the development of DSM-IV treated most childhood disorders as dimensions, albeit the use of cutoff scores on item lists arbitrarily created categories out of these dimensions (Spitzer et al., 1990). DSM-5 has continued this trend with a greater emphasis on dimensional measures of psychopathology across development.

Performance-Based Diagnostic Information

Performance-based information and/or observational measures provide additional sources of diagnostic information that may be sensitive to differences among children exhibiting similar self- or other-reported symptoms (Frick, 2000; Kazdin & Kagan, 1994). These measures assess children’s performance on standardized tasks, usually ones that reflect basic biological, cognitive, affective, or social functioning.

For example, tasks involving behavioral observations of fear and avoidance, recall memory under stressful conditions, delayed response times to threatening stimuli, and the potentiation of the blink reflex following exposure to a threatening stimulus have all been suggested as potentially useful in diagnosing groups and/or subgroups of children with anxiety disorders (Kazdin & Kagan, 1994; Vasey & Lonigan, 2000). Similarly, tests of behavioral inhibition (e.g., the stop-signal paradigm) and tasks involving sustained attention (e.g., the continuous-performance test) have proven useful for research on children with ADHD (Rappport, Chung, Shore, Denney, & Isaacs, 2000). Measures of low resting heart rate as an early biological marker for later aggressive behavior (Raine, Venables, & Mednick, 1997); facial emotion recognition tasks and gambling tasks in identifying children with psychopathic tendencies (Blair, Colledge, & Mitchell, 2001; Blair, Colledge, Murray, & Mitchell, 2001); and a variety of cognitive tasks for children with autism spectrum disorder (Klinger & Renner, 2000) have also been found to have diagnostic value.

A study by Rubin, Coplan, Fox, and Calkins (1995) illustrates the utility of performance-based diagnostic information. These researchers differentiated groups of preschool children on the two dimensions of “emotionality” (i.e., threshold and intensity of emotional response) and “soothability” (i.e., recovery from emotional reaction based on soothing by self and others), and on their amount of social interactions with peers. Children’s dispositional characteristics and behavioral styles were used to predict outcomes. Asocial children with poor emotion regulation had more internalizing problems. In contrast, social children with poor emotion regulation were rated as having more externalizing difficulties. When behavioral and emotional dimensions were incorporated into classification, it was possible to make finer predictions—for example, that only a certain type of asocial children (i.e., reticent children with poor emotion regulation) would display later problems.

The use of performance-based measures in diagnosis is predicated on the availability of reliable and valid performance indicators for groups of children with known characteristics. Although such data are available in varying amounts for a wide range of disorders, there is a need to validate such findings for the purposes of diagnosis and against other sources of information. It is also the case that performance criteria for these measures are based on information obtained from children

who were themselves previously identified using other diagnostic procedures. This raises the question of non-independence and representativeness of samples. There is also little normative information available regarding the base rates of children in the general population who exhibit certain patterns of responding on these tasks.

ISSUES IN CLASSIFICATION

Categories, Dimensions, or Both?

Psychological studies of child psychopathology have tended to conceptualize behavior, affect, and cognition on quantitative/continuous dimensions, whereas child psychiatry has tended to conceptualize child psychopathology in categorical terms. Both approaches are relevant to classifying childhood disorders, in that some disorders may be best conceptualized as qualitatively distinct conditions and others as extreme points on one or more continuous dimensions. However, there is ongoing debate regarding which childhood disorders are best conceptualized as categories and which as dimensions (Coghill & Sonuga-Barke, 2012). It has been suggested that many childhood disorders, such as anxiety, depression, ADHD, and the disruptive behavior disorders, appear to reflect dimensions of personality rather than categorical conditions (e.g., Werry, 2001). For example, childhood ADHD symptom clusters of inattention–disorganization and hyperactivity–impulsivity have been related to personality dimensions of low conscientiousness and low agreeableness, respectively (Nigg et al., 2001). Furthermore, children naturally vary in terms of their capacity to attend and in terms of how active they are (e.g., Rothbart, 2007), and in many other dimensional behaviors that overlap with clinical conditions (e.g., temperamental fearfulness and anxiety disorders—Goldsmith & Lemery, 2000; positive and negative emotionality and depression—Klein, Durbin, & Shankman, 2009). Even autism spectrum disorder, which has frequently been viewed as categorically distinct, can be conceptualized as an extreme version of a more normative style of approaching and understanding the world and other people (Baron-Cohen, 2000; Lawson, Baron-Cohen, & Wheelwright, 2004). For disorders that reflect underlying dimensions, the concern is that the practice of categorical diagnosis creates arbitrary distinctions between normality and abnormality (e.g., children who score just below the cutoff for a diagnosis may meet full criteria

at another assessment due to random fluctuations, and often show impairment comparable to that of children who meet full criteria). Since any classification scheme represents a construction rather than a reality, it seems unlikely that most disorders will fall neatly into one designation or the other (Lilienfeld & Marino, 1995). Whether or not particular conditions are construed as qualitatively distinct categories, as continuous dimensions, or as both will probably depend on the utility, validity, and predictive value of particular groupings and subgroupings for certain purposes related to understanding and remediating child psychopathology (e.g., Kendall, Brady, & Verduin, 2001). Regardless of the particular approach one adopts for the classification of childhood psychopathology, diagnostic decisions need to be based on a comprehensive assessment of the individual child—one that incorporates sensitivity to, and understanding of, the complexity of multiple antecedents, developmental considerations, comorbidity, continuity–discontinuity, and the constantly changing nature of the child (Frick, Barry, & Kamphaus, 2010; Mash & Hunsley, 2007). Given the general trend for cognitive-behavioral approaches to be the most effective available for childhood disorders (Mash & Barkley, 2006), functional analysis of child behavior should also play a key role.

Comorbidity

An issue that has important ramifications for theory and research in defining and classifying child psychopathology is comorbidity (Achenbach, 1995; Angold, Costello, & Erkanli, 1999; Carey & DiLalla, 1994; Caron & Rutter, 1991; Sonuga-Barke, 1998). “Comorbidity” generally refers to the manifestation of two or more disorders that co-occur more often than would be expected by chance alone. For example, although the base rates for ADHD and conduct disorder in the general population are less than 10% for each disorder, epidemiological studies have found that among children diagnosed with ADHD, approximately 50% are also diagnosed with conduct disorder (Kazdin & Johnson, 1994; Loeber & Keenan, 1994). Comorbidity has been reported to be high in community samples and even higher in clinic samples (Bird et al., 1988; Caron & Rutter, 1991; Costello, Mustillo, et al., 2003). Some of the more commonly co-occurring child and adolescent disorders include conduct disorder and ADHD, autism spectrum disorder and intellectual disability, and child/adolescent depression and anxiety disorders.

There is continuing debate regarding the definition and nature of comorbidity (Angold, Costello, & Erkanli, 1999; Blashfield, McElroy, Pfohl, & Blum, 1994; Cunningham & Ollendick, 2010; Lilienfeld, Waldman, & Israel, 1994; Meehl, 2001; Robins, 1994; Rutter, 1994b; Sameroff, 2000). Some researchers contend that the term is wholly inadequate because it does not distinguish accurately between manifest conditions seen in organic medicine (e.g., diseases) and latent conditions described in mental health (e.g., syndromes and disorders (Lilienfeld et al., 1994). Others argue that the dispute over whether one should use the term “comorbidity,” “co-occurrence,” or “covariation” is largely a semantic one (Rutter, 1994b; Spitzer, 1994; Widiger & Ford-Black, 1994).

Several possible reasons why comorbidity may be exaggerated or artificially produced have been identified in the literature (Angold, Costello, & Erkanli, 1999; Lilienfeld et al., 1994; Rutter, 1994b; Verhulst & van der Ende, 1993; Wolff & Ollendick, 2006). There may be a sampling bias that occurs when estimates of disorder prevalence are derived from treatment-seeking or clinic samples. In such cases, the clinic samples will contain a disproportionately large number of subjects who display comorbid conditions, as the probability of being referred to mental health services is higher for a child with a comorbid condition than for a child with only one disorder. Related to this sampling bias are various other referral factors that may inflate the degree of co-occurring disorders among clinic samples. Clinics and clinicians specializing in treatment of more complicated cases, for example, may be more likely to receive referrals in which comorbid conditions are present. In addition, children with internalizing difficulties such as depression are more likely to be referred by their parents or the school system if they also show externalizing symptoms, largely because externalizing problems are viewed as more disruptive by referral sources.

Comorbidity may also reflect various sources of nosological confusion arising from the manner in which different childhood disorders have been conceptualized and organized. For instance, Widiger and Ford-Black (1994) claim that excessive rates of co-occurrence seemed to appear concomitantly with the changes that occurred in DSM-III (e.g., increased coverage, divisions of diagnostic categories, the provision of separate and multiple axes). Another example is that DSM-5 makes it possible to have multiple diagnoses in the absence of multiple syndromes (Cantwell, 1996; Robins, 1994). One source of confusion stems from the overlap-

ping criterion sets within contemporary classification schemes (Drabick & Kendall, 2010; Rutter, 2010). In DSM-5, diagnoses are based on a set of polythetic criteria that include specific symptom constellations. In many cases, the presence of concomitant symptoms of a different kind are ignored, resulting in an increased likelihood that the accompanying symptoms will be represented in a different diagnostic category (Caron & Rutter, 1991). Sonuga-Barke (1998) argues, however, that although earlier diagnostic systems steered clear of comorbidity by using a hierarchical set of exclusionary criteria, “these approaches were abandoned because they clearly led to a misrepresentation of the structure of disorder” (p. 119). For example, they led to low base rates of disorders and poor interrater agreement.

Apart from the various artifactual contributors to comorbidity, there are also indicators in support of “true” comorbidity (Rutter, 1994b). It is possible that general propensities toward and/or struggles with adaptation are at the core of every disorder, but that the expression of the phenotype is contingent upon a myriad of environmental conditions and person–environment interactions (Caron & Rutter, 1991). Consistent with this notion, Lilienfeld and colleagues (1994) maintain that comorbidity in childhood disorders may be partly a function of developmental level—that is, of underlying processes that have not yet achieved full differentiation. Differing rates of comorbidity with age may also reflect the fact that the appearance of one disorder or problem may precede the appearance of the other, as is the case for anxiety preceding depression (Brady & Kendall, 1992) or for impulsivity preceding attentional problems (Hart et al., 1995). Still another possibility is that comorbidity reflects “a more amorphous early expression of psychopathology in young children that does not crystallize into more definitive psychopathology until later in life” (Cantwell, 1996, p. 4). Comorbidity can also arise as a result of a causal association in which the severity of one disorder may lead to or greatly increase the later risk for another disorder (e.g., ADHD and oppositional defiant disorder) or a shared underlying cause, such as common genetic influences (e.g., conduct disorder and depression) or neurobiological processes (e.g., anxiety and depression). In the case of shared etiology, poorly drawn boundaries between disorders may contribute to the appearance of multiple co-occurring disorders, when the reality is that two disorders are different manifestations of the same underlying neural circuit disruptions (Morris & Cuthbert, 2012).

In summary, it would appear that some cases of comorbidity are the results either of ambiguity in the definition of dysfunctionality that is used, or of artifactual/methodological issues. However, as Kazdin and Kagan (1994) note, “the broader point is still relevant and not controverted with specific diagnostic conundrums—namely, multiple symptoms often go together in packages” (p. 40). This is not to suggest that *all* disorders cluster together into packages; rather, the fact that many frequently do has important implications for how child psychopathology is conceptualized and treated. The complexity of comorbidity behooves researchers to move beyond singular models and to examine multiple expressions, etiologies, and pathways of childhood dysfunction (Beauchaine, Hinshaw, & Pang, 2010; Burt, Krueger, McGue, & Iacono, 2001; Kazdin & Johnson, 1994).

THE DEVELOPMENTAL PSYCHOPATHOLOGY PERSPECTIVE

The developmental psychopathology perspective aims to provide a useful working framework for conceptualizing and understanding child psychopathology. This approach integrates multiple theories (e.g., psychodynamic, behavioral, cognitive, biological, family systems, and sociological), each of which focuses on different sets of variables, methods, and explanations (Achenbach, 2000), to provide a template and principles for understanding the processes underlying how and why psychopathology in children emerges, how it changes over time, and how it is influenced by a child’s developmental capacities and by the contexts in which development occurs (Cicchetti & Toth, 2009). Long described as a macroparadigm that subsumes several theoretical approaches (Cicchetti, 1984; Cicchetti & Cohen, 1995; Lewis, 2000; Luthar et al., 1997; Rutter & Sroufe, 2000; Sameroff, 2000), “developmental psychopathology” has been defined as “*the study of the origins and course of individual patterns of behavioral maladaptation, whatever the age of onset, whatever the causes, whatever the transformations in behavioral manifestation, and however complex the course of the developmental pattern may be*” (Sroufe & Rutter, 1984, p. 18; original emphasis). Put simply, developmental psychopathology provides a general framework for understanding both normal development and its maladaptive deviations. Its main focus is an elucidation of developmental processes and their functioning through

an examination of extremes in developmental outcome and of variations between normative outcomes and negative and positive extremes. Developmental psychopathology does not focus exclusively on the study of childhood disorders, but serves to inform the understanding and treatment of disorders through the study of a full range of developmental processes and outcomes.

A developmental psychopathology perspective is consistent with both transactional and ecological views, and assumes that within ongoing change and transformation there exist coherence and predictability for adaptive and maladaptive development (Campbell, 1989; Cicchetti & Toth, 1997). This perspective also emphasizes the importance of endogenous (e.g., genetic, neurobiological) and exogenous (e.g., family, social, and cultural factors) and the interaction of the two in predicting and understanding developmental changes (Achenbach, 2000; Lewis, 2000). In this way, developmental psychopathology attempts to address the complex influences surrounding the development of the child across the lifespan. In attempting to do so, it draws on knowledge from multiple fields of inquiry (including psychology, psychiatry, sociology, education, criminology, epidemiology, and neuroscience) and attempts to integrate this knowledge within a developmental framework (Rutter & Sroufe, 2000).

The focus of developmental psychopathology is on normal developmental patterns, continuities and discontinuities in functioning, and transformational interactions over different developmental periods that produce adaptive or maladaptive outcomes. The processes underlying both healthy and pathological development are seen as stemming from idiosyncratic transactions between a child and his or her unique context (Achenbach, 2000; Sroufe & Rutter, 1984). Thus a central tenet of this approach is that to understand maladaptive behavior adequately, one needs to view it in relation to what may be considered normative for a given period of development (Edelbrock, 1984). Significant challenges for research, then, are to differentiate those developmental deviations that are within normative ranges from those that are not, and to ascertain which among the plethora of interacting variables account for developmental deviation. A developmental psychopathology perspective is also guided by several other principles, including the notion that the individual child plays an active role in his or her own developmental organization, that developmental outcomes are best predicted through consideration of prior experience

and recent adaptations examined in concert, and that transitional turning points or sensitive periods in development represent times when developmental processes are most susceptible to positive and/or negative self-organizational efforts (Cicchetti & Tucker, 1994).

Until recently, the developmental psychopathology perspective has been more of a conceptual enterprise than a well-validated approach (Lewis, 2000). However, in a very short period of time, it has proven to be an enormously useful framework for understanding and guiding research in child psychopathology, and it represents an important shift in thinking away from single causal hypotheses toward a view based on complex and multiple pathways of influence: “After each effort to support an explanatory model by collecting a set of data, the results have required modifications in the model, forcing the field to evolve from a concern with causes and effects to an increasing appreciation of the probabilistic interchanges between dynamic individuals and dynamic contexts that comprise human behavior” (Sameroff, 2000, p. 297).

Within the integrative framework of developmental psychopathology, efforts are made to understand the different pathways through which similar forms of psychopathology emerge, and the reasons why seemingly similar developmental pathways may lead to different outcomes. Numerous disorder- and problem-focused theories have been proposed. These models are empirically based and are sensitive to the specific characteristics and processes that research has identified as important for understanding a particular disorder or problem. A few examples of representative models include Barkley’s (2004, 2012a) theory of “inhibitory and executive dysfunction,” which initially proposed behavioral inhibition as the primary and central deficit underlying the attentional, cognitive, affective, and social difficulties of children with ADHD. The subsequent iteration of this theory has now expanded this idea to include other executive functions, such as working memory, besides the inhibitory deficits as being central to this disorder (Barkley, 2004, 2012a). These initial deficits produce numerous effects at increasing spatial and temporal distances into the social ecology of the individual that comprise the extended phenotype of the disorder (Barkley, 2012b). Another example is the Cummings and Davies (1996, 2010; Davies & Cummings, 1994) “emotional security hypothesis,” which proposes that emotional insecurity resulting from a number of sources (e.g., maternal depression, marital conflict) may lead to child difficulties in self-regulation, efforts to overregu-

late others, and maladaptive relational representations. Crick and Dodge's (1994) model of social information-processing deficits in aggressive children provides yet another example, which views aggression as a outcome of a child's use of biased or distorted interpretational processes in social situations.

Other theories that have been proposed to account for these and other problems and disorders are presented in the subsequent chapters of this volume. The growth in the number of such theories reflects an increasing trend toward models that focus on the processes underlying specific forms of child psychopathology, rather than on child psychopathology in general. However, most contemporary causal models that emphasize specific disorders have not conducted the necessary empirical tests to determine the specificity of putative etiological factors, despite the fact that there are likely to be common factors (e.g., personality, genetic risks, family discord/stress) that increase risk for many different types of disorder (Epkins & Heckler, 2011). Identifying how etiological influences are similarly versus differentially related to disorders is an important task for future research.

GENERAL THEORIES OF CHILD PSYCHOPATHOLOGY

Several major theories have been proposed to account for the emergence of psychopathology in children (see Table 1.4). These include psychodynamic (Dare, 1985; Fonagy & Target, 2000; Shapiro & Esman, 1992), attachment (Atkinson & Goldberg, 2004; Bowlby, 1973, 1988), behavioral/reinforcement (Bijou & Baer, 1961; Skinner, 1953), social learning (Bandura, 1977, 1986), interpersonal (Gotlib & Hammen, 1992; Joiner & Coyne, 1999; Rudolph, Flynn, & Abaied, 2008); cognitive (Beck, 1964; Beck, Rush, Shaw, & Emery, 1979; Evraire, Dozois, & Hayden, in press; Ingram, Miranda, & Segal, 1998), constitutional/neurobiological (e.g., Cappadocia, Desrocher, Pepler, & Schroeder, 2009; Heim & Nemeroff, 2001; Matthys, Vanderschuren, & Schutter, 2013; Tripp & Wickens, 2009), affective (Davidson, 2000; Rubin, Cheah, & Fox, 2001), and family systems (Cowan & Cowan, 2002; Davies & Cicchetti, 2004; Grych & Fincham, 2001) models. A detailed discussion of the basic tenets of each of these general theories is beyond the scope of this chapter. For comprehensive discussions of these theories, the reader is directed to original sources and to specific references

TABLE 1.4. General Models Used to Conceptualize Child Psychopathology

Psychodynamic models

Inborn drives, intrapsychic mechanisms, conflicts, defenses, psychosexual stages, fixation, and regression.

Attachment models

Early attachment relationships; internal working models of self, others, and relationships in general.

Behavioral/reinforcement models

Excessive, inadequate, or maladaptive reinforcement and/or learning histories.

Social learning models

Vicarious and observational experience, reciprocal parent-child interactions.

Interpersonal models

Interactional styles, social skills deficits, social difficulties, stressful interpersonal environments.

Cognitive models

Distorted or deficient cognitive structures and processes.

Constitutional/neurobiological models

Temperament, genetic influences, structural and functional neurobiological mechanisms.

Affective models

Dysfunctional emotion-regulating mechanisms.

Family systems models

Intra- and intergenerational family systems, and the structural and/or functional elements within families.

Note. Models are highlighted in terms of their relative emphasis.

cited throughout this volume. What follows is a discussion of several general points related to some of these theories.

Each general theoretical approach reflects a diversity of viewpoints. For example, psychodynamic theory encompasses traditional Freudian and Kleinian psychoanalytic constructs and their many derivatives as reflected in ego-analytic and object relations theory (Fonagy & Target, 2000; Lesser, 1972). Behavioral/reinforcement perspectives include traditional operant/classical conditioning constructs, mediational mod-

els, and contemporary theories of learning (Klein & Mower, 1989; Krasner, 1991; Viken & McFall, 1994). Cognitive theories include cognitive-structural models, models of cognitive distortion, and models of faulty information processing (Clark, Beck, & Alford, 1999; Ingram et al., 1998; Kendall & Dobson, 1993). Family systems theories include systemic, structural, and social learning models (Jacob, 1987). Therefore, when one is discussing any theory, it is critical to distinguish among the different perspectives encompassed by the approach.

Many theories of child psychopathology are derivatives of earlier approaches. For example, psychodynamic theories dominated thinking about child psychopathology for the first half of the 20th century. These theories contributed to our understanding of child psychopathology through their emphasis on the importance of relationships, early life experiences, mental mechanisms, and unconscious processes, and they spawned a number of other models—for example, attachment theory (Rutter, 1995). The emergence of attachment theory reflected a shifting of attention from the more traditional psychoanalytic role of intrapersonal defenses to that of interpersonal relationships (Bretherton, 1995). Similarly, the emergence of social learning theory reflected disenchantment with nonmediational models of learning and a growing interest in the role of symbolic processes.

A number of general points can be made regarding theories of child psychopathology:

1. Each theory offers an explanation regarding the etiology of child psychopathology. The strength of each theory rests on its specificity in predicting various forms of psychopathology and its degree of empirical support.

2. The varying degrees of support for each conceptualization suggest that no single model can fully explain the complexities involved in understanding child psychopathology. In light of this, increased understanding may accrue if greater integrative and collaborative efforts are undertaken.

3. Many explanations of childhood disorders implicitly or explicitly assume a simple association between a limited number of antecedents and a given disorder. However, as we have discussed, the concept of multiple pathways that lead to different outcomes depending on the circumstances represents a more viable framework in light of current research findings.

4. Although the testing of specific models is consistent with the spirit of parsimony, far greater attention needs to be given to the unique contexts and conditions under which a particular model does or does not apply.

5. Research on dysfunction frequently examines static conditions and influences such as the expression of a disorder at a given age or the influence of a specific stressor. However, evidence indicates that the expression and etiology of psychopathology in children are continuously changing over time, and theories need to account for these types of changes.

Current models are becoming increasingly sensitive to the many different components of childhood dysfunction. Indeed, constitutional, behavioral, cognitive, emotional, and social factors cross a number of theoretical domains; this is reflected in the emergence of hybrid models (e.g., cognitive-behavioral, social information processing, cognitive-neuropsychological), as well as the inclusion of family and ecological constructs across many different theories. Behavioral models, which have frequently been characterized as having a narrow emphasis on conditioning principles, are also becoming increasingly sensitive to systems influences (Viken & McFall, 1994).

Four interrelated theoretical approaches have received increased attention in current research on child psychopathology: (1) attachment theory, (2) cognitive theories, (3) emotion theories, and (4) constitutional/neurobiological theories. Each of these approaches is highlighted in the sections that follow.

Attachment Theory

Bowlby's (1973, 1988) theory of attachment is based on both an ethological and a psychoanalytic perspective (Cassidy & Shaver, 2008; Cicchetti, Toth, & Lynch, 1995). Nevertheless, Bowlby rejected the psychoanalytic ideas that individuals pass through a series of stages where fixation at or regression to an earlier state can occur, and that emotional bonds are derived from drives based on food or sex. Drawing on ethology and control theory, Bowlby and his successors replaced Freudian concepts of motivation based on psychic energy with cybernetically controlled motivational-behavioral systems organized as plan hierarchies (Bowlby, 1973; Bretherton, 1995). Within attachment theory, instinctive behaviors are not rigidly predetermined, but rather become organized into flex-

ible goal-oriented systems through learning and goal-corrected feedback. Motivational-behavioral systems (e.g., attachment, exploration) regulate time-limited consummatory behaviors and time-extended instinctive behaviors that maintain an organism in relation to its environment. Attachment belongs to a group of stress-reducing behavioral systems that operate in conjunction with physiological arousal-regulating systems. The child is motivated to maintain a balance between familiarity-preserving, stress-reducing behaviors, and exploratory and information-seeking behaviors. Self-reliance develops optimally when an attachment figure provides a secure base for exploration (Bretherton, 1995).

It is via the attachment relationship that the infant develops an “internal working model” of the self and others. Bowlby (1988) argued that the development of psychopathology is directly related to the inability of the caregiver to respond appropriately to the child’s needs. This assertion is, however, a point of contention among researchers. Sroufe (1985), for example, has questioned the direct role of parental influence, arguing that infant temperament and the reciprocal interaction of a “difficult temperament” with parental response may better account for the variance in the attachment relationship and its ensuing insecure attachment difficulties. On the basis of a review of several studies examining infant temperament and attachment, Sroufe suggests that although some studies have supported the notion that differences between secure and insecure attachments may be due to temperament, the bulk of evidence suggests that infants change their attachment patterns with different caregivers.

In postulating an association between early attachment and later psychopathology, one must exercise caution, in that there does not appear to be one specific subtype of attachment that leads to one particular childhood disorder. Rather, the trajectory for developmental pathways and manifestations of psychopathology emerges as the result of environmental experience, biological predispositions, and learning. When one is identifying possible developmental paths as factors related to subsequent psychopathology, the concept of the child’s internal working model is useful; however, it is important to bear in mind that the internal working model represents a set of active constructions that are subject to change, and that the association with later psychopathology is probabilistic rather than absolute.

Rutter (1995) has highlighted a number of key issues surrounding attachment, including (1) the need

to identify mechanisms involved in proximity-seeking behavior; (2) broadening the basis for measuring attachment to include dimensions as well as categories; (3) studying relationship qualities that may not be captured by “insecurity”; (4) understanding the relationship between temperament and attachment; (5) dealing with how discrepant relationships are translated into individual characteristics; (6) operationalizing internal working models; (7) defining attachment quality across the lifespan, and determining whether or not meanings are equivalent at different ages; (8) determining how one relationship affects others; and (9) identifying the boundaries of attachment vis-à-vis other aspects of relationships. Several issues—the association between attachment and later functioning; the linkage between parenting and attachment quality; the adaptive value of secure attachment (e.g., insecure attachment does not equal psychopathology); disorders of attachment associated with abuse and neglect; and the diffuse attachments associated with institutionalization—are all in need of further investigation. Bowlby’s attachment theory has played an important role in focusing attention on the quality of parent-child relationships, the interaction between security in relationships and the growth of independence, the importance of placing emergent human relationships within a biological/evolutionary context (e.g., Kraemer, 1992), the concept of internal working models, and insecure early attachments (e.g., Barnett & Vondra, 1999) as the basis for the development of psychopathology (Rutter, 1995).

Cognitive Theories

Considerable research has focused on the role of cognition (i.e., mental processes that include attention, memory, learning, problem-solving and decision-making) in both adult and child psychopathology (Clark et al., 1999; Ingram et al., 1998; Ingram & Price, 2001). Several theoretical perspectives have been concerned with childhood cognitions. These have included cognitive-structural models (Ingram et al., 1998; Selman, Beardslee, Schultz, Krupa, & Poderefsky, 1986), information-processing approaches (Crick & Dodge, 1994; Ingram & Ritter, 2000; Taylor & Ingram, 1999), and cognitive-behavioral approaches (Braswell & Kendall, 2001; Dobson & Dozois, 2001; Meichenbaum, 1977). Representative examples of the information-processing and cognitive-behavioral approaches are described below. Recently cognitive theories have focused on the importance of positive cognitions, the role of cognitive

specificity, the role of context on cognitions, the impact of comorbidity, the use of information-processing risk paradigms, a movement away from simple cognitive diathesis–stress models to looking at information-processing mediators, and the need for theoretical integration.

Information Processing

Biased information processing has been implicated in a number of childhood disorders. For example, socially aggressive children have been found to display negative attributional biases (Dodge & Pettit, 2003; Schwartz & Proctor, 2000); children with anxiety disorders show attentional biases to threatening stimuli (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van IJzendoorn, 2007; Waters, Henry, Mogg, Bradley, & Pine, 2010); and depressed children exhibit greater encoding biases for negative material, less endorsement and recall of positive information, and other forms of negative cognition (Abela & Hankin, 2008; Lakdawalla, Hankin, & Mermelstein, 2007). Research on information processing and child psychopathology has emanated from three streams: one focusing on deficits in basic information processing related to attention, memory, and other cognitive functions (e.g., Carter & Swanson, 1995); another related to social information processing (Crick & Dodge, 1994); and a third focusing on maladaptive cognition (e.g., Ingram et al., 1998; Ingram & Ritter, 2000; Taylor & Ingram, 1999).

Dodge’s model as applied to socially aggressive boys illustrates the social information-processing approach (Dodge & Pettit, 2003; Dodge & Somberg, 1987). In the initial model, a series of thought processes and behaviors (i.e., encoding, interpretation, response search, response decision, and enactment) was postulated to occur during the course of appropriate social interactions and to be absent or distorted during inappropriate social interactions. The model has evolved, positing the same basic information-processing steps, but at each stage there is ongoing reciprocal interaction between the information-processing skills required during social transactions in context and the individual’s “database” (a collection of social schemas, memories, social knowledge, and cultural values or rules) (Crick & Dodge, 1994; Dodge & Pettit, 2003). Instead of a linear processing model, there are postulated to be cyclical feedback loops connecting all stages of processing. Increased recognition of the influence of peer appraisal and response, emotional processes, and the

development and acquisition of cognitive skills as important contributors to social adjustment are meaningful additions to the reformulated model. In addition to the enhanced sensitivity to developmental trajectories, the reformulated model emphasizes the role of early dispositions (e.g., temperament) and other factors (e.g., age, gender, social context) that serve to moderate the relationship between information processing and social adjustment. A number of studies have provided empirical support for the expanded model (Contreras, Kerns, Weimer, Getzler, & Tomich, 2000; Gomez & Gomez, 2000; Gomez, Gomez, DeMello, & Tallent, 2001).

Cognitive-Behavioral Theories

Cognitive-behavioral theories represent “a purposeful attempt to preserve the positive features of the behavioral approaches, while also working to incorporate into a model the cognitive activity and information-processing factors of the individual” (Kendall & MacDonald, 1993, p. 387; see also Braswell & Kendall, 2001), and cognitive vulnerabilities to depression and anxiety in particular are firmly established as central models of risk and treatment. Research on such cognitive models initially focused on adults, using a wide array of operationalizations of cognitive risk; this research has generated a vast corpus of results generally supporting the central tenets of cognitive theories, in that cognitive vulnerability has been found to be a diathesis that interacts with negative life events to predict increases in symptoms (Ingram et al., 1998).

Four elements of cognition are distinguished for the purpose of understanding the pathogenesis of psychiatric disturbances: cognitive structures, content, operations, and products (Beck et al., 1979; Dozois & Dobson, 2001; Ingram et al., 1998; Kendall & Dobson, 1993). “Cognitive structures” represent the way in which information is organized and stored in memory, and serve the function of filtering or screening ongoing experiences. “Cognitive content” (or propositions) refers to the information that is stored in memory (i.e., the substance of the cognitive structures). Together, cognitive structures and content make up what is termed a “schema.” A schema stems from a child’s processing of life experiences and acts as a guideline or core philosophy influencing expectations and filtering information in a fashion consistent with the child’s core philosophy. As such, cognitive schemas have also been referred to as “filters” or “templates” (see Kendall & MacDonald, 1993). A schema is postulated to affect the relative ob-

served consistency in the child's cognition, behavior, and affect (Stark, Rouse, & Livingston, 1991). According to Beck's model, maladaptive schemas develop in early childhood and remain dormant until some untoward event triggers the latent schemas, and the individual begins to encode, process, and interpret information in a schema-congruent way. Individuals with a depression schema, for instance, process and interpret information about themselves, the world, and the future in a negatively biased fashion, whereas persons with an anxiety schema interpret environmental stimuli with a cognitive focus on future threat. In addition, what appears to be specific to depression is a lack of positive cognition (Gencoez, Voelz, Gencoez, Pettit, & Joiner, 2001). "Cognitive processes" or "cognitive operations" pertain to the manner by which the cognitive system functions. Thus cognitive processes, which are guided by schemas, suggest the mode by which an individual perceives and interprets both internal and external stimuli. Finally, "cognitive products" are the ensuing thoughts that stem from the simultaneous and reciprocal interactions among the various components of the cognitive system.

Work testing cognitive models of depression has shifted in recent years toward the exploration of the utility of these models in adolescents and children (Abela & Hankin, 2008). Reviews of this literature support the claim that cognitive vulnerability in youth is an important prospective predictor of depressive symptoms (e.g., Hankin et al., 2009), usually when examined in conjunction with stressful life events. More specifically, most studies have focused on testing whether the interaction between negative cognition and stress predicts elevations in children and adolescents' depressive symptoms. These studies have shown that stressful life events show stronger associations with depression when youth possess negative cognitive styles (such as maladaptive attributional styles), information-processing biases favoring enhanced processing of negative stimuli, and other aspects of depressive cognition (Abela & Hankin, 2008; Lakdawalla et al., 2007).

A potentially useful distinction can be made between "cognitive deficits" and "cognitive distortions." Kendall (1993) argues that this distinction is useful in describing, classifying, and understanding a variety of juvenile disorders. Children with "deficits" display an absence of thinking where it would be beneficial. Aggressive youth, for example, frequently lack the ability to encode interpersonal information (Coy, Speltz, DeKlyen, & Jones, 2001; Pakaslahti, 2000; Schwartz

& Proctor, 2000) or to solve social problems adequately (Crick & Dodge, 1994; Lochman & Dodge, 1994), and impulsive children often fail to think before they respond (Moore & Hughes, 1988). Conversely, children who display "distortions" typically do not lack the ability to organize or process information; rather, their thinking is described as biased, dysfunctional, or misguided (Kendall, 1993; Kendall & MacDonald, 1993). A depressed individual's negative view of him- or herself, the world, and the future is an example of distorted thinking. Kendall (1985, 1993) notes that the distinction between deficient and distorted thinking is relevant to the distinction that has been made between externalizing and internalizing disorders (cf. Achenbach, 2000). Generally, internalizing disorders are related to distortions in thinking, whereas externalizing disorders are more commonly associated with cognitive deficits. However, empirical evidence suggests that aggressive behaviors usually include both distortions and deficits (e.g., Lochman, White, & Wayland, 1991).

Cognitive models have both strengths and weaknesses. The theoretical model asserts that stable, latent schemas develop in childhood and are dormant until a triggering negative event; the model thus generates strong hypotheses regarding the assessment of cognitive risk and the work of therapy (Braswell & Kendall, 2001; Kendall, 1993). Importantly, these theories thus assert the stability of cognitive risk markers that emerge early in life. There is ongoing debate regarding when meaningful, stable aspects of cognitive vulnerability emerge (e.g., Abela & Hankin, 2011; Cole et al., 2008; Garber, 2010; Gibb & Coles, 2005; Hammen & Rudolph, 2003). Furthermore, work that speaks to the stability of childhood cognitive vulnerability is accruing (e.g., Cole et al., 2009; Hankin, 2008; Hayden, Olino, Mackrell, et al., 2013), and evidence is consistent with both stability and change (Hankin et al., 2009). This literature indicates that while some rank-order stability emerges in later childhood, significant change also occurs for some children. However, this work has focused on self-reported cognitive risk in later childhood and early adolescence, and across relatively brief follow-ups—factors that may serve to indicate increased stability compared to research on younger samples using laboratory-based measures and longer follow-up intervals. Further work on emerging cognitive risk in younger children, indexed via approaches that map more fully onto the array of methodologies indexing cognitive risk in depression, is clearly needed. If a period in development can be identified in which

children's cognitive vulnerability has both meaningful implications for disorder risk *and* evidence of some degree of plasticity, such a period could represent an important window for preventative efforts.

Another limitation of these models is that the developmental origins of emerging cognitive risk have yet to be fully explored, particularly in the context of broader models childhood cognitive risk. More specifically, relatively little research has attempted to identify early precursors of negative cognition implicated in disorder risk. In recent years, work tying negative cognition to early adversity (Gibb, 2002), parental psychopathology, emotional traits (Davidson et al., 2002; Hamburg, 1998; Hayden et al., 2006), and genetic risk (Gibb, Beevers, & McGeary, 2013; Hayden, Olino, Bufferd, et al., 2013) has begun to emerge, although more comprehensive models that test the possibility of dynamic interplay among multiple factors are still lacking.

Emotion Theories

Emotion and its regulatory functions are constructs that cross several conceptual models—including psychodynamic theory, with its concept of defense mechanisms; cognitive-behavioral theory, which stresses the role of thought patterns and behavior as determinants of emotion; attachment theory, with its premise that an internal working model is formed on the basis of early relations and continues to regulate emotion in subsequent relationships (Cassidy, 1994); and biological theories, which emphasize the structural and neurochemical correlates of emotion regulation (Pennington & Ozonoff, 1991; Posner & Rothbart, 2000). Emotion and its regulation played a central role in the conceptual paradigms of early models of child psychopathology. For example, psychoanalytic theory emphasized the regulation of emotions through the use of defense mechanisms, with an absence of such regulation leading to anxiety and psychopathology (see Cole, Michel, & Teti, 1994). By giving individuals the opportunity to avoid, minimize, or convert emotions, defense mechanisms were hypothesized to serve the function of regulating emotional experiences too difficult to manage at the conscious level.

Although the advent and growth of cognitive and behavioral models shifted attention away from an interest in affective processes, the study of emotional processes in child psychopathology has experienced a resurgence of interest (Arsenio & Lemerise, 2001; Belsky, Friedman, & Hsieh, 2001; Insel, 2003; Rubin et al., 2001), in recognition that children's emotional experience,

expression, and regulation are likely to affect the quality of their thinking, social interactions, and relationships (e.g., Flavell, Flavell, & Green, 2001; Rubin et al., 2001; Schultz, Izard, Ackerman, & Youngstrom, 2001). From a functionalist perspective, emotions are viewed as playing a causal role in organizing and directing the way in which children react to environmental events. This perspective is illustrated by findings showing that induced negative child emotions increase children's distress, negative expectations, and appraisals of adult conflict, whereas induced positive emotions have the opposite effect (Davies & Cummings, 1995). Several discussions have focused on the development of emotion regulation and its ability to influence both adaptive and maladaptive functioning (Fredrickson, 2001; Kagan, 1994b; Mayer & Salovey, 1995; Thompson, 2011). In general, there is growing support for the view that emotionality and regulation are related to children's concurrent and long-term social competence and adjustment (Eisenberg, Fabes, Guthrie, & Reiser, 2000).

Emotion systems have as their primary functions the motivation/organization of behavior and communication with self and with others. Emotions represent patterns that include at least several of the following components: (1) activating neural, sensory–motor, cognitive, and/or affective stimulus events; (2) dedicated neural processes; (3) changes in physiological responses; (4) changes in motoric/expressive behavior; (5) related cognitive appraisals; and (6) concomitant alterations in subjective experiences or feeling states (Cicchetti, Ackerman, & Izard, 1995; Izard, 1993; Kagan, 1994b).

Different theories have viewed child psychopathology as emanating from the following: (1) unrestrained emotions (i.e., emotions that are unconnected to cognitive or affective–cognitive control processes); (2) deficits or distortions in cognitions and behaviors that interfere with emotion modulation (i.e., emotions connected to cognitive processes and behavior that are situationally inappropriate); (3) emotional interference with planful cognitive processes (i.e., emotional flooding); (4) dysfunctional patterns of emotion processing and communication, involving problems with recognition, interpretation, and expression; and (5) difficulties in coordinating emotional and cognitive processes in the regulation of emotion (Cicchetti, Ackerman, & Izard, 1995).

Emotion dysfunction may emanate from several sources, including variations in biological vulnerabil-

ity and stress. In studying child psychopathology, it is important not to focus on negative emotions without also recognizing several other factors: the beneficial and buffering effects of positive emotions (Fredrickson, 2001; Masten, 2001; Tugade & Fredrickson, 2004; Wichers et al., 2007); the adaptive value and facilitating effects of negative emotions of moderate or at times even extreme intensity; and the ongoing importance of emotion content and meaning for a child's behavior. Also, since negative emotions are neither topographically nor functionally unidimensional, it is important to identify the *discrete* emotions and emotional patterns underlying different forms of child psychopathology (Cicchetti, Ackerman, & Izard, 1995). For example, the negative affect that is associated with depression may involve sadness, anger, or guilt, in the same way that negative behaviors in depressed children may be both aggressive/confrontational and depressive/distressed (Hops, 1995).

It may be useful to distinguish between the two dimensions of "emotion reactivity" and "emotion regulation." "Reactivity" refers to individual differences in the threshold and intensity of emotional experience, whereas "regulation" describes processes that operate to control or modulate reactivity (e.g., attention, inhibition, approach-avoidance, coping styles) (Rubin et al., 1995). According to Rubin and colleagues (1995), this distinction is important because it highlights the need to focus on the dynamic interaction between general temperament and specific regulatory mechanisms, and in turn the need to recognize that emotional arousal (reactivity) can serve to inhibit, facilitate, or disrupt behavior. The distinction can also be made between problems in regulation and problems in dysregulation, with regulation problems involving weak or absent control structures or structures overwhelmed by disabling input, and dysregulation involving existing control structures that operate in a maladaptive manner and direct emotion toward inappropriate goals (Cicchetti, Ackerman, & Izard, 1995). Functions of emotion involve the emotion knowledge of self and others in identifying feelings and behavior, including monitoring of self and environment. Absent or weak monitoring may result in dissociated emotional and cognitive processes and emotional leakage, whereas excessive monitoring may lead to a narrow sampling of emotional signals and excessive use of specific emotions in communication (Cicchetti, Ackerman, & Izard, 1995).

Of interest to the present chapter is the manner in which emotion regulation has been defined and con-

ceptualized with respect to psychopathology (Keenan, 2000). The processes of emotion regulation include the attenuation or deactivation of an ongoing emotion, the amplification of an ongoing emotion, the activation of a desired emotion, and the masking of emotional states (Cicchetti, Ackerman, & Izard, 1995). Thompson (1994) defines emotion regulation as consisting of "the extrinsic and intrinsic processes responsible for monitoring, evaluating, and modifying emotional reactions, especially their intensive and temporal features, to accomplish one's goals" (p. 27). This definition highlights several important characteristics of emotion regulation. First, it involves enhancing, maintaining, or inhibiting emotional arousal for the purpose of meeting one's goals. Second, there are both internal and external factors that influence the development and use of emotion-regulating strategies. Finally, there is a temporal dimension: Sometimes there are sudden and transitory changes in emotional arousal that must be dealt with (e.g., acute or state anxiety), whereas at other times there are longer-lasting ramifications of emotional arousal created by years of experience (e.g., chronic or trait anxiety; Kagan, 1994b; Terr, 1991). However, an important conceptual issue that is central to the question of what is currently known about the role of emotion regulation in child development and psychopathology concerns the extent to which research has adequately differentiated between emotion experience (i.e., the strength of an initial emotional response) and regulatory processes (i.e., processes that modulate this initial response). Although emotion and emotion regulation are theoretically distinct, Campos, Frankel, and Camras (2004) have cogently argued that the processes that underpin the two overlap almost entirely, and that adequately differentiating between them for the purposes of assessment is a potentially intractable problem. Indeed, a review of the literature indicates that many studies methodologically conflate high emotionality (e.g., the expression of high levels of negative emotions) with deficits in regulatory processes by using indicators that apply to both constructs (see Lewis, Zinbarg, & Durbin, 2010, for an eloquent discussion of these considerations). In order for a better understanding of the incremental utility of emotion regulation for psychopathology to emerge, greater efforts to differentiate it from near-neighbor constructs are essential.

The development of emotion regulation or dysregulation is thought to derive both from innate predispositions and from socialization. At the level of constitutional factors are various neural circuits and temperamental

characteristics. For example, inhibited children appear to bring a high state of reactivity into their environment, particularly in novel or unfamiliar situations. This biological propensity is thought to be the result of a number of neurological factors that include interrelating messages sent to and from neuroanatomical structures (*vis-à-vis* neuroelectricity and neuropharmacology) to the central and peripheral nervous system (Fox, Henderson, Marshall, Nichols, & Ghera, 2005). Cognitive and language development also contribute to emotion regulation. Growth in cognitive development allows the child increasingly to differentiate and cope with a diverse set of emotion-arousing stimuli. The development of emotion language also affords an opportunity for the communication of emotion meaning to others and its management through self-regulatory mechanisms (Cole et al., 1994; Thompson, 1994).

Finally, emotion regulation is also embedded within the unique context of the child. Socialization influences within the family, peer group, and culture are important in the development and expression of emotion, and may support or hinder emotion regulation in a variety of ways. One important influence is the way in which parents respond to the child's initial expressions of emotion, and how emotions are communicated in the context of the ongoing interactions between the parents and child (Cassidy, 1994; Volling, 2001). The development of emotion regulation may also come about through the modeling of appropriate or inappropriate emotional expression (e.g., Shipman & Zeman, 2001). Finally, the rules or boundaries of emotional expression, which are established by both the family and the community at large, also influence the development of emotion regulation (Cole et al., 1994).

Emotion dysregulation begins with context-specific efforts at self-regulation, which may then develop into more stable patterns of responding and thereby contribute to the development of psychopathology. The determination of emotion regulation as adaptive or maladaptive varies with the circumstances, but it generally involves the degree of flexibility of the response, the perceived conformity of the response to cultural and familial rules and boundaries, and the outcome of the response relative to the child's and parents' short- and long-term goals (Thompson, 1994).

Some forms of emotion dysregulation may be adaptive in one environment or at one time, but maladaptive in other situations or at other points in development (Fischer et al., 1997; Thompson & Calkins, 1996). For example, in discussing children who have been emo-

tionally and sexually abused, Terr (1991) describes the process of “numbing” (a symptom of a posttraumatic stress reaction), which serves to protect a child from overwhelming pain and trauma. However, when numbing becomes a characteristic way of coping with stressors later in life, it may interfere with adaptive functioning and with long-term goals. Another example stems from studies on attachment quality. In response to attachment figures that are rejecting or inconsistent, infants may develop an insecure/avoidant attachment in which emotional expression is minimized. Such an infant's reduced emotional expression, while serving the strategic function within the attachment relationship of minimizing loss by reducing investment in the relationship, may establish a pattern of emotional responding that is maladaptive for the development of subsequent relationships (Cassidy, 1994).

In summary, emotion theorists conceptualize the development of emotion regulation as involving a variety of increasingly complex developmental tasks. The degree of interference with these tasks depends on the characteristics of the child and his or her environment, as well as on their interaction. Emotion dysregulation is believed to be the consequence of interference in the associated developmental processes. Dysregulation is associated with a wide range of emotions; depending on the overall context, it may or may not become a stylistic pattern, and it may or may not lead to later psychopathology.

Genetic/Neurobiological Theories

In attempting to understand child psychopathology, genetic/neurobiological theorists recognize individual differences in genetically based, neurobiological characteristics and processes. From this perspective, mental disorders are represented in the brain as a biological entity (Insel et al., 2010). The goal of research in this field is therefore to characterize the genetic, structural, and functional brain bases of psychopathology. Diverse lines of research, including family and twin studies, molecular genetic, neurobiological, neurophysiological, and neuroanatomical studies, suggest a heritable, neurobiological basis for many childhood disorders, including ADHD, autism spectrum disorder, adolescent depression, pediatric bipolar disorder, social withdrawal, some anxiety disorders, and obsessive–compulsive disorder, to name a few. Research on brain structure and function using neuroimaging procedures has implicated specific brain regions for ADHD (e.g., Frodol

& Skokauskas, 2012; Peterson et al., 2009), anxiety disorders (De Bellis et al., 2002; McClure et al., 2007), autism spectrum disorder (Di Martino et al., 2009), and many other disorders, as reviewed in subsequent chapters. There is also increasing interest in neural network perspectives on disorder, as few disorders (if any) arise from a single brain region; such work has aimed to characterize both functional (Gaffrey, Luby, Botteron, Repovš, & Barch, 2012) and structural connectivity (e.g., Zielinski et al., 2012) between brain regions that work in conjunction to influence processes relevant to psychopathology (e.g., self-referential processing; Hamilton et al., 2011).

Neuroimaging studies tell us that one region or another may be involved, but they do not tell us why, and the findings for particular disorders are not always consistent from study to study, for children of different ages, or for boys versus girls. Furthermore, much of this work has failed to meet the standards of other types of research in the field of developmental psychopathology. For example, very little neuroimaging research adequately disentangles the time course by which disorder is linked to brain structure and function (i.e., differences in brain structure and activity can emerge as both causes and consequences of a disorder; longitudinal work is needed to address this possibility). In addition, many of these studies have used small samples, and other methodological inconsistencies raise questions about the robustness of some findings (Vul & Pashler, 2012). Research into specific neurotransmitters has also provided promising leads, although findings have also been inconsistent. One of the difficulties in research in this area is that many forms of child psychopathology involve the same brain structures and neurotransmitters, making it difficult to assess the specificity of their contributions to particular disorders. Such findings may reflect the limitations of existing categorical diagnostic systems, as discussed earlier in the section describing the RDoC initiative (Insel et al., 2010).

With respect to genetic influences on child psychopathology, familial aggregation has been viewed as an important initial step in providing evidence for genetic mechanisms. Once familial clustering is demonstrated, twin studies, adoption studies, segregation analyses, and linkage studies can be conducted (cf. Szatmari, Boyle, & Offord, 1993). “Familial aggregation” refers to the nonrandom clustering of disorders or characteristics within a given family, relative to the random distribution of these disorders or characteristics in the gen-

eral population (Szatmari et al., 1993). This paradigm rests on the premise that if there is a genetic component to a given disorder, the frequency of the phenotype (or manifest pathology) will be higher among biological relatives of the proband than in the general population (Lombroso, Pauls, & Leckman, 1994).

Twin studies are beneficial in helping to ascertain the contribution of genetic factors in the etiology of child psychopathology. The twin study approach emerged from the long-standing “nature versus nurture” or “genes versus environment” debate (Lombroso et al., 1994). Although twin studies provide a powerful research strategy for examining the role of genetic influences in both psychiatric and nonpsychiatric disorders, numerous methodological issues necessitate that caution be exercised in interpreting findings. For example, although Willerman (1973) found a concordance rate for hyperactivity of approximately 70%, this does not necessarily mean that 70% of the variance in hyperactivity is accounted for by genetic variation. Research suggests, for instance, that monozygotic twins spend more time together, frequently engage in similar activities, and have many of the same friends in common (Torgersen, 1993). Thus the common or shared environment presents a potential confound in any twin study, and unless twins are reared apart, or dizygotic twins are employed as the comparison group, it becomes difficult to separate the effects of genetic and environmental influences. Moreover, mutations can occur very early in cell proliferation in one twin fetus that result in phenotypic discordance (Czyz, Morahan, Ebers, & Ramagopalan, 2012). While such differences are clearly of genetic origin, they would be classified as “environmental” in parsing the variance in the trait under study. Representativeness and generalizability to the general population are other problems with twin studies (Lombroso et al., 1994; Torgersen, 1993). Growing up with a sibling of an identical age, for example, introduces its own special challenges (e.g., competition between siblings, greater dependency on each other) that make the twin environment unique.

Adoption studies have been used to circumvent some of the problems with twin and familial aggregation studies. They explicitly attempt to control for environmental variation in the heritability equation. The assumption behind this strategy is that when a disorder has a genetic etiology, the frequency of its expression should be greater among biological relatives than among adoptive relatives. Conversely, when environmental factors assume a larger role in the etiology of

psychopathology, the frequency of the disorder would be expected to be greater among the parents of adoptive relatives than among biological parents (Lombroso et al., 1994; Torgersen, 1993).

Several reasons may be advanced to account for the sparse number of investigations using the adoptive strategy. One obstacle has been the difficulty of obtaining reliable information regarding the biological parents of adoptees. The timing of adoption placements also represents a potential confound. Since children are typically adopted at different ages, it is difficult to determine what environmental influences the biological parents may have had during the earliest years of life (Lombroso et al., 1994). Similarly, many children are placed in residential settings prior to adoption; these conditions, which may affect a child's development, would be unaccounted for by an adoptive strategy. A confound analogous to the problem of timing is the high probability of being placed in an adoptive home that is similar to the home environment of the biological family. For instance, adoption agencies are quite strict in their criteria for adequate placements, and the adoptive home must, at a minimum, meet current middle-class standards (Torgersen, 1993).

However, while the aforementioned designs (i.e., family, twin, and adoption studies) play a vital role in providing evidence for the heritability of a disorder, and thus laying the groundwork for future research on genetic etiology, they are not equipped to identify specific genetic variants that play a role in the pathogenesis of disorder. The identification of etiologically relevant genes (i.e., those implicated in the pathophysiology of disorder) has the potential to greatly enhance our understanding of a disorder, as well as potential treatment mechanisms (Stodgell, Ingram, & Hyman, 2000). Toward this goal, the past few decades have witnessed rapid advances in researchers' ability to derive vast amounts of information on individual differences in genetic factors potentially relevant to disorder risk, and psychopathologists have accordingly availed themselves of these technologies. As a result, specific genetic variants have been implicated in virtually all forms of psychopathology (e.g., Allen et al., 2008; Gizer, Ficks, & Waldman, 2009; Levinson, 2006).

Unfortunately, replication of molecular genetic findings remains a significant concern, and there is disagreement regarding the best way forward in the search for the genetic bases of psychopathology (Hudziak & Faraone, 2010; Willcutt et al., 2010). Concerns raised in regard to efforts to model links between single genes

and disorder include (1) the fact that such designs fail to capture the polygenic basis of psychopathological phenomena; (2) the arguably low likelihood that diagnostic syndromes are adequate phenotypes for molecular genetic study; (3) the probability that some variants operate in a context-dependent manner (i.e., the case of gene–environment interaction, or $G \times E$); relatedly, (4) the fact that gene function is a dynamic phenomenon influenced by the environment, other genetic variants, and multiple epigenetic processes not captured by studies that assess genotype–phenotype associations only; and (5) the possibility that many cases of disorder are related to yet-to-be identified rare variants, making the a priori selection of candidate genes misguided. We address these points in the following few paragraphs.

Genome-wide association studies have emerged as a means of capturing polygenic influences on psychopathology, although their replication record appears variable, and overall effect sizes have been criticized for their small magnitude (Manolio, 2010; McCarthy et al., 2008); furthermore, it is unclear how to incorporate such studies within frameworks that also capture environmental influences on disorder, as well as $G \times E$. With respect to concerns regarding the use of diagnostic phenotypes, many investigators interested in the molecular basis of disorder elect to avoid the use of these entirely, focusing instead on endophenotypes (Gottesman & Gould, 2003), or markers of disorder risk that are thought to lie more proximal to the actions of genes than diagnostic outcomes. For example, endophenotypes related to neuropsychological function (such as reaction time variability, time reproduction, and response inhibition) have been applied to the genetic investigation of ADHD (e.g., Nigg, 2010), and biases in memory may be a promising endophenotype for depression risk (e.g., Hayden et al., 2006; Hayden, Olino, Bufferd, et al., 2013), although variability across studies in terms of how endophenotypes have been operationalized has made replication attempts difficult.

Regarding the conditional effects of genes, one of the more controversial directions in psychiatric genetics is the emergence over the past decade of studies testing $G \times E$, which attempt to capture the interplay between specific genetic variants and environmental risk factors in producing psychopathological outcomes (Kendler, 2011; Uher, 2011). Although the earliest of these seminal studies focused on psychiatric disorder in adults (e.g., Caspi et al., 2003), this literature has frequently focused on the role of early childhood adversity in potentiating the effect of genetic risk variants on

later disorder; this may account for the tremendous appeal of this approach to developmental psychopathologists, who tend to have a keen interest in the dynamic relationship between endogenous child and contextual risk factors. Moreover, studies identifying G×E hold the promise of accounting for the poor rate of replication of studies seeking to identify single-gene main effects, if many genetic influences are context-dependent or conditional. It is therefore not surprising that journals have been flooded with studies testing G×E across development.

Unfortunately, many of these studies are plagued by the same limitations found in poorer-quality molecular genetic association studies (e.g., small sample sizes; testing genetic influences on relatively complex, biologically implausible phenotypes), and may represent false-positive findings (Duncan & Keller, 2011). Also, attempts to model single-gene effects on complex psychiatric phenotypes, even within the context of environmental risk, may be misplaced. For many of the more popular G×E models, meta-analyses have been conducted that have supported (Karg, Burmeister, Shedden, & Sen, 2011; Kim-Cohen et al., 2006) and refuted (e.g., Risch et al., 2009) these findings. It has been argued that poor measurement approaches (e.g., self-report questionnaires) to the phenotype and the environmental context limit the ability of studies to detect true G×E (Monroe & Reid, 2008). Furthermore, Brown (2012) recently noted that studies of adults in which support for a G×E involving the serotonin transporter genotype and stress was evident were those in which adult stress could be interpreted as a marker of childhood adversity, suggesting that research on G×E should focus on developmental periods of greater plasticity (i.e., childhood), as this is when environmental moderation of genetic effects is unfolding. In conclusion, while it seems unquestionable that genetically influenced responses to the environment are an important force in risk for psychopathology, the question of how best to model this interplay has yet to be resolved.

Aside from these concerns, few would argue that the fact that studies of G×E are attempting to model an unknown, underlying biological process through statistical methods is an unimportant limitation. In other words, tests of G×E statistically model the conditional effects of genes without knowledge of the biological mechanisms through which these conditional effects emerge (Mill, 2011). While accurate genotyping of the loci implicated in psychopathology risk is now relatively affordable, a host of dynamic processes (see Mill,

2011, for an overview of these) known as “epigenetic influences” that further shape the actions of genes are less well understood or readily characterized to date, although these appear to play a more important role in gene function than previously thought. An emerging body of research is exploring epigenetic markers in psychiatric disorders and related processes in humans (Petronis, 2010); however, it is unclear whether the noninvasive methods available for human epigenetic research adequately reflect epigenetic processes in the human brain, which are presumably the most relevant to mechanisms of psychopathology.

Finally, it has been argued that, in sharp contrast to the widely held notion that disorder arises from the summed influence of many genes with small individual effects, individual rare variants with a large, harmful impact on neural function play a key role in the genetic basis for psychiatric disorder (McClellan & King, 2010). Although such rare variants by definition do not account for a large number of cases of disorder, the hope is that through their study, a better understanding of the pathophysiology of disorder can be gained. This is a relatively new approach that, due to its novelty, is difficult to evaluate in terms of the insights it has yielded to date.

SUMMARY AND CONCLUSIONS

In this introductory chapter, we have described a developmental–systems framework for child psychopathology that emphasizes three central themes: (1) the need to study child psychopathology in relation to ongoing normal and pathological developmental processes; (2) the importance of context in determining the expression and outcome of childhood disorders; and (3) the role of multiple and interacting events and processes in shaping both adaptive and maladaptive development. The research findings presented in the subsequent chapters of this volume illustrate the importance of these themes for understanding children and adolescents displaying a wide range of problems and/or disorders.

A developmental–systems framework eschews simple linear models of causality and advocates for a greater emphasis on systemic and developmental factors and their interactions in understanding child psychopathology. Multiple etiologies and their interplay represent the norm for most forms of child psychopathology. For example, in the study of conduct disorder, genetic in-

fluences, constitutional factors, insecure attachment relationships, impulsivity, biased cognitive processing, parental rejection, a lack of parental supervision, interpersonal difficulties, and many other influences have been implicated. However, many of these influences have also been implicated in other disorders, and not all children who exhibit such risks develop conduct disorder. There is a need for research that will help to disentangle the role of these multiple sources of influence and their interactions in relation to different childhood disorders.

We have argued that all forms of child psychopathology are best conceptualized in terms of developmental trajectories, rather than as static entities, and that the expression and outcome for any problem will depend on the configuration and timing of a host of surrounding circumstances that include events both within and outside a child. For any dynamically changing developmental trajectory, there also exists some degree of continuity and stability in structure, process, and function across time. Understanding such continuity and stability in the context of change represents a challenge for future research; it necessitates that psychopathology in children be studied over time, from a number of different vantage points, utilizing multiple methods, and drawing on knowledge from a variety of different disciplines.

Given the complexities associated with a developmental–systems framework for understanding child psychopathology, there is a clear need for theories to guide our research efforts. We have argued that a developmental psychopathology perspective provides a broad macroparadigm for conceptualizing and understanding childhood disorders in general, and that complementary disorder- and problem-specific theories are also needed to account for the specific configurations of variables commonly associated with particular disorders. Such problem-specific theories are presented in the subsequent chapters of this volume. The conceptualization of child psychopathology in terms of developmental trajectories, multiple influences, probabilistic relationships, and diverse outcomes suggests that some influences are likely to be common to many different disorders and that others are probably specific to particular problems. Our theories need to account for both types of influence.

As we have seen, childhood disorders constitute a significant societal problem, and in the absence of an empirically grounded knowledge base, unsubstantiated theories have frequently been used as the basis

for developing solutions to these problems. There is a pressing need for further longitudinal research to inform our intervention and prevention efforts. If such work is to succeed in capturing the multiple interacting influences and changes over time outlined in this chapter, such research will require new ways of conceptualizing childhood disorders; greater collaboration across disciplines; and the use of novel technologies, sophisticated designs, and complex statistical tools. Considerable advances have been made in all of these areas since earlier editions of this book appeared. The chapters in the present volume provide a state-of-the-art review and critique of current definitions, theories, and research for a wide range of childhood disorders. They also identify current needs and forecast likely future directions for research into child psychopathology.

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NOTES

1. As a matter of convenience, we use the terms “children” and “child” in this chapter and volume to refer to children of all ages, from infancy through adolescence. The diversity within this wide age range will necessitate the use of more specific designations of age and developmental level as appropriate to each discussion. We use the terms “child psychopathology” and “developmental psychopathology” interchangeably in this chapter and in this volume. Other terms that have been used to describe problems during childhood are “abnormal child psychology,” “childhood disorders,” “atypical child development,” “childhood behavior disorders,” “childhood emotional and behavioral problems,” and “exceptional child development.” These differences in terminology reflect the many disciplines and theoretical perspectives that are concerned with understanding and helping disturbed children.

2. We recognize that theory and research in child psychopathology need to be put to the test in the applied arena. However, in this volume we do not consider in any detail the range of assessment, treatment, or prevention strategies available for the problems under discussion. Our decision not to address assessment, treatment, and prevention in this volume was based on two factors. First, we perceived a need for a

substantive review of what we currently know about childhood disorders. Many current treatments for childhood disorders are relatively untested (Kazdin, 2000; Mash & Barkley, 2006), and it was felt that future efforts to test treatment approaches would benefit from a detailed discussion of our current knowledge base for child psychopathology. Second, we wished not to dilute the discussion of theory and research in child psychopathology by attempting to provide cursory coverage of assessment and intervention. Instead, we refer the reader to companion volumes to this one, which have as their primary focus child assessment (Mash & Barkley, 2007) and child treatment (Mash & Barkley, 2006), respectively.

3. A complete discussion of the scope and complexity of issues surrounding the concept of harmful dysfunction is beyond the scope of this chapter. The reader is referred to papers in the *Journal of Abnormal Psychology* (see Clark, 1999, for an overview) and in *Behaviour Research and Therapy* (Houts, 2001; McNally, 2001; Wakefield, 1999a, 1999b, 2001) for excellent discussions of these and related issues.

4. ICD-10 is currently under revision, and ICD-11 is expected to appear in 2015. For information about ICD-11, see its website (www.who.int/classifications/icd/revision/icd-11faq/en).

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