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Internalizing problems of childhood and adolescence: Prospects, pitfalls, and progress in understanding the development of anxiety and depression

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Abstract
The focus of this article is on internalizing problems that are experienced by children and adolescents. We provide an historical perspective, selectively examine the current state of knowledge, consider advances and gaps in what is known, and identify new research directions. Diagnosis, epidemiology, theory, and research first are considered separately for anxiety and depressive disorders. These internalizing problems, however, whether clinical or subclinical, share many common features and show high comorbidity rates. We emphasize the importance of systematic analysis of comorbid anxiety and depression, including their comorbidity with externalizing problems. This could lead to more valid classification of subtypes of internalizing problems and further an understanding of the diverse conditions that constitute internalized distress. We highlight the need to study anxiety and depression within a developmental psychopathology framework, as well as to include both categorical and dimensional assessments of these problems in the same research designs. This will be essential for understanding the complex interplay of biological and environmental processes that contribute to the emergence, progression, and amelioration of internalizing problems over time.

The most common forms of child and adolescent psychopathology have been categorized into two broad classes: internalizing and externalizing problems. Whereas externalizing problems are characterized by behaviors that are harmful and disruptive to others, internalizing disorders signify a core disturbance in intropunitive emotions and moods (e.g., sorrow, guilt, fear, and worry). In fact, both types of dysfunction have behavioral and affective components, as well as characteristic cognitive features. There is also substantial comorbidity of internalizing and externalizing disorders. The primary focus here is on internalizing problems. We provide an historical overview, selectively review the current state of knowledge, consider advances and gaps in what is known, and identify new research directions.

We consider both clinical and subclinical anxiety and depression. Initial theories and research on anxiety and depressive disorders proceeded largely along independent tracks. Although we follow this precedent for historical clarity, communalities soon become evident, reflecting in part the high comorbidity rates for anxiety and mood disorders (Nottlemann & Jensen, 1995). Investigation of comorbid conditions and also their links to externalizing problems will be central to advances in understanding the origins and development of different types of internalizing problems.

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We highlight the need to consider anxiety and depressive disorders within a developmental psychopathology framework, in order to address a number of core questions. How and why do these serious emotional disturbances emerge over the course of childhood and adolescence? Are there developmental precursors of these disorders in adolescents and adults? How are anxiety and depression conceptualized during the first 2 decades of life? What are some of the major biological and environmental factors that contribute to their emergence? To their co-occurrence? To continuity and change over time? How can the study of normative biological, developmental, and socialization processes advance understanding of the etiology and development of internalizing problems?

Psychiatric diagnoses of anxiety and mood disorders are based on discrete classification systems. A clinical disorder is a distinct entity, construed as a disease; it is either present or absent, depending upon whether specific diagnostic criteria are met. Disorders have traditionally been viewed as qualitatively different from the lesser problems of the "walking wounded" or "worried well." In psychology, dimensional classification systems typically are used to characterize internalizing problems (i.e., there is a gradation or continuum of severity of disturbance). This approach does not yield psychiatric diagnoses, though scores typically are normed, in an effort to distinguish normal, subclinical, and clinical problems. There also tends to be less differentiation among subtypes of internalizing problems in dimensional than categorical systems. Factor analytic work has provided a rationale for combining symptoms of anxiety and depressed mood into one scale (Achenbach, 1991). At the same time, this precludes comparisons of the two most prominent types of internalizing problems.

We consider both categorical and dimensional perspectives in this article. The different approaches have led to a series of questions that have just begun to surface. Do risk and protective factors known to influence the development of internalizing disorders also function on a continuum? In other words, do lower levels lead to less severe disturbance seen in normal and subclinical problems? Or instead, do different etiologic factors operate to produce different levels of severity of problems? In what ways might early subsyndromal forms of internalized distress increase the later likelihood of diagnosed anxiety or depressive disorders? Different definitions of problems, heterogeneity of measures, variations in samples, and severity of problems studied across disciplines have made generalizations difficult. The current article reflects both the tensions and opportunities inherent in attempts to integrate psychiatric and psychological approaches. This integration is one of the major tasks in the new century.

The study of internalizing disorders in children and adolescents originally was based on application of adult models to earlier periods of development. This did not encourage, and sometimes impeded, an understanding of the origins and development of these disorders. However, with the advent of a developmental psychopathology perspective, there has been an increase in the use of longitudinal and age cross-sectional research designs to provide information about adaptive and maladaptive functioning in childhood and adolescence. This makes it increasingly possible to develop explanatory models and treatments of internalizing disorders that are grounded in a firm understanding of the phenomenology of these problems in childhood and adolescence.

Anxiety Disorders

Diagnosis and classification

The diagnosis and classification of anxiety disorders in children and adolescents is relatively new. The first *Diagnostic and Statistical Manual of Mental Disorders* used to classify these problems (DSM-II; American Psychological Association [APA], 1968) recognized the existence of two disorders: withdrawing reaction and overanxious reaction. The DSM-III (APA, 1980) marked a historic shift to empirically based, behaviorally descriptive diagnostic criteria. A separate diagnostic section on "anxiety disorders of childhood and adolescence" included separation anxiety disorder, overanxious disorder, and

avoidant disorder of childhood or adolescence. The DSM-IV (APA, 1994) identifies separation anxiety disorder as the only anxiety disorder unique to childhood. Overanxious disorder and avoidant disorder now are subsumed under generalized anxiety disorder and social phobia, respectively. There are several other anxiety disorders in DSM-IV that apply to both adults and children (e.g., panic disorder, agoraphobia, specific phobia, social phobia, obsessive–compulsive disorder, generalized anxiety disorder, and posttraumatic stress disorder).

Epidemiology

Advancements in identification and diagnosis of anxiety disorders in children have contributed to improved epidemiological studies (and vice versa). As recently as the mid-1980s, in a review of the extant research (Orvaschel & Weissman, 1986) no data were available regarding anxiety disorders. A decade later in a comprehensive review (Costello & Angold, 1995), prevalence rates of 5.7–17.7% for any anxiety disorder in children were reported. Other studies have confirmed similar rates. Differences in prevalence rates may reflect several factors. Differentiation of anxiety symptoms from anxiety disorders has largely depended upon the presence or absence of functional impairment and subjective distress criteria. The inclusion of functional impairment criteria has been shown to decrease rates of anxiety disorders. The informant may also influence rates of reported cases (e.g., children often report more anxiety symptoms than are seen in their parents' reports).

Age-related appearance of specific anxiety disorders in children has been well documented (e.g., Costello & Angold, 1995). Separation anxiety disorder, the fear of separation from primary caretakers, presents most frequently during early and middle childhood. Specific phobias (i.e., fear and avoidance of identifiable objects or situations) have been described in children of all ages. Elevated rates of animal phobias may appear in early childhood in comparison to social-related phobias in adolescence (Marks & Gelder, 1966). Generalized anxiety disorder is characterized by pervasively excessive worry and is seen most often in older children and adolescents. Social phobia (i.e., fear and avoidance of social and performance situations) becomes more common in middle childhood and adolescence. Panic disorder is rare in childhood but more frequent in adolescence. The emergence of panic disorder may be associated with puberty (Hayward, Killen, Hammer, Litt, & Wilson, 1992). Obsessive–compulsive disorder consists of repetitive intrusive thoughts (obsessions) or behaviors (compulsions). Onset occurs during early and middle childhood (e.g., Swedo, Rapoport, Leonard, Lenane, & Cheslow, 1989; Riddle, 1998).

Theory and research

Our contemporary understanding of biological, environmental, and developmental factors in the etiology of anxiety disorders reflects a preceding era characterized by periods of critical reappraisal and integration. Shifts from general to specific, singular to integrative, and conceptual to quantitative can be seen within and between major orientations of study. A historical review of representative theories and research exemplifies some of these transitions.

Psychoanalytic, psychodynamic, and relational theories. Freud (1936) attributed anxiety to the presence of unconscious infantile libidinal or aggressive wishes towards parental figures, a conception that lost favor over time. However, the focus on childhood experiences and the quality of the parent–child relationship in shaping children's sense of security as well as their anxieties and fears has remained central to several of the psychodynamic and relational theories that followed. The collaboration of a psychoanalyst, John Bowlby, and a developmental psychologist, Mary Ainsworth, led to theory-driven research on the early formation of anxiety problems that develop in the context of mother–infant interactions and depend on the nature of the attachment relationship. Caregiver sensitivity and consistency contribute to secure attachment, presumably by heightening the infant's comfort and sense of safety (Ainsworth,

1978; Bowlby, 1973, 1988). Attachment theorists proposed a relationship between insecure attachment (anxious–resistant) and the development of anxiety in children. Anxious–resistant attachment is, in fact, associated with increased fearfulness and inhibited behaviors in children (e.g., Cassidy & Berlin, 1994). It also predicts later anxiety disorders in children and adolescents (Warren, Huston, Egeland, & Sroufe, 1997).

The theoretical models and empirical research generated by Bowlby and Ainsworth reflect one of the first efforts to investigate the etiology of (internalizing) disorders within a developmental psychopathology framework. Their pioneering work led to research not only on the beneficial effects of sensitive caregiving but also on the adverse consequences of negative parenting for children's anxiety. Examples of the latter include parental restriction and negative feedback (e.g., Krohne & Hock, 1991), parental high control leading to perception of low personal control (e.g., Chorpita & Barlow, 1998), and maternal intrusiveness and overprotectiveness (e.g., Bowen, Vitalo, Kerr, & Pelletier, 1995).

Behavioral and cognitive learning theories. Pavlov's classical conditioning model of anxiety, first used to study the learning of fear in animals, soon was extended to research with humans. Its early application to children was seen in the case of "Little Albert," a young child who was conditioned to develop a specific phobia (Watson & Rayner, 1920). Several theorists (e.g., Rachman, 1977; Seligman, 1971) challenged the overencompassing characteristics of the original behavioral model, noting that some stimuli are more biologically prone to become feared objects. Also, several additional learning processes have been identified over time. Anxiety could be learned from caregivers or others in the child's environment (e.g., through modeling and contagion of anxious, fearful behavior; explanatory styles that emphasize frightening and dangerous aspects of life; or specific child-rearing and discipline practices likely to induce fear in children through threats or actual infliction of harm). While causal inferences about the environment in the learning of anxiety could be drawn from early experimental studies, generalizations from naturalistic research are less straightforward. Parents who "model" anxiety and their children who "reproduce" it may also indicate shared genetic susceptibility either to anxiety problems per se or to biologically based, predisposing characteristics (e.g., low thresholds for physiological arousal).

The role of cognition in theories about anxiety has been emphasized in recent decades. Research on anxiety disorders in adults describes a bias toward increased attention to threatening or dangerous situations (e.g., Beck & Clark, 1997). Research on anxiety in children suggests similar cognitive processes. Anxious children have been found to interpret ambiguous information as threatening more often than nonanxious children (e.g., Hadwin, Frost, French, & Richards, 1997). Biased attention to signals of threat or danger are thought to create cognitive distortions through a process of overactivation. Biased attention could occur for a number of reasons. In addition to socialization experiences that sensitize children to dangerous, anxiety-provoking conditions, temperament may render some children particularly vigilant and vulnerable to overactivation.

Biological models

Genetic influence. Twin and adoption studies support the theory that heritable factors play some role in the expression of anxiety problems and disorders (Kendler, Neale, Kessler, Heath, & Eaves, 1992). There is a familial pattern seen in the pathogenesis of childhood anxiety disorders. For example, there is an increased prevalence in first-degree relatives of children with anxiety disorder compared with relatives of children with attention-deficit hyperactivity disorder (ADHD) or never psychiatrically ill children (e.g., Last, Hersen, Kazdin, Orvaschel, & Perrin, 1991). While twin and family studies provide indirect evidence of heritability, specific genetic mechanisms at the molecular level have not been identified and environmental influences are also likely to be operative.

Temperament. Particular temperament traits may be early indicators of fearfulness or anxiety. Behaviorally withdrawn and inhib-

ited behavior when encountering unfamiliar persons and situations is one such trait (Kagan, Reznick, Snidman, Gibbons & Johnson, 1988). It is postulated to reflect a lowered threshold of activity in limbic and hypothalamic structures and has been associated with increased sympathetic activity (Kagan, Reznick, & Snidman, 1987). Family and twin studies indicate a relationship between behavioral inhibition and increased risk of anxiety disorders (see review by Turner, Beidel, & Wolf, 1996). Generalized social anxiety at adolescence (but not specific fears, separation anxiety, or performance anxiety) can be predicted from behavioral inhibition in the 2nd year of life, though only for girls, suggesting the moderating effects of gender (Schwartz, Snidman, & Kagan, 1999).

Physiological regulatory processes. Research on the psychophysiology of internalizing (and externalizing) problems has produced models wherein both classes of psychopathology have been associated with distinct physiological profiles. The focus has been mainly on the autonomic nervous system (ANS) and the hypothalamic–pituitary–adrenal (HPA) axis systems, both of which are involved in the body's stress response. Anxiety symptoms and disorders have been associated with high heart rates (e.g., Gerardi, Keane, Calhoon, & Klauminzer, 1994) and delayed electrodermal habituation (Birket–Smith, Hasle, & Jensen, 1993). Children hospitalized with separation anxiety disorder were found to have higher heart rates than other patients (Rogeness, Cepeda, Macedo, & Fischer, 1990).

The HPA axis system is important in the regulation of arousal (Gold, Goodwin, & Chrousos, 1988; Stansbury & Gunner, 1994). Healthy adaptation (indeed survival) relies on the ability to produce increased levels of cortisol under stress and to reduce production once stress has abated. However, chronic exposure to stress may predispose individuals to anxiety and affective disorders, by causing long-term overproduction of hormones such as cortisol in the HPA system. Children and adolescents with internalizing problems tend to have higher baseline cortisol, whereas those with externalizing problems tend to have lower baseline cortisol than control groups (see review by Stansbury & Gunnar, 1994). Youths with clinical levels of anxiety have shown high cortisol reactivity relative to controls, in response to a social challenge (Granger, Weisz, & Kauneckis, 1994).

Neurobiological processes and brain circuitry. Advances in science and technology over the past few decades have contributed greatly to our understanding of the biological substrates of adult anxiety disorders. Studies indicate the likely involvement of multiple neurotransmitters and brain structures. Abnormalities in the serotonin, noradrenergic, and GABAergic systems have been implicated (Johnson & Lydiard, 1995; Longo, 1998). There has been very little research with children.

Gray's neurophysiological model of anxiety focuses on approach and withdrawal patterns (Gray, 1982, 1991). They are part of the fight–flight system that responds to protect the organism. Research on neural regulation suggests differentiating roles of left and right anterior frontal lobe brain regions in controlling approach and withdrawal behaviors (Davidson & Fox, 1982; Fox, 1991). The left frontal brain region is implicated in approach behaviors and positive emotions, while the right frontal region is associated with withdrawal behavior and negative emotions. Infants observed to be high on motor activity and negative affect have shown greater right frontal activation at 9 months of age and more inhibited behavior at age 14 months (Calkins, Fox, & Marshall, 1996). In another study (Fox, Rubin, Calkins, Marshall, Coplan, Porges, Long, & Stewart, 1996), increased right frontal EEG activation was observed in socially inhibited 4-year-old children. In contrast, left frontal activation was correlated with outgoing behavior. These studies provide additional evidence for biological manifestations of inhibited behavior thought to play a role in the development of anxiety disorders.

Functional neuroimaging research also provides insights into the neurobiology of anxiety disorders (Rauch, Savage, Alpert, Fischman, & Jenike, 1997). Anatomical brain circuits connecting specific brain regions may be involved, and disruption of components at

varying points in the circuit may produce similar symptoms. Here too, research has been based primarily on adult and animal models. Pine and Grun (1999) review neuroanatomical models of childhood anxiety correlates of adult phobias, generalized anxiety, and panic disorder. These disorders are postulated to reflect abnormalities in limbic-based amygdala, septohippocampal, and brain stem–hypothalamic circuits. A potentiated startle reflex via enhanced amygdala activation may identify children at risk for phobic disorders (Davis, 1997; Grillon, Dierker, & Merikangas, 1997). Septohippocampal dysfunction is believed to contribute to generalized anxiety disorder in adults and children (Daleiden & Vasey, 1997). Neurocircuits connecting brain stem periaquaductal gray and the medial hypothalamus may play a role in the etiology of separation anxiety and panic (Panksepp, 1998; Shekhar & Keim, 1997). Caution is warranted, as little is yet known of the biological development of specific brain circuits and how this relates to development of anxiety.

Bridging research efforts are needed to further our understanding of the complex interplay of biological and environmental factors that lead children toward or away from having anxiety problems. Davidson and Rickman (in press) report associations between right frontal brain activation and behavioral inhibition, in a sample of children studied at both 3 and 9–10 years of age. Inhibition was stable over time, only for a small subgroup that also had shown right frontal asymmetry at both time points. In keeping with a diathesis–stress model, the authors speculate that behavioral inhibition may not remain stable for most children with a biological predisposition, due to the impact of early training and experience on neural plasticity. Many individuals are saturated with particular emotional climates from early in life. These different emotional environments could contribute either to stability or change in inhibition over time, through differential, experiential shaping of the emotional circuitry of the brain. While Davidson and Rickman emphasize the importance of early experience, plasticity (and hence shaping) is possible at other points in development. Adolescence is a period of rapid change, where parental influences on children's emotional states have been found (Ge, Best, Conger, & Simons, 1996; Ge, Conger, Lorenz, Shanahan, & Elder, 1995). This may be another "critical period" in which to study the interaction of nature and nurture.

Depressive Disorders

Diagnosis and classification

Our focus is on the two most common mood disorders, major depressive disorder (MDD) and dysthymic disorder (DD; APA, 1994). Manic–depressive disorder is rare in childhood and adolescence and is not considered here. There is relatively little systematic data available on its phenomenology and rates of occurrence. This is changing, however, and there is now heightened interest in the study of bipolar disorder within a developmental framework (Akiskal, 1998; Carlson, Bromet, & Sievers, 2000). By the 1970s, similarities were noted in childhood and adult unipolar depression, and they came to be considered isomorphic disorders (e.g., Malmquist, 1971). Depression in children and adolescents began to be diagnosed using adult criteria. Major depression in childhood is characterized by a period of disturbance in mood that may include depressed affect, anhedonia, or irritability. Cognitive or vegetative symptoms must also be present. Dysthymic disorder is a persistent, milder, but more chronically depressed mood (or irritability for children). Cognitive or vegetative symptoms also may be present. With few exceptions (e.g., irritability manifest instead of depressed mood, duration criteria for DD), DSM-IV criteria for child and adult mood disorders are nearly identical.

Epidemiology

Overall the prevalence rate of MDD children ages 4–18 years is between 2 and 8% (Poznanski & Mokros, 1994). Rates are low in childhood. Less than 1% of preschoolers and about 2% of school-aged children meet criteria for MDD (Kashani & Orvaschel, 1988), but by adolescence the rates are between 2

and 8%. There is an increase in prevalence between ages 15 and 18 years. Adolescents report lifetime prevalence rates of MDD as high as 15–20%, comparable to adult rates (e.g., Lewinsohn, Clarke, Seeley, & Rohde, 1994). The prevalence of DD is less than 2% in childhood and up to 8% in adolescence (Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993). Children whose first affective episode is DD are about 2 years younger at time of onset than children whose first episode is MDD (Kovacs, Akiskal, Gatsonis, & Parrone, 1994).

There are also age changes in the phenomenology of depression. In a comparison of depressive symptoms in children and adolescents with MDD (Ryan, Puig–Antich, Ambrosini, Robinovitch, Robinson, Nelson, Iyengar, & Tworney, 1987), many similarities were found (e.g., severity, depressed mood, insomnia, irritability, suicidal ideation). However, prepubertal children had more somatic complaints and psychomotor agitation (as well as separation anxiety and phobias), while adolescents had more anhedonia, hopelessness or helplessness, and hypersomnia. Relative to adults, adolescents are more often changed by an episode of major depression, in ways suggesting that early-onset depression is a more pernicious, severe form of the disorder (Rohde, Lewinsohn, & Seeley, 1994).

Theory and research

Psychoanalytic, psychodynamic, and relational theories. Beginning with Freud, psychoanalytic theories have emphasized the importance of unresolved childhood experiences that result in adult depression, in particular the loss of a real or imagined love object. One common theme is that repeated disappointments and parental failures to meet the child's psychological needs may give rise to depression. Unconscious reactions to early childhood circumstances, which also included sexual and aggressive impulses toward parents, were thought to result over time in harsh superego development, as extreme guilt, high standards, and self-blame became internalized. Adult depression, but not childhood depression, was believed to result from these internalized processes. It was assumed that children did not have the ego- and cognitive-developmental capabilities to experience sustained guilt, misery, and despair (i.e., those symptoms of internalized distress that are part of the depressive experience). Later it was proposed that depression in children might be "masked" or manifest as "depressive equivalents," seen in somatic complaints or behavioral disturbances (e.g., Cytryn & McKnew, 1972; Sperling, 1959). It is now clear that young children are capable of the experiences implicated in the phenomenology of depression, such as excessive guilt (see review by Zahn–Waxler & Kochanska, 1990) and other intropunitive emotions. Spitz and Wolf's (1946) seminal work on anaclitic depression in infants in orphanages indicated the presence of depressive syndromes very early in life. Acknowledgment that young children are capable of grief reactions (Bowlby, 1960) also helped pave the way for theory and research on the development of depression in childhood.

Psychodynamic views of family relations influenced later research by developmental psychologists on parenting practices and family processes associated with child and adolescent depression. Based on an attachment perspective, healthy development was thought to be promoted by a family environment characterized both by close relationships and the fostering of autonomy. Low levels of parental warmth and support, as well as parental rejection, hostility, and family conflict, are known to be associated with depression in children and adolescents (McCauley, Pavidis, & Kendall, in press). Longitudinal research designs demonstrate the predictive power of observed parental characteristics such as hostility and low warmth on adolescents' depressive symptoms over time, controlling for initial symptoms levels (Ge et al., 1996). Other observational research also has identified adverse conditions (i.e., less supportive, more conflictual family environments associated with greater depressive symptomatology in adolescents, both concurrently and prospectively; Sheeber, Hops, Alpert, Davis, & Andrews, 1997). Longitudinal research has tended to focus on samples with low to moderate levels

of depressive symptoms. The impact of the environment on depressive *disorders* is unknown and remains an important topic for investigation. An increasingly prominent view attributes youth depression to a combination of psychological stress and biological vulnerability that impact the individual (i.e., a diathesis-stress model; Post, Weiss, Leverich, George, Frye, & Ketter, 1996).

Behavioral and cognitive learning theories. Behavioral theories of depression emphasize the importance of learning and environmental contingencies. The initial demonstration of "experimental neurosis" in Pavlov's laboratory led to a host of studies using similar paradigms to create symptoms of anxiety and depression (reviewed by Mineka & Kihlstrom, 1978). Overmier and Seligman's (1967) study was the first, however, to have specific implications for depressive symptoms. Dogs initially exposed to uncontrollable shock became impaired in subsequent efforts to escape from or control shock. The model has been applied to other species (Maier & Seligman, 1976; Mineka & Henderson, 1985), culminating in the "learned helplessness" model of depression where the organism learns to give up in challenging, aversive, stressful situations.

The concept of depression stemming from learned helplessness was expanded to include cognitive distortions characterized by certain attributional styles in adults (e.g., failure attributed to stable, global, internal factors; Seligman, Abramson, Semmel, & von Baeyer, 1979). A crosscutting theme of several cognitive–behavioral theories is that repeated, uncontrollable aversive environmental experiences create distortions (e.g., negative schemas, biased attributions, negative self-concept) or behavior deficits (e.g., deficient social skills). Distortions thus can create risk for or become part of the depressive experience. While most cognitive–behavioral theories originally focused on adult depression, there have been downward age extensions to childhood and adolescence. Several studies have shown associations between depression and cognitive style in children that are consistent with adult models (e.g., Seligman, Peterson, Kaslow, Tanenbaum, Alloy, & Abramson, 1984). Greater integration of this literature with developmental research on self-concept and emotion regulation is warranted, given the low self-regard and dysregulated emotions that characterize depression.

Biological models.

Genetic influence. Twin and adoption studies in adult populations suggest that genetic factors account for at least 50% of the variance in the transmission of mood disorders. Although genetic factors may increase risk for depression, environmental factors, particularly nonshared experiences within and outside the family, including differential treatment of children by parents (Plomin, 1994) also have an impact. The interaction of genetic and environmental factors has been hypothesized in the development of more severe forms of depression (Rende, Plomin, Reiss, & Hetherington, 1993). Increased prevalence of depressive disorder has been observed in relatives of depressed youth. Children of a depressed parent are at substantial risk for depression, and risk increases further if both parents are affected (Birmaher, Ryan, Williamson, Brent, Kaufman, Dahl, Perel, & Nelson, 1996; McCauley et al., in press).

Physiological regulatory processes. In contrast to research on anxiety, there has been little emphasis on the role of temperament in depression. Physiological processes, however, have been studied and many factors associated with anxiety are also part of a depressive profile in adults. High resting heart rates and low electrodermal activity and reactivity typically have been found for adult depressed patients (Lahmeyer & Bellur, 1987; Thorell, Kjellman, & D'Elia, 1987), a pattern reported earlier for anxiety. Little research has been done with depressed children and adolescents.

Disturbances in HPA axis function also are associated with depression. Many adult patients tend to have elevated basal cortisol levels, show a flattening of the circadian rhythm, and fail to suppress cortisol production in response to the DST test (Akil, Haskett, Young, Grunhaus, Kotun, Weinberg, Greden, & Watson, 1993). This line of research has been extended to the study of depressed children and adolescents. Evidence of HPA axis dysregula-

tion has not been observed in children and adolescents with the frequency and consistency seen in studies of depressed adults. Several studies of baseline plasma cortisol secretion comparing depressed child and adolescent outpatients with controls revealed no significant differences. Some other studies have found results similar to those with depressed adults who fail to show cortisol suppression following the Dexamethasone Suppression Test (DST; see review by Birmaher et al., 1996).

Neurobiological processes and brain circuitry. Consistent with research on anxiety disorders in adults, several neurotransmitters have been identified in the pathophysiology of mood disorders, including abnormalities in the serotonergic, noradrenergic, and GABAergic systems. Information about brain regions and circuitry associated with depression in adults has been derived from accumulated research based on brain lesions, neuroimaging, and electroencephalographic (EEG) activity. As previously discussed, the left frontal brain region is associated with approach and positive emotions; the right frontal lobe is involved in internalizing patterns of withdrawal and negative emotions that include sadness and depressed mood. Studies that investigate mood have implicated both frontal and parietal regions of both left and right hemispheres (Davidson, 1994; Heller, 1990). Individuals with depressed moods show frontal lobe asymmetry, reflected mainly in low activation of the left hemisphere relative to controls. Depression also has been associated with reduced activity in the right parietal region. Relations between frontal and parietal lobe activity remain to be studied, as do interconnections of cortical and limbic systems implicated in the regulation of moods, including depression. Neurochemical transmission between limbic and cortical structures that affect arousal and activation of various brain structures may be a crucial link in the cortical–subcortical interaction (Heller, 1990).

While research with children and adolescents is quite limited, some studies indicate patterns of brain activity in children at risk that parallel findings with depressed adults. Infants and young children of depressed mothers show reduced left frontal EEG activity (e.g., see Dawson, Frey, Self, Panagiotides, Hessl, Yamada, & Rinaldi, 1999). This pattern has been replicated many times, and recent work suggests the role of environmental processes in patterns of brain asymmetry related to emotionality. Reduced left frontal EEG activity can be predicted from observed maternal behaviors toward their infants during play even after accounting for maternal depression (Dawson, Frey, Panagiotides, Self, Hessl, & Yamada, 1997). These mothers were less affectionate, less engaged during play, and more intrusive.

Post et al. (1996) have presented a model that places affective illness in the context of an evolving developmental neurobiological framework and a series of molecular neurobiological adaptations. Environmental experiences are postulated to mediate effects on gene expression, including psychosocial stressors and the neurobiology of episode recurrence. In this view, social support may be capable of generating an inhibitory effect on illness progression by decreasing the perception and neurobiological impact of stressors (even at the level of gene expression).

Anxiety and Depression: A Developmental Psychopathology Perspective

Developmental psychopathology has been defined as "the study of the origins and course of individual patterns of behavioral maladaptation" (Sroufe & Rutter, 1984, p. 18), with an emphasis as well on factors that contribute to resilience and adaptive functioning (Cicchetti, Ganiban, & Barnett, 1991; Cicchetti & Schneider–Rosen, 1984; Cicchetti & Toth, 1995, 1998). Because many psychological problems generally do not arise de novo, but emerge over time from prodromal or subclinical symptoms, a developmental perspective permits study of the evolution of a disorder. It is assumed that the organism and the environment are mutually and interactively involved in determining developmental course. Even with an established history of problems, the particular form of psychopathology may still be in formative stages (Newman, Moffitt, Caspi, Magdol, Silva, & Stanton, 1996). What

factors contribute to stability or change in internalizing problems over time? To improvement or deterioration in functioning? To shifts in types of disorders (e.g., from anxiety to depression)? To different patterns of comorbidity?

Recent conceptual, methodological, and analytic advances make it increasingly possible to incorporate key factors into transactional, longitudinal research, designed to test ways in which nature and nurture interact to affect developmental outcomes. We now know more about putative biological and environmental factors that confer risk or protection. However, we lack data that would help to distinguish atypical from normative patterns of development. For example, how do frontal lobe activity (EEG) and reactivity to stress (ANS and HPA axis activity) differ as a function of age? What constitutes nonnormative reactions at any given age, as well as across age? Similarly, how do environmental processes likely to include observational methods (e.g., affective climate, parental warmth or rejection, expression of authority, instructional and explanatory style) differ as a function of age? We also lack descriptive data on normative and nonnormative aspects of the experience, expression, and regulation of emotion, as well as many aspects of social and interpersonal development. How do these processes differ at a specific point in time, or across time, including developmental variations that may result from different socialization experiences? Considerable research effort will need to be devoted to this issue in the 21st century. There are several other issues central to an understanding of the phenomenology and etiology of internalizing problems from a developmental perspective that have not received adequate attention. We consider them next. They include the roles of emotion, comorbidity, gender, and culture.

Emotions and Internalizing Problems

Emotion regulation and dysregulation

The lack of emphasis on analysis of the roles and functions of emotions in the development of anxiety and mood disorders is both notable and curious. Within a functionalist framework, emotions are viewed as organizing, adaptive, and having regulatory functions for internal dynamic processes and interpersonal interaction (Campos, Barrett, Lamb, Goldsmith, & Stenberg, 1983). No emotion is intrinsically more adaptive or dysfunctional than any other emotion. Emotions per se are not viewed as dysfunctional, but negative emotions often have maladaptive qualities of high intensity, long duration, and situational inappropriateness (Watson & Clarke, 1992). Among the prominent dysfunctions are disconnections between the experiential and expressive components of emotion (Plutchik, 1993), as well as regulatory problems (Cole, Michel, & Teti, 1994). Internalizing problems not only involve prolonged, intense expressions of anxiety and sadness but also efforts to control or suppress negative emotions. Young female adults asked to suppress negative emotion show greater physiological reactivity, which may be costly in the long term (Richards & Gross, 1999). There is a need for research on processes by which dysregulation and disconnection develop in childhood, and how they relate to psychopathology.

One of the earliest developmental tasks in childhood involves the ability to regulate behaviors and emotions in an appropriate manner. Most children show varying degrees of mastery of this process. They are able to cope with the challenges in their everyday lives in ways that allow them to become increasingly independent and socially skilled. A number of children show deficits in the first years of life, and may be diagnosed with regulatory disorders. Problems in these early years are amorphous and difficult to characterize, compared with later development, where there is increased differentiation and specificity. The ways in which very young children's fearfulness, anger, and failures to regulate emotional distress relate to internalizing (and comorbid externalizing) problems by late preschool and early elementary school remains unclear.

Negative emotions become more manageable with time for most children. Nervous systems mature, children come to understand the meaning, complexities, and consequences of their feelings, and they develop coping

strategies. Socialization also is thought to play a role, including the learning of display rules (i.e., what to conceal, what to reveal, what are the appropriate forms of expression). This literature, however, is based mainly on age cross-sectional comparisons of different age groups. We still know little about individual differences in regulatory processes, how at-risk samples compare with normative groups, and how this relates to the development of specific emotional and behavioral problems. We do know that regulatory problems continue throughout childhood, adolescence, and adulthood, the implications of which merit further attention.

Emotions and the development of psychopathology

Some emotion theorists have focused on functional continuities between emotions as episodic states and psychopathology (Izard, 1977; Malatesta & Wilson, 1988). The repetitive nature and the emotional salience of everyday social interactions become the basis for affective biases. These biases are thought to become the central organizing axes for personality over time. When they become consolidated through repetitions of discrete emotions, and organized into rigid patterns, this may lead to specific forms of psychopathology. Examples include anger biases or traits in antisocial personality, sadness in depression, and fear in some anxiety disorders. Models based on these views remain to be tested in longitudinal research, focusing both on child characteristics (e.g., stress reactivity seen in ANS and HPA arousal) and socialization experiences (e.g., how parents and peers respond to negative emotion displays) that shape particular affective biases implicated in particular disorders.

Advances in research on emotions and internalizing disorders will require expanded conceptions of the structure and organization of affects implicated in these problems. Both anxiety and depressive disorders clearly are characterized by more than one prevailing emotion. For example, while sadness is a central emotion–trait–bias linked to depression, other emotions are arguably as important. From a developmental perspective, early affective biases that may precede or be part of the depressive experience are likely also to involve worry and anxiety; guilt, shame, and self-reproach; lack of pleasure (anhedonia); suppression of anger and hostility; and empathic overarousal where the self becomes submerged in the problems of others. Recall that the moral emotions such as guilt were first implicated in adult depression by Freud, who emphasized childhood precursors of this disorder, seen in a harsh superego and internalization of criticism and blame. Different configurations of emotional profiles may be markers of different subtypes of anxiety and depression.

Comorbid anxiety and depression

Comorbidity rates for anxiety and depressive disorders can range as high as 70%, with rates from 20 through 50% most likely to be reported (see reviews by Angold, Costello, & Erkanli, 1999; Brady & Kendall, 1992). In adolescents, there is actually greater comorbidity between anxiety and depressive disorders than among anxiety disorders, and comorbidity is much more common than the "pure" types. Comorbidity rates are likely to be underestimated (e.g., major depression accompanied by subclinical anxiety would not qualify as comorbidity). Yet the co-occurring anxiety could have significant clinical implications later in development. Links between depression and anxiety have been construed in different ways: comorbidity may indicate the presence of a single underlying dimension; separate, distinct disorders; or conceptual and measurement problems signaling the inability of categorical systems to reliably characterize the relevant phenomena. There is now increased recognition of ways in which the study of comorbidity can inform our understanding of the development of the different forms of psychopathology (Angold et al., 1999).

Anxiety disorders in childhood and adolescence often precede and predict later depressive disorders. Initial evidence was based on retrospective accounts of lifetime histories obtained from diagnostic interviews. For exam-

ple, in one study of children with comorbid anxiety and depression, two thirds became anxious before they became depressed (Kovacs, Gatsonis, Paulauskas, & Richards, 1989). Several prospective, longitudinal studies have found that anxiety temporally precedes depression in children, adolescents, and even young adults (e.g., Breslau, Schultz, & Peterson, 1995; Cole, Peeke, Martin, Truglio, & Seroczynski, 1998; Lewinsohn, Gotlib, & Seeley, 1995). Also, prepubertal-onset anxiety disorder precedes later recurrent major depressive disorder across several generations of families at high risk for depression (Warner, Weissman, Mufson, & Wickramaratne, 1999).

A common theme surfaces across theories, regarding the causal role anxiety may play in the emergence of some forms of depression. The precipitating role of anxiety in the development of depression is consistent with an attachment perspective (Bowlby, 1960): the initial response to object loss consists of anxiety and agitation, depleting the organism and resulting in despair. Over time, the anxiety becomes intolerable and the organism gives up, resulting in depression. Cognitive theories postulate causal models in which anxiety has a direct influence on liability for major depression. In the learned helplessness model of depression, the initial response to an uncontrollable situation is to become highly anxious. One developmentally relevant model focuses on the role of rumination in the prediction of current and subsequent depression (Nolen–Hoeksema, 1998; Nolen–Hoeksema & Girgus, 1994). Rumination is the tendency to focus passively and repetitively on one's symptoms of depression without taking action to relieve them. It involves worry, perseveration, and even obsession about one's inner state. Anxiety and worry have childhood origins; hence, ruminative qualities may emerge at point in time prior to signs of depression. The ruminative process can feed upon itself so as to magnify the problems, overwhelm the child, and eventually create a state of depression. Unlike disruptive behavior problems, depression does not show a great deal of stability from childhood to adolescence. It may be a discontinuous phenomenon, reflecting the fact that depression is relatively rare in childhood. Or it may be that continuity is present but takes a less direct form (i.e., from early anxiety to later depression). Anxiety does show stability over time (e.g., first-grade children's self-reported anxiety symptoms had significant prognostic value in terms of their anxiety symptoms and adaptive functioning in fifth grade, based on an epidemiologically defined sample; Ialongo, Edelsohn, Werthamer–Larrson, Crockett, & Kellam, 1995).

Because anxiety involves dysregulation of limbic, vegetative, and autonomic systems, the heightened, sustained arousal eventually taxes these systems in ways that cause the organism to shut down and withdraw from environmental stimulation—in short, to become depressed. Not all forms of depression are preceded by anxiety, and anxiety does not invariably lead to depression. But the pattern occurs with sufficient regularity to postulate a subtype of depression that differs in its developmental history from depression that has not been preceded by anxiety. It is noteworthy, too, that the opposite pattern of depression leading to later anxiety has not emerged as a developmental phenomenon.

The question of whether anxiety and depressive disorders are meaningfully distinct or part of a common constellation has been considered extensively in work on "negative affectivity" or "internalizing syndrome" in both children and adults (e.g., Brady & Kendall, 1992; Clark & Watson, 1991; Kendler, Neale, Kessler, Heath, & Eaves, 1992; King, Ollendick, & Gullone, 1991). Overlapping and distinct features of anxiety and depression have been explained using a tripartite model (Clarke & Watson, 1991). The model postulates that anxiety and depression share a general distress factor referred to as "negative affectivity." Low levels of positive affectivity are relatively unique to depression, and somatic tension and arousal are relatively unique to anxiety. This three-factor model has been challenged by data suggesting other factor solutions (Burns & Eidelson, 1998) and variations in factor solutions for children as a function of age (Cole, Truglio, & Peeke, 1997). However, it provides an important starting point for further research on the phe-

nomenology of internalizing problems, by focusing on different affective cores that underlie different types and combinations of internalizing problems.

Anxiety and depression also have been associated with different forms of cognitive processing (see review by Mineka, Watson, & Clark, 1998). An attentional bias for threatening information is primarily associated with anxiety, memory biases are primarily associated with depression, and judgmental or interpretive biases are associated with both anxiety and depression. This is consistent with Oatley and Johnson–Laird's (1987) proposal that there may be unique modes of cognitive operation associated with different basic emotions. In this view, depression and anxiety evolved in response to different environmental circumstances. Anxiety, like fear, is associated with scanning the environment in anticipation of potential threat. Depression involves reflective consideration of events that have led to failure and loss. Research on childhood antecedents of different biases is warranted (Zahn–Waxler, 2000).

Comorbid internalizing and externalizing problems

The incidence of disruptive behavior disorders (conduct, oppositional, ADHD) is elevated in youth with either mood or anxiety disorders (see reviews by Compas & Hammen, 1994; Nottelmann & Jensen, 1995). There is also substantial comorbidity between internalizing and externalizing syndromes, seen in Achenbach scores in the .50s and .60s (e.g., Hinden, Compas, Howell, & Achenbach, 1997). The common and distinct features of internalizing and externalizing problems have been studied in a birth cohort at ages 18 and 21 years (Krueger, Caspi, Moffitt, & Silva, 1998). Latent structure and stability of 10 common DSM-III-R (APA, 1987) mental disorders were examined. A two-factor model in which some disorders reflected internalizing problems and others reflected externalizing problems provided the most optimal fit to the data. Individuals retained their relative positions to a significant degree, over the 3-year period. Even though internalizing and externalizing problems clearly emerge as separate factors, they are also significantly intercorrelated, again indicating their often comorbid nature.

There is growing evidence that diverse constellations of behavior may reflect diverse etiologies, having different correlates and developmental pathways. For example, Harrington, Fudge, Rutter, Pickles, and Hill (1990) found that children with depressive disorder were 4 times more likely to suffer from some depressive disorder in adulthood. However, the children with depression and conduct disorder had lower rates of depression in adulthood than did children with pure depression. The presence of anxiety in antisocial youth, assessed in terms of autonomic reactivity, also appears to reduce later criminal behavior (e.g., Raine, Venables, & Williams, 1995). Depression or anxiety may function as a protective factor, reducing conduct problems over time. Children with conduct disorder also have shown higher levels of cortisol when a comorbid anxiety disorder was present (McBurnett, Lahey, Frick, Risch, Loeber, Hart, Christ, & Hanson, 1991), providing corroborative neuroendocrinological evidence for the anxiety component. Other research has found the opposite pattern. For example, the link between aggression early and late in the school year in first-grade males was strengthened by the presence of comorbid anxious symptoms, rather than attenuated (Ialongo, Edelsohn, Werthamer–Larrson, Crockett, & Kellam, 1996). Considerable conceptual and empirical work is needed to unravel the complex and diverse ways in which having one type of problem influences the developmental course of another comorbid condition.

Calculations of comorbidity rates for internalizing and externalizing problems are based on the research sample as a whole. While subsamples can be identified based on different constellations of overlapping disorders, there have been few efforts to develop distinctive profiles based on these subgroups. Such an approach would permit greater in-depth analysis of possible different etiologies. Both categorical and dimensional approaches could be used in concert to identify and investigate subtypes of internalizing problems that be-

come lumped together in calculations of comorbidity. This would facilitate examination of the implications of symptoms versus a diagnosis of depression (Gotlib, Lewinsohn, & Seeley, 1995). This complementary approach could help to establish more veridical profiles for types of internalizing problems (Klimes–Dougan, Kendziora, Zahn–Waxler, Hastings, Putnam, Fox, Suomi, & Weissbrod, 1997). Detailed analysis of *different* constellations of symptoms within a diagnostic category could help further to refine the classification process.

A number of broad classes of dysfunction, expected to create a range of adverse environmental conditions for children, have been associated with both internalizing and externalizing problems (e.g., Gotlib & Avison, 1993)—for example, psychosocial stress, poverty, parental marital discord, parental psychopathology, maltreatment, and parental emotional unavailability. A concentration of multiple risk factors is typically found in more severe and varied problems in children (Sameroff, Seifer, & Bartko, 1997). Selection of well-defined samples will be important for identifying environmental factors specific to the development of internalizing, externalizing, and comorbid problems.

Depressive symptoms and antisocial behavior appear to share a common genetic diathesis. They may co-occur because of a common genetic liability that increases vulnerability to both types of problems (O'Conner, McGuire, Reiss, Hetherington, & Plomin, 1998). In this work, genetic influences were identified separately for depressive symptoms and antisocial behavior, and for their co-occurrence, in adolescents studied using a twin-sibling design. O'Conner et al. (1998) hypothesize that depressive and antisocial symptoms may be correlated because genes influence a neurotransmitter system associated with both behavioral syndromes. Such a view would be consistent with the demonstrated efficacy of some psychopharmacological agents in treating both internalizing and externalizing syndromes. The medications may help to regulate emotionality and, hence, act more generally upon the wide a range of negative emotions associated with problems (e.g., anger, fear, sorrow).

Future research would benefit from longitudinal research designs that also include variables based on direct assessments of biological (e.g., DNA, dopaminergic and serotonergic neurotransmitters) and environmental (e.g., specific types of childrearing and discipline practices) processes. The influence of these factors could then be estimated in models based on direct assessments of these factors rather than on inferences derived solely from the degree of genetic relatedness. With regard to environmental processes, other longitudinal research on the co-occurrence of adolescent depressive symptoms and conduct problems in adolescents has measured parenting effects directly (Ge et al., 1996). Different observed parenting behaviors predicted both the separate occurrence and the co-occurrence of these internalizing and externalizing problems at later time points, controlling on initial problems. Research that incorporates the strengths of both types of designs (i.e., genetically informed samples and actual measurement of environmental, and biological, variables) will be central to scientific advances in this area.

Finally, we consider the implications of comorbidity of emotions for understanding the phenomenology of internalizing problems. Basic or discrete negative emotions such as fear, sadness, and anger co-occur as blends in normal individuals (e.g., Watson & Clark, 1992) and in those with clinical conditions (Biederman, Farone, Mick, & Lelon, 1995; Kovacs, Feinberg, Crouse–Novak, Paulauskas, & Finkelstein, 1984). Terms such as "sullen," "moody," or "irritable" are used to describe emotional states that become more traitlike blends of different negative emotions seen in both internalizing and externalizing problems. Future research could focus on processes by which certain emotions within the comorbid constellation come to predominate (e.g., the child who initially is both angry and fearful, but who eventually is characterized primarily by fearfulness). Negative emotions thus could become differentially channeled (e.g., as one comes to predominate over time to influence the ultimate form of disorder).

Symptoms of irritability, in particular, characterize both internalizing and externalizing disorders. Features of major depression among clinically referred children and adolescents include a high frequency of dysphoria and irritable or angry mood (Biederman et al., 1995; Kovacs et al., 1984). Irritability in depressed children is commonly described by parents and teachers, and is often what leads to a diagnosis of depression (Poznanski, 1982). And it is not uncommon for anxious children to show heightened irritability. This further underscores the importance of research on the etiology of the dysregulated emotions central to internalizing disorders. As noted, children who meet criteria for major depressive disorder frequently meet criteria for ADHD, conduct disorder, and anxiety disorders (Biederman et al., 1995). These relationships would be consistent either with processes whereby depression reflects hostile impulses turned inward or antisocial, disruptive behavior reflects masked depression. Neither process is likely to be completely "successful," hence the manifest comorbidity. Affective disturbances associated with disruptive behavior disorders and depressive disorders thus are not as distinctly different as they might first appear to be (Cole & Zahn–Waxler, 1992). Careful analysis of patterns of emotion dysregulation associated with internalizing and externalizing problems might help to elucidate their common and separate pathways. This will require research designs that incorporate genetic, psychiatric, and developmental research designs. The resources and cooperation necessary make this a daunting task.

Gender

During childhood girls exhibit far fewer externalizing problems (and less overall psychopathology) than boys, with no clear sex differences evident in depressive and anxiety disorders. By adolescence, girls (but not boys) show a marked increase in anxiety and mood disorders and symptoms (Lewinson et al., 1993; Nolen–Hoeksema & Girgus, 1994). Female sex becomes the strongest risk factor for internalizing problems. Females are at least twice as likely (sometimes more) as males to become anxious and depressed, a pattern that continues throughout adulthood. Comorbidity of anxiety and depressive disorders is much more common in girls than boys (Lewinsohn, Rohde, & Seeley, 1995); moreover, having more than one anxiety disorder during childhood or adolescence is virtually exclusive to females (Lewinsohn, Zinbarg, Seeley, Lewinsohn, & Sack, 1997).

Until recently, most of the research on childhood disorders has focused on externalizing problems known to be more prevalent in males throughout the life span, and beginning in early childhood. Because the nature of problems more common to girls are also less disruptive to others and surface later, research on etiology and development of internalizing problems has lagged. The presence of this very strong sex difference, suggests the need to better understand both constitutional and environmental factors that place females at such high risk for anxiety and mood disorders. Most explanations have focused on biological processes and experiential factors associated with adolescence and pubertal development (see Zahn–Waxler, 2000). Here we emphasize the significance of processes set in motion much earlier in development that are more difficult to detect.

Dispositional characteristics. Early constitutional advantages for girls may provide a number of qualities that decrease risk for early behavior problems. Young girls, on average, show greater language skills, more rapid physical maturation, and advanced regulatory capabilities. This may contribute to their known advantage in terms of frustration tolerance, ego and impulse control, internalized standards of conduct, empathic sensitivity and ability to interpret others' emotions, social maturity, and responsible interpersonal behavior (Zahn–Waxler, 2000). Why then do girls, who appear resilient and relatively impervious to childhood mental disorders, later show so many internalizing problems? Continuities may present but in a more subtle form. Qualities that protect against antisocial behavior could also create risk for internalizing problems.

We indicated earlier that anxiety could often be a precursor of depression, beginning in childhood. Girls are more prone to early fearfulness and worry than boys (Silverman, LaGreca, & Wasserstein, 1995), even normatively, and they are somewhat more likely to be shy and inhibited. Dienstbier (1984) has proposed that different temperaments might lead to different emotion-attributional styles and levels of guilt. Proneness to emotional tension should result in intense discomfort and distress following transgression. When distress is internal, the child is more likely to experience the links between tension and transgression, and come to experience anticipatory anxiety. Temperamentally anxious children may develop "affective maps" of their experiences where threat or stress-related information becomes particularly salient (Derryberry & Reed, 1994). Similarly, Damasio, Tranel, and Damasio (1991) refer to an automatic guiding system that activates "somatic markers" associated with one's past experience. These "maps" or "somatic markers" may facilitate the early, rapid development of mechanisms related to conscience, such as guilt and restraint.

Because anxiety is more common in girls than boys in childhood, and because it is associated with autonomic arousal (elevated heart rate and electrodermal activity), girls may become more physiologically aroused than boys under particular conditions of interpersonal conflict and distress (Zahn–Waxler, Cole, Welsh, & Fox, 1995). Girls may more readily develop somatic markers that facilitate internalization of norms and standards of conduct. This proneness to experience internalized distress also may lead to early rumination, creating a constellation of factors that could heighten risk for developing anxiety and mood disorders. Although this is a viable hypothesis, it remains to be tested. Because females are known to be more emotionally responsive than males to the problems of *others,* a wider range of interpersonal contexts may arouse them. Their automated, internal reactions that also include ruminative scripts may then contribute to entrenched, generalized patterns that are later seen as anxiety and depression. Adolescent and adult females ruminate more than males, and rumination predicts future depression (Nolen–Hoeksema & Girgus, 1994). We propose that risk for some forms of later depression begin in childhood, more often for girls because of their heightened reactivity and ruminative styles.

Socialization experiences. The socialization data reviewed here is quite recent in origin and often based on middle-class samples. Differential treatment of boys and girls may create conditions that predispose females more often than males to anxiety and depression. Girls are more likely than boys to be socialized in ways that interfere with self-actualization (i.e., to be dependent, compliant, and unassertive; Hops, 1995). A number of different experiences are likely to create greater risk for females than males (reviewed in Zahn–Waxler, 2000). On average, sons are more valued than daughters. Even when there is serious conflict between parents, these couples show more mutual investment in their sons than daughters. Socialization practices (particularly in fathers) that encourage sex-stereotyped activities could lead to greater submissiveness and dependency on the opinions of others in girls. Parents more often discourage exploration of the physical environment in girls than boys. And they more often place pressure on girls to anticipate the consequences of their negative acts. Girls are more likely to be reinforced for shyness and dependency, their successes more often overlooked, and their physical aggression deemed more serious than for boys.

Parents often have higher expectations for mature interpersonal behavior in girls, are less tolerant of their anger and misbehavior, and more likely to override or negate assertive behaviors in girls than boys. Many of the socialization practices directed more often toward girls contain messages that reflect pressures to be prosocial, suppress anger, and curtail antisocial behavior. Suppression of anger, assertion, and other forms of self-expression may heighten internalized distress. Over time this may result in the indecision, self-criticism, self-blame, and low self-esteem that become part of the phenomenology of the depressive experience. This is one hypothetical example

of how disconnection between experience and expression of emotions (here, anger and hostility) could contribute to the development of internalizing problems. It is noteworthy that females and males do not appear to differ in their experiencing of anger and hostility but rather in their expression and acting out on these feelings.

Culture and the development of internalizing problems

Almost without exception, research considered here is based on samples of children and adolescents from the United States. It should not be assumed that etiology, prevalence, and phenomenology are consistent across cultures. Cultures vary both on some biological dimensions and in the kinds of behaviors they value, forbid, and condone. Both may shape the degree and direction of problems in childhood. There are differences in cultural norms regarding displays and regulation of emotions, self-expression, and the role of the self in relation to others (Markus & Kitayama, 1991; Han, Leichtman, & Wang, 1998). These variations, as well as different culturally defined ways of coping with distress, also may help to determine whether internalizing problems become predominant (McCarty, Weisz, Wanitromanee, Eastman, Suwanlert, Chaiyasit, & Bond, 1999). This is a little-researched area, one that is ripe for inquiry both into the universality and cultural specificity of processes that underlie anxiety and depression.

Cross-cultural differences exist in the prevalence and patterning of problems. Internalizing problems in children occur more often in some countries (e.g., Greece, Thailand, Puerto Rico), whereas externalizing problems are more common in other countries (e.g., United States, Germany, Sweden; Crijnin, Achenbach, & Verhulst, 1997; MacDonald, Tsiantis, Achenbach, Motti–Stefanidi, & Richardson, 1994). While comorbidity of internalizing and externalizing problems is common across countries and cultures, the nature of these relationships will vary depending upon which type of problem is more prevalent in the different cultures.

Girls show more internalizing problems than boys across cultures (Crijnin et al., 1997). While adult depressive disorders and syndromes are more prevalent in females than males in virtually all cultures, the ratios differ markedly (Kleinman & Cohen, 1997). In China, for example, female-to-male rates of depression and completed suicides are strikingly disproportionate, with suicide described as a "normative" response for poverty-stricken rural females. This indicates the need to examine cultural characteristics that lead women from some parts of the world, such as China, to take their lives more often than men, whereas in other places such as the United States the reverse is true.

Reflections and Projections

It has been illuminating to chart the remarkable progress made in our understanding and treatment of early anxiety and depression over a relatively short period of time. Until quite recently these disorders were not thought to be present in children and adolescents. Since then, several internalizing disorders have been identified, assessed, classified, and treated with a variety of psychotherapeutic and pharmacological approaches. A number of biological and environmental factors implicated in the expression of these disorders have been studied, as well as risk and protective factors that heighten or diminish the probability that such problems will surface. We have seen an increase in multivariate, longitudinal designs used to more fully explicate the etiology of the internalizing symptoms and disorders that so commonly afflict children and adolescents. Considerable attention has been devoted to the development of methods to assess symptomatology and the factors implicated in their expression. But it is just a beginning. As a field we are still in a very early developmental period with regard to our theoretical and applied knowledge base. While transactional, developmental research is commonly recommended, we have not yet fully incorporated this perspective into our study designs. Moreover, we are still some distance from ascertaining the most salient constructs and, in turn, having the requisite armamentarium of

valid measures needed to test transactional models.

The fluid nature of many affective and behavioral symptoms in children and adolescents continues to raise questions about the ultimate forms disorders will take over the course of time. There are indications of progression from comorbidity to specificity, where comorbidity is seen as an earlier developmental phenomenon (Nottlemann & Jensen, 1995). Initially, psychopathology is amorphous in form of expression, becoming more differentiated and clearly defined as personalities begin to crystallize and the central nervous system reaches maturity, especially after puberty and midadolescence. For example, while current lifetime histories of depression in adolescents are highly comorbid with other disorders, the rates are substantially lower in adults (Rohde, Lewinson, & Seeley, 1991). Adolescent-onset depression may represent a more serious form of the disorder. Alternatively, it may initially be more undifferentiated, with greater specificity developing over time. Longitudinal research would help to address questions about determinants of these different developmental pathways.

The literature on resilience is beginning to identify factors associated with a good outcome despite being reared under conditions of risk. We know relatively little about what factors are protective with regard to internalizing disorders as compared with other types of psychopathology. Many definitions of resilience center on achievement, social competence, or finely honed interpersonal skills (Beardslee & Podorefsky, 1988; Luthar & Zigler, 1991). Internalizing problems often coexist with competencies, and "high" functioning also can reflect aspects of disorder (Luthar & Zigler, 1991). Consequently, additional methods are needed to assess psychological health as a protective factor.

The scope of this article did not include discussion of assessment and treatment, topics that are less often considered within a developmental psychopathology framework. Scientific knowledge of the origins, development, and phenomenology of internalizing problems is important in its own right. But ultimately this work is also in the service of valid assessment and successful treatment of these forms of human suffering. To do so requires a profound understanding of the nature of children and adolescents across the time course. Symptoms of internalizing problems are known to vary across different periods of development. Historically, the use of top-down models (i.e., studying anxiety and depression in adults and then working backward to understand similar problems in children and adolescents) created problems that continue to the present day. In psychiatric research, participants who vary widely in age (e.g., from 6 to 16 years) often are considered as one group for analytic purposes. This can limit generalizations and, hence, the utility of the research findings. It is difficult to develop valid assessment systems and programs for effective prevention, intervention, and treatment in a nondevelopmental context. Psychotherapeutic and pharmacological approaches to treatment also originated from top-down models. While many treatment approaches are now highly sensitive to developmental level, some are not. Public attention recently has been drawn to giving children in the first years of life the medications that had been developed and approved for treatment of depressive disorders in adults and attentional problems in school-age children. While developmentally sensitive research has become more common, it often follows rather than precedes treatment decisions.

Psychologists have in-depth knowledge of developmental processes. They also have methods that, if used in conjunction with those developed within a psychiatric tradition, would help to establish an integrated perception of the essential need for both categorical and dimensional approaches to assess and classify internalizing problems. It is important that both psychiatry and psychology deal with a full range of symptomatology. There is a reluctance to wrestle with questions about whether and when a quantitative difference becomes a qualitative difference as well, or about dynamic processes that move the child from normative, to subclinical, to clinical symptoms (or in the other direction). Psychologists who rely solely on quantitative, dimensional measures often do not convey the degree of psychopathology (or lack thereof) that is reflected in their findings. Psychiatrists who

rely solely on qualitative, categorical measures based on diagnostic information often do not have sufficient appreciation of the significance of subthreshold symptoms. Collaborative work would elucidate the wide range of significant symptoms.

A related issue concerns the models and statistical approaches utilized in different disciplines. It has been common in developmental research for psychologists to test explanatory models using a variable-centered approach, attempting to understand developmental trajectories through analyses of the group as a whole. Such a process does not lend itself well to understanding of subtypes of anxiety and depression either in "pure" or comorbid form. It is easy for the individual to become lost in what has come to be viewed as a "one-size-fits-all" model. A person-oriented approach that focuses on attempting to define different clusters of individuals is also essential. The contributions of developmental psychology and psychopathology will become more salient to child and adolescent psychiatry (and vice versa) when the disciplines are able to interface more on problems that would be expected to be of common interest.

A developmental psychopathology framework has much to offer for understanding the etiology and development of internalizing problems. There is evidence from prospective research of continuity between adolescent and adult depression (Rao, Hammen, & Daley, 1999). Longitudinal research on links between childhood depression and adolescent or adult depression has not been conducted. Not all depression subsequent to childhood would be expected to have very early origins, but depressed adults often report symptoms dating back to childhood. Clinical depression may emanate eventually from subsyndromal depressive styles that in childhood may appear as temperamental qualities or personality characteristics. In contrast to the stability of externalizing problems across time, there is little empirical evidence of continuity of depression. Lack of continuity may reflect the lack of developmental research on internalizing problems, along with the fact that prodromal signs may be less obvious. Even less is known about the history of anxiety syndromes across these developmental periods, and probably for the same reasons. Even with the increased awareness of internalizing disorders in children, only a minority receives treatment. When children evidence more "pure" forms of anxiety or depression, symptoms may go unnoticed as they suffer in silence. When they are accompanying externalizing problems, the disruptive behaviors may actively divert attention away from the anxiety or depression. Some early expressions of anxiety could even be seen as positive as they may motivate children to behave in ways valued by adults.

In summary, anxiety and depressive disorders are common in childhood and adolescence. These disorders are still understudied relative to other disorders, highlighting the need to focus on biological and psychosocial underpinnings of these illnesses. Longitudinal studies of the course and outcome of anxiety and depressive disorders in childhood and adolescence are critical—not only to further our knowledge of the impact of these disorders during the earlier stages of human development but also to increase our knowledge of their emergence in adulthood. The progress that has been made and the foundations that have already been established for achieving future goals should encourage us. We also should be prepared for sweeping changes in how internalizing problems are viewed and studied in this century.

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