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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., Luebbe, 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 influences; 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 psychopathology 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 serious 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 increasing 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 parents 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. Notwithstanding 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, Shirtcliff, & 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 childrearing 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 subclinical 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 exhibit 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