CHAPTER 19

Internalizing Problems During Adolescence

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The development of psychopathology during adolescence has been perhaps the single most studied area in the field of adolescence (Steinberg & Morris, 2001). Extensive focus has been on depressive disorders, conduct disorder, and related subclinical problems and symptoms; multiple behaviors and problems fall generally under the categories of internalizing and externalizing behaviors. Internalizing problems are generally considered to be the subgroup of psychopathology that involve disturbances in emotion or mood, whereas externalizing problems have tended to refer to dysregulations in behavior. However, the affect versus behavior distinction between internalizing and externalizing is not clear cut, especially when considering such emotional experiences as anxiety and anger, which are consistently considered when discussing both internalizing and externalizing problems. Regardless, the general identification of internalizing problems as focused on emotional components such as sadness, guilt, worry, and the like is consistent across several definitions. More specifically, depression and anxiety disorders and the subclinical problems in these areas typically comprise discussions of internalizing problems and disorders (see Kovacs and Devlin, 1998; and Zahn-Waxler, Klimes-Dougan, and Slattery, 2000, for recent reviews of internalizing problems and disorders in childhood and adolescence, and chapter 20, this volume, for a discussion of externalizing problems during adolescence).

Interestingly, the concept of internalization has been investigated as a core task of early childhood (Kochanska, 1993). In this case, internalization is the incorporation into the self of guiding principles (as values or patterns of culture) through learning or socialization. Stemming from psychoanalytic and social learning theories, internalization is commonly thought of in the context of the regulation of moral behavior or the development of conscience (Kochanska, 1993). Kochanska suggests that internalization comes about in early childhood through parent–child communication with a focus on developing feelings of empathy, guilt, and prosocial affect such as concern for others, and cause and effect of behaviors (e.g., understanding how one's behavior makes others feel). In psychoanalytic theory, anxiety and guilt are the internalized emotions that replace parental control of behavior (Muuss, 1996). In the present usage, *internalizing* refers to problems or disorders of emotion or mood; the dysregulation of emotion might be thought of as overinternalization of certain emotions such as guilt, anxiety, or overinvolvement in the emotions of significant others (e.g., the inability to distinguish one's own responsibility for another's emotional state from non-self causes of distress in others).

Mood variability or emotionality has historically been viewed as a defining characteristic of adolescence (e.g., Hall, 1904; Muuss, 1996, for a review). However, the literature on universal or typical changes in mood or emotion during adolescence has been limited (with a few exceptions such as Larson, Csikszentmihalyi, & Graef, 1980) until

very recently (e.g., Weinstein, Mermelstein, Hankin, Hedeker, & Flay, 2007). In contrast, a vast literature exists on depressive disorders, subclinical problems, and symptoms during adolescence. As such, the goal of this chapter is not to provide a comprehensive review of that literature or even of the etiology or epidemiology of depression (see Costello, Foley, & Angold, 2006; Hammen, & Rudolph, 2003, for recent reviews). Instead, this chapter provides an overview of internalizing symptoms, problems, and disorders and their etiology throughout the adolescent period (approximately the second decade of life). The chapter focuses on models and factors that may be particularly salient to understanding which types of problems become prevalent during adolescence and on two specific questions that have long interested scholars of internalizing problems: "Why adolescence?" and "Why more girls?"

TRENDS IN STUDYING ADOLESCENCE AND INTERNALIZING

A surge of interest in adolescent development in the 1980s resulted in several longitudinal projects that spanned the middle school and sometimes high school or young adult periods. These studies grew from an interest in understanding the role of transitions on the course of development (e.g., Elder, 1985) as well as specific interest in the combined or transactional influences of biological, social, and psychological processes in determining pathways of adjustment (e.g., Petersen, 1984; Simmons & Blyth, 1987). It could be argued that these initial studies were undertaken to understand the typical or "normal" problems of adolescence with perhaps greater attention to variations in adjustment (e.g., moodiness, parent–adolescent conflict, academic achievement) rather than disorder per se. Such projects reported on the range and diversity of normative adolescent development but also delineated the nature of problems experienced by adolescents. A subsequent surge in the 1990s of community and epidemiology based studies attempted to determine the severity of these problems by focusing on the assessment and experience of disorder.

Most developmental scientists agree that behavior and adjustment during adolescent transitions are dependent on the nature of the transitions and how they are navigated, as well as on developmental experiences prior to making the transitions (Graber & Brooks-Gunn, 1996; Rutter, 1994). In the study of internalizing problems, a few studies have examined childhood behaviors in connection with subsequent adjustment or behavior in adolescence, sometimes even accounting for a transitional experience (e.g., Caspi & Moffitt, 1991). More often, though, studies of childhood experiences conclude with suggestions that subsequent influences on adolescent internalizing behaviors would be expected, and studies of adolescence note that preexisting patterns, behaviors, and experiences were undoubtedly factors in who developed problems during adolescence. Notably, some comprehensive longitudinal studies have been initiated that allow for the examination of continuity and change from infancy or early childhood into adolescence (e.g., NICHD Study of Early Child Care; Belsky et al., 2007) and beyond (e.g., the Dunedin Longitudinal Study; Krueger, Caspi, Moffitt, & Silva, 1998).

PATHWAYS FOR CONTINUITY AND CHANGE

Differentiating who will and who will not develop serious problems, and differentiating normative experience from atypical, are central themes of the field of developmental psychopathology. In particular, under this rubric, describing pathways both to psychopathology and to normative, healthy, or competent development are essential for understanding etiology and treatment of problems (e.g., Cicchetti & Cohen, 1995; Masten & Curtis, 2000). Masten and her colleagues have defined competence as ". . . adaptational success in the developmental tasks expected of individuals of a given

age in a particular cultural and historical context" (Masten & Curtis, 2000, p. 533). As such, the definition of competence is not static and not singular; that is, individuals may meet developmental challenges in some areas (e.g., academic achievement) but fail to demonstrate adaptational success in other domains (e.g., behavior or emotion regulation). When considering adaptive versus maladaptive development, competence has often been assessed as the absence of problems or significant deficits in an area. For mood disorders, Masten and Curtis (2000) note that the links between competence and pathology is complex and that direction of effects are difficult to determine. That is, individuals who do not successfully meet developmental challenges may experience increased symptoms as a result of this failure; or, in contrast, symptoms of disorders may interfere with successful adaptation. Of course, when viewed over time and across developmental challenges, both experiences are likely to occur.

As noted by others, in this volume and elsewhere, nearly all youth experience challenges during the transition into adolescence, and often throughout the adolescent decade. As such, all youth should have dramatic shifts in behavior and potentially damaging effects from the experience of simultaneous and cumulative challenges. Alternatively, these changes are endemic to the developmental process and all youth should have the appropriate resources to adapt to such normative challenges and sustain adaptive behavioral patterns. In reality, as has been repeated in nearly all discussions of continuity and change (e.g., Kagan, 1980; Rutter, 1994), some youth demonstrate continuity of either successful adaptation or psychopathology, whereas others evince change in the ability to meet developmental challenges.

The development of internalizing problems or disorders during adolescence is in many cases not about substantial behavioral change or new problems arising, but rather is dependent on individual characteristics that existed well before adolescence (Bandura, 1964). For some youth, the challenges of adolescence exacerbate or accentuate these characteristics, resulting in decreased functioning and serious dysregulation in mood. These preexisting characteristics, or vulnerabilities, are often traitlike and develop over the course of childhood and adolescence (Ingram & Luxton, 2005). In turn, for some youth, internalizing problems may emerge at this time of development in connection with more concurrent or recent experiences. Therefore, the meaningful developmental questions regarding internalizing problems in adolescence must focus on individual differences in development. The important issue regarding continuity and change is not whether the normative transitions of adolescence result in difficulties but rather *why* they result in difficulties for certain individuals but not others. However, prior to a discussion of why some individuals experience internalizing problems and others do not, it is useful to consider the rates of these problems during adolescence.

EPIDEMIOLOGY OF ANXIETY AND DEPRESSION IN ADOLESCENCE

Although internalizing problems or symptoms may broadly encompass disturbance in emotion or mood, discussions of internalizing problems are usually limited to the investigation of depression or anxiety, typically as distinct phenomena. Compas and colleagues (Compas, Ey, & Grant, 1993; Compas & Oppedisano, 2000; Petersen et al., 1993) have developed a framework for viewing depressive disorders and subclinical symptoms that is applicable to internalizing problems more generally. Within this framework, internalizing symptomatology can be classified into three levels or types: *disorders*, as determined by diagnostic criteria; *syndromes* or subclinical problems; and internalizing *moods* or dysregulated emotion or moods. In contrast, rather than clear qualitative distinctions between disorder and symptoms, others argue that depression exists on a continuum (i.e., individuals are more or less

depressed; Hankin & Abela, 2005). Despite support for the latter perspective, many researchers continue to investigate disorder, subclinical symptoms, and mood separately.

Zahn-Waxler and colleagues (2000) note that theories and research on anxiety and depression in childhood and adolescence often have been separate lines of investigation. At the same time, as will be evident in the following discussion and in recent investigations (Compas & Oppedisano, 2000; Krueger et al., 1998), the extent to which anxiety and depression are distinct experientially or in the course of development is debatable. A review of prevalence rates in childhood versus adolescence for anxiety and depressive disorders sheds light on connections between the two domains. That is, whereas rates of some anxiety disorders clearly increase from childhood to adolescence, others are confined almost exclusively to early childhood or may be present at any point in development over the life span. In contrast, rates of depression are low in childhood and increase dramatically during adolescence. Despite differences in overall developmental trends, anxiety and depression share symptoms and have substantial co-occurrence or comorbidity. (*Comorbidity* refers to the occurrence of a second disorder in an individual with an existing disorder.) Hence, some joint processes of etiology are likely among internalizing problems and disorders.

Prevalence Rates for Internalizing Disorders

Detailed information on symptoms and criteria for diagnosis of disorders are found in the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition, Text Revision (DSM-IV-TR; American Psychiatric Association, 2000). Disorders must include significant impairment in daily functioning along with requisite symptom severity and duration in order for diagnostic criteria to be met. Criteria for impairment often have been more subjective and drawn from psychiatric interview. At the same time, these criteria are relatively straightforward for adults (e.g., disruption in work, home life, interpersonal relationships). The challenge for practitioners or researchers conducting studies of disorder has been the identification of developmentally salient criteria for impairment for disorders in childhood and adolescence (Masten & Curtis, 2000). Difficulty in assessing severity or impairment may lead to an over- or underdiagnosis of disorder, ultimately leading to a misrepresentation of rates of incidence.

Anxiety Disorders

The DSM-IV-TR identifies six main subcategories of anxiety disorders that are applicable to children and adolescents: separation anxiety disorder, generalized anxiety disorder (GAD), obsessive-compulsive disorder, posttraumatic and acute stress disorder (PTSD), and specific phobias. Recent studies, both clinical and epidemiological, show that some anxiety disorders are more likely to emerge in childhood (separation anxiety disorder and specific phobias), whereas others usually begin in adolescence (social phobia and panic disorder; Costello, Foley, & Angold, 2006). Across investigations and collapsing across anxiety disorders, Costello, Egger, and Angold (2004) reported that prevalence rates for any anxiety disorder in children and adolescents ranged from 2.2% to 27%. However, prevalence rates varied dramatically by time criteria. As expected, studies with short assessment intervals and a single data wave had the lowest prevalence and studies using a lifetime criterion produced the highest rates. For example, studies with 3-month assessment periods reported a range of 2.2% to 8.6% prevalence; studies with a 6-month period reported a range of 5.5%–8.6%; studies with a 12-month period reported 8.6%–20.9%; studies assessing lifetime prevalence up to age 19 reported a range of 8.3%–27.0% (Costello et al., 2004). Reports suggest a slight trend for rates to increase with age (Costello & Angold, 1995), but such data must be interpreted with caution as it is based on "any anxiety disorder." Disparities

in prevalence rates across studies are in part accounted for by whether or not the diagnostic criteria used included assessment of functional impairment. When impairment is considered, rates of anxiety disorder decrease (Vasey & Ollendick, 2000; Zahn-Waxler et al., 2000); more children and adolescents report the requisite symptoms of the disorder, but a smaller number indicate significant impairments in functioning along with these disorders.

In addition, girls have higher rates of several of the anxiety disorders with the possible exception of PTSD (Costello et al., 2006; Vasey & Ollendick, 2000). The gender difference in rates varies by type of anxiety disorder and for some anxiety disorders data are limited on whether gender differences are consistently demonstrated. For example, separation anxiety, which tends to be found in young children, demonstrates a 3-to-1, girls to boys, gender difference in rates. In contrast, GAD may have equal prevalence rates by gender or higher rates for boys than girls in childhood, but among adolescents it is more prevalent in girls (Bowen, Offord, & Boyle, 1990; McGee et al., 1990). Criteria for GAD are shown in Table 19.1.

Depressive Disorder

Most discussions of mood disorders during adolescence focus on major depressive disorder (MDD), as this is the most commonly diagnosed mood disorder in childhood and adolescence. An overview of the criteria for a major depressive episode is listed in Table 19.1. A depressive episode is characterized by feelings of depression, sadness, and the like, or a loss of pleasure for a period of 2 weeks or more, coupled with a minimum number and frequency of the other symptoms listed. Typically, prevalence rates of MDD have ranged from 0.4%–8.3% among adolescents (Birmaher et al., 1996). Estimates of lifetime prevalence for MDD among children and adolescents range from 4% to 25% (Kessler, Avenevoli, & Merikangas, 2001)

TABLE 19.1 Selected *DSM-IV-TR* Criteria for General Anxiety Disorder and Major Depressive Episode[a]

Symptoms and Criteria for Generalized Anxiety Disorder	
A	Excessive anxiety or worry on most days for 6 months about a number of events or activities
B	Difficulty controlling the worry
C	Anxiety and worry are associated with three or more of the following symptoms: (1) Restlessness (2) Easily fatigued (3) Difficulty concentrating (4) Irritability (5) Muscle tension (6) Sleep disturbance
Symptoms and Criteria for a Major Depressive Episode	
A	Depressed mood or loss of interest for a 2-week period (or irritability among children and adolescents), plus
B	Four or more of the following symptoms in the same 2-week period: (1) Weight loss or weight gain (2) Insomnia or hypersomnia (3) Being restless or being slow (psychomotor agitation or retardation) (4) Fatigue or loss of energy (5) Feelings of worthlessness or inappropriate guilt (6) Inability to concentrate (7) Recurrent thoughts of death or suicide ideations or plans
Additional Criteria for both GAD and MDD	
A	Symptoms result in significant impairment in social and occupational functioning
B	Symptoms are not due to physical illness or drug use

[a]Criteria are adapted from *DSM-IV-TR* (APA, 2000).

but have been found to range from 15% to 20% for adolescents specifically (Birmaher et al., 1996; Lewinsohn & Essau, 2002). In comparison, prevalence of MDD in school age children is 1.5%–2.5%. Not only do rates of MDD increase dramatically from childhood to adolescence, but MDD is the most commonly occurring disorder among adolescents. As there are now several longitudinal studies of depression that have followed samples across adolescence into adulthood (e.g., the Oregon Adolescent Depression Project [OADP], the Great Smoky Mountain Study [GSM], the Dunedin Multidisciplinary Health and Development Study), detailed estimates of lifetime prevalence, 1-year incidence, and rates of recurrence are available across studies.

DSM-IV TR diagnostic criteria for MDD in childhood and adolescence are similar to criteria in adulthood, with the primary exception being that, among children and adolescents, the DSM-IV-TR allows mood disturbance to manifest as irritability as well as sadness. Although diagnostic criteria may be equally applicable for MDD across age groups, some variations in symptoms experienced and sequelae of disorder may vary between depressed children and adolescents. Specifically, Yorbik and colleagues (2004) found that depressed adolescents had significantly more negative cognitions (hopelessness, helplessness, pessimism, and discouragement), fatigue, hypersomnia, weight loss, and suicidal ideations and acts compared to depressed children. In comparison to adults, adolescents often experience substantial comorbidity of MDD with other disorders (Rohde, Lewinsohn, & Seeley, 1991). Despite these differences in manifestation across the life span, MDD appears to be the same core disorder regardless of the age at which it is experienced.

As noted, rates of MDD are fairly low in childhood and begin to rise during early adolescence. In the OADP, the mean age of onset was 14.9 (Lewinsohn, Rohde, & Seeley, 1998). This age is consistent with other community-based studies, although studies of clinical samples tend to report earlier ages of onset for first depressive episode (e.g., 11 years of age in Kovacs, Obrosky, Gatsonis, & Richards, 1997).

Studies of variations in rates by subgroups of the population have focused predominantly on gender. Whereas most studies find no gender differences in rates of MDD in childhood, by age 15, the gender difference in MDD is at the adult rate of about 2 to 1, girls to boys. Much less attention has been paid to sociodemographic and cultural variations in rates of disorders in childhood and adolescence. Sampling strategies have frequently not allowed for disentangling racial or ethnic variations from those variations due to poverty or other demographic factors (e.g., rural versus urban environments). In the GSM, White adolescents (ages 9–17) had higher rates of MDD than Black adolescents (Costello, Keeler, & Angold, 2001). Moreover, poverty was predictive of disorder among White but not Black youth.

Despite differences in rates of internalizing disorders as a function of gender, race, and socioeconomic status (SES), depression is clearly a significant concern when discussing the experience of internalizing disorders during adolescence across all groups; rates may also increase for anxiety, or at least for GAD, during this time period. At the same time, both types of disorders appear to be more common among girls than boys by mid-adolescence.

Pediatric Bipolar Disorder (PBPD)

Until recently, studies of depression and reviews of this literature have not included bipolar disorder. Bipolar disorder (previously referred to as manic depression) is characterized by dramatic mood swings from very high (i.e., mania) to very low (i.e., depression), with normal mood in between these episodes or cycles. Initial studies suggested that bipolar disorder did not emerge until late adolescence or adulthood and hence was less relevant to discussions of depression in adolescents (e.g., Birmaher et al., 1996). Recent epidemiological studies estimate that less than 1% of children and adolescents have PBPD (Kessler et al., 2001). In general, there is consensus that cases of bipolar disorder are evident in adolescents and children. Yet,

controversy still exists over developmentally appropriate diagnostic criteria.

Very rarely do adolescents manifest bipolar disorder according to DSM-IV-TR criteria established for diagnosis in adults. In particular, adolescents frequently do not display distinct manic episodes or periods of relatively good functioning between episodes. Rather, adolescents generally exhibit chronic or ultradian cycles of mood shifts (cycling between manic and depressive states within a 24-hour period) that are frequently accompanied by irritability, rage, and aggression. In fact, symptoms of PBPD in adolescence, such as hyperactivity, distractibility, racing thoughts, pressure to talk, and impulsivity, overlap with those associated with other disorders (e.g., attention-deficit/hyperactivity disorder [ADHD]), suggesting that the appearance of PBPD in childhood or adolescence may be an instance of comorbidity between depression and other disorders (Hammen & Rudolph, 2003). Recently, several key symptoms of mania have been identified that more concretely distinguish manic episodes or chronic mania in childhood from symptoms of other disorders, in particular, ADHD; these symptoms include elation, grandiosity, flight of ideas/racing thoughts, decreased need for sleep, and hypersexuality (Geller, Zimmerman, & Williams, 2002). Presence of these five symptoms aids in confirming PBPD as a distinct diagnosis rather than simply a manifestation of comorbid childhood depression and ADHD. Although there is increasing evidence supporting the notion of PBPD, little evidence exists to suggest that pediatric PBPD is associated with the occurrence of adult bipolar disorder. Moreover, insufficient information on the etiology of PBPD makes it difficult to consider commonalities with or distinct pathways from MDD. Future prospective studies are necessary to better understand the prognosis of adolescents diagnosed with PBPD.

Subclinical Problems, Syndrome, or Symptoms

In contrast to disorders, depressed or anxious mood are reports of emotional states that are not assessed in terms of their duration or in connection with other symptoms. As such, in studies of adolescent moods or symptoms, rates of depressed mood have varied dramatically; for example, some reports indicated as many as 40% of the sample experienced depressed mood (Compas et al., 1993; Petersen et al., 1993). Similar compilation of rates of anxious mood in different age ranges or across studies has not been made.

Measures of depressive symptoms typically report age and gender differences in elevated symptoms that parallel differences found for disorders. In a meta-analysis of the Children's Depression Inventory (CDI), Twenge and Nolen-Hoeksema (2002) found no gender difference in scores during childhood but a significant gender difference beginning at age 13, with higher scores among girls. Another study examining the rates of depressive symptoms in 8- to 17-year-olds found a small but highly significant interaction between age and gender such that boys and girls reported similar levels of depressive symptoms prior to age 12; but after age 12, scores increased among adolescent girls, whereas boys' scores fell slightly from childhood to adolescence (Angold, Erkanli, Silberg, Eaves, & Costello, 2002). In addition, in their meta-analysis, Twenge and Nolen-Hoeksema (2002) found no effects of sociodemographic status on depressive symptoms when analyzing data across studies, although this information was not available in all reports. Also, no differences in mean scores were found between White and Black children and adolescents. However, Hispanic children were found to have significantly higher scores than other children.

Syndromal classifications are based on endorsement of a constellation of symptoms that co-occur in a statistically consistent manner. Achenbach (e.g., 1993) has derived a syndrome that taps anxiety and depression and distinguishes referred from nonreferred adolescents across multiple cultures and nations (Ivanova et al., 2007). In this approach, about 5%–6% of adolescents evince

anxious–depressive syndrome as determined by scores above/below a predetermined cut point (Compas et al., 1993; Petersen et al., 1993). The assessment of syndrome is based on statistical associations among a checklist of symptoms as rated by several reporters (i.e., parent, teacher, self). In this approach, separate distinct syndromes for depression and anxiety are not found; rather, these symptoms consistently co-occur, a point that is salient to the next section.

The syndromal, category as defined by Achenbach, is one approach to defining a subclinical internalizing problem based on a specific measure of symptoms. Several other measures have been developed to assess depressive and anxiety symptoms and problems. Many have established cutoff scores for identifying potential disorders. Such measures are useful for comparing the experience of symptoms among individuals in the general population of youth but also as screening instruments for identifying individuals who may have more serious disorders. In this case, individuals who exceed the determined cutoff score are the most likely to have a disorder if a full diagnostic protocol is used. However, such measures and cutoff scores are not identical to diagnostic interview protocols and often identify individuals with elevated symptoms who do not have a disorder. Community epidemiologic surveys of self-reported depressive symptoms have found that between 20% and 50% of individuals between the ages of 11 and 18 years exceed cutoff points for clinically significant depression (Kessler et al., 2001). As indicated, prevalence rates of MDD based on diagnostic interviews are considerably lower than these rates. This discrepancy may be due in part to an overreporting of mild mood disturbances or a large number of adolescents who experience subthreshold symptoms of depression. That is, individuals may endorse high levels of symptoms and be over an established cutoff point on a screening instrument but may not meet all necessary DSM-IV-TR criteria for diagnosis of a disorder.

Interestingly, in the development of the Center for Epidemiological Study Depression scale (CES-D), Radloff (1991) recommends different cutoff points for adolescents versus adults. On this measure, the cutoff point used to identify adults at high risk for depressive disorder is lower than that used to identify the comparable high risk group of adolescents. Such findings indicate that adolescents report greater numbers/frequency of symptoms of depression than adults even though the rates of disorder may not vary by mid- to late adolescence to adulthood. Avenevoli and Steinberg (2001) also suggest that adolescents have a "differential manifestation" of symptoms in comparison to other age groups due to unique developmental experiences of this period. As symptom measures typically include a range of symptoms related to depression or internalizing problems but not limited to the diagnostic criteria, there is evidence that symptom profiles differ for older versus younger adolescents, in particular among girls (Yorbik et al., 2004).

The particularly high rates of depressed mood and moderate rates of syndromes or problems have led to questions about the importance of these experiences in terms of concurrent difficulties or predictability to subsequent disorder. Numerous discussions have focused on whether there is merit in considering subclinical symptoms and factors that influence variations in mean scores on symptom and emotion scales. At one point in time, much of the literature was limited to assessments of affect or symptoms and did not include assessment of disorder (see Costello & Angold, 1995). As longitudinal studies that included diagnostic interviews were conducted, the literature expanded dramatically, with extensive information available on the predictors of disorder, continuity of disorder, and related issues. Such studies have shown that subclinical problems are particularly salient to identifying individuals who are most likely to develop a subsequent disorder. In the OADP, the best predictor of developing a depressive disorder over a 1-year period was having elevated symptomatology (i.e., over a cutoff

on a screener) at the first assessment (Gotlib, Lewinsohn, & Seeley, 1995). Thus, at this end of the spectrum of symptoms, there seems to be greater continuity of symptomatology than among individuals with mid- or low-level symptoms. Moreover, for depressive symptoms (Gotlib et al., 1995) and other problems (e.g., eating problems and disorders; Graber, Tyrka, & Brooks-Gunn, 2002), individuals with elevated symptoms or problems but not disorder tend to have impairment in functioning that is similar to that seen among youth who meet criteria for disorder.

Thus, factors that predict disorder or predict progression on a pathway to internalizing disorder are central to the discussion of internalizing problems in adolescence. In contrast, factors that influence perturbations or minor changes in emotion or symptoms may hold promise for future investigation but may not be important in understanding who is at risk for serious dysfunction, or who may need treatment.

A More Nuanced View of Moodiness

As mentioned previously, extreme moodiness has historically been viewed as a defining feature of adolescence. This notion of adolescence as a period of "storm and stress" has been debunked by a burgeoning number of studies examining daily fluctuations in mood during adolescence. As a means of examining daily mood and what factors influence change, Larson and colleagues (Larson et al., 1980; Larson & Ham, 1993) used the experience sampling method (ESM; reports of mood at intervals throughout the day), in what is now considered to be seminal work in the field. They found that, although adolescents demonstrated more mood changes during the day than did adults and children, frequent shifts in mood were highly dependent on negative or positive daily experiences.

A new wave of ESM studies of adolescent mood is underway, examining the effects of gender and age on changes in specific moods as well as global positive and negative affect across adolescence. For example, Weinstein and colleagues (2007) studied young and mid-adolescent youth at 6-month intervals for 1 year. At each time, an ESM protocol was conducted as was a standard symptom questionnaire. As expected, mid-adolescents reported lower levels of positive affect compared to young adolescents. In contrast, global depressed mood (from the symptom questionnaire) did not change over time, and girls reported higher levels of depressed mood than boys in both age groups and across the year. These findings suggest that the declines in mood in adolescence, which are typical, may be driven by deteriorations in positive affect, rather than increases in negative affect (Weinstein et al., 2007). Other recent studies suggest that atypical fluctuations in mood may identify adolescents at risk for more serious symptomatology or disorder. Schneiders and colleagues (2006) found that adolescents at high risk for internalizing disorders (categorized based on internalizing and externalizing symptoms, self-esteem, loneliness, etc.) were more emotionally reactive to negative events, showing greater decreases in positive affect and greater increases in depressive symptoms, compared to low-risk adolescents. Together, these findings suggest that atypical peaks in negative mood may be a risk factor for more serious symptomatology. Thus, whereas some mood variability and even decline in positive mood may be normative in adolescence, particular patterns of mood, especially if they persist and begin to interfere with activities such as school, work, or interactions with peers, are not normative and may be indicators of potentially serious psychopathology.

Comorbidity or Co-occurrence of Anxiety and Depression

As we have already alluded to, internalizing problems and disorders do not occur in isolation of other disorders and problems. Unfortunately, many studies, most often of subclinical symptoms, focus on a single outcome. With the utilization and development of comprehensive

diagnostic interviews that assess multiple disorders (see McClellan & Werry, 2000, for a special issue on these protocols), examination of co-occurrence or comorbidity has become more common. As indicated, comorbidity refers to the occurrence of a second disorder in an individual with an existing disorder; comorbidity may exist concurrently or over time. Comorbidity poses a special concern in the study of adolescent psychopathology. Caron and Rutter (1991) note that failure to identify comorbid conditions leads to two main problems. First, effects associated with the identified condition may be attributable to the other condition; and second, the experience of the other condition may influence the course of the first. Identifying comorbid conditions and the correlates of these conditions is essential for understanding the developmental processes of psychopathology across adolescence. Moreover, comorbidity may influence severity or impairment experienced by the individual and certainly impacts the course and outcomes of intervention.

It has been widely demonstrated that depression and depressive symptoms frequently co-occur with other symptoms and disorders (Kessler et al., 1996; Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993). Nearly half, or even two-thirds, of all adolescents who meet diagnostic criteria for depression have a comorbid condition (McGee et al., 1990; Rohde et al., 1991). Research also suggests that in most cases the other disorder preceded the depressive episode (Kessler et al., 1996; Rohde et al., 1991). For anxiety disorders, comorbidity is also commonly reported across studies (Kovacs & Devlin, 1998; Zahn-Waxler et al., 2000). Most importantly, comorbidity observed between anxiety and depression is quite high, with the OADP reporting a lifetime comorbidity of anxiety with MDD of 73.1% (Lewinsohn, Zinbarg, Seeley, Lewinsohn, & Sack, 1997). In particular, adolescent depression has frequently been preceded by childhood anxiety disorders.

As mentioned, Achenbach (1993) has demonstrated empirical evidence that questions whether childhood and adolescent anxious and depressive symptomatology are distinct. Compas and Oppedisano (2000) suggest that a lack of discrimination between anxiety and depression may also occur at the diagnostic level; examination of symptoms of MDD and GAD reveal several similarities—for example, symptoms of restlessness, fatigue, and irritability (see Table 19.1). Children and adolescents with mixed syndromes of anxious and depressive emotions and symptoms may be at heightened risk for development of subsequent disorders as well as for increased impairment in other areas (e.g., social interactions) than individuals with only elevated anxious or only elevated depressive symptoms.

In the Dunedin study, Krueger and his colleagues (1998) examined the extent to which specific disorders may actually be indicators of what they termed "stable, underlying core psychopathological processes." A two-factor model of internalizing versus externalizing disorders demonstrated the best fit at ages 18 and 21 years, and individuals demonstrated substantial continuity in their relative position on these latent constructs over this period. Such an approach may explain concurrent comorbidity rates within internalizing disorders (i.e., MDD and anxiety disorders) as well as the longitudinal links between prior anxiety disorders and subsequent adolescent depressive disorder.

In addition, subclinical internalizing symptoms and problems also demonstrate high rates of co-occurrence with other types of problems. For example, several studies report moderate to high correlations between scores for internalizing and externalizing symptoms (see Zahn-Waxler et al., 2000 for a brief review). In a study of subclinical eating problems and depressive symptomatology (Graber & Brooks-Gunn, 2001), individuals with co-occurring problems reported the greatest disturbances in family and peer relationships in comparison to individuals with only one or the other problem. Adolescent girls may be at particular risk not only for disorder but also

for comorbidity of multiple disorders due to unique developmental issues surrounding pubertal maturation among females. A community study of young to mid-adolescent girls examined associations between early pubertal maturation and comorbidity in depression, substance use, and eating problems and found that early menarche in girls is associated with greater risk for comorbid depression and substance use (Stice, Presnell, & Bearman, 2001). Thus, co-occurrence of problems and comorbidity of disorder is fairly normative for adolescent psychopathology. This fact may partially explain why many risk factors for internalizing problems are not found to be specific to internalizing problems but rather are linked to various psychopathologies.

DEVELOPMENTAL MODELS FOR CHANGES IN INTERNALIZING SYMPTOMS AND DISORDERS DURING ADOLESCENCE

A number of models have been proposed to explain the development of internalizing problems, and more specifically depression, throughout the life span. Some models identify general processes that may lead to elevations in depressed or anxious mood or even disorder but do not specify why rates shift dramatically for depression during adolescence. Other models focus on adolescent development and internalizing problems but may or may not be applicable to general processes that lead to depression at other periods in the life span. For example, puberty is often included in models explaining increased internalizing problems during adolescence but rarely is mentioned in general process models of depression in adulthood. In the adolescence literature, focus has centered on models that incorporate developmental experiences in order to explain individual differences in behavioral and adjustment changes (e.g., Graber & Brooks-Gunn, 1996). The models stem from interactional or transactional approaches that might apply to development throughout the life span, but in these cases, particular transitions or experiences of adolescence are focal to understanding adjustment or behavioral change at this time (Graber & Brooks-Gunn, 1996). Models that are particularly salient for understanding developmental changes and individual differences in internalizing problems during adolescence are: (a) diathesis-stress (Ingram & Luxton, 2005); (b) cumulative or simultaneous events (Petersen, Sarigiani, & Kennedy, 1991; Simmons & Blyth, 1987); (c) accentuation (Caspi & Moffitt, 1991; see also Susman & Dorn, this volume); and (d) differential sensitivity (Graber & Brooks-Gunn, 1996).

Diathesis-Stress Models

As noted in this chapter and throughout this volume, adolescence is remarkable as a developmental period because of the confluence of transitions and challenges that occur during this decade of life. General models of psychopathology, in particular, typically focus on the experience of stressful life events (Ingram & Luxton, 2005). That is, significant stressful occurrences commonly precede depressive episodes, and dysregulation of the physiological stress system occurs in the face of high levels of psychosocial stress. However, events in and of themselves rarely fully explain changes in affect or onset of disorder. Attention must also be given to the interplay of vulnerability, risk, and protective factors across developmental stages. As indicated, vulnerability, or "diathesis," refers to predispositional, usually intraindividual, factors that predict internalizing symptoms, (problems, or disorders, i.e., emotion regulation, physiological responses to stress, or temperament) that are in part shaped by experience but often become stable during childhood and adolescence (Ingram & Luxton, 2005). In contrast, risk factors are those factors associated with increased probability of internalizing problems but may not clearly be identified as causal (e.g., Ingram & Luxton, 2005). For example, gender is a risk factor for internalizing problems during adolescence because gender is associated with higher probability of developing these problems but does

not explain why the problems occur more often for girls. Protective factors moderate or buffer the impact of risk factors and potentially impede the development of internalizing problems.

The diathesis-stress model of depression predicts that major transitions or negative events interact with prior vulnerabilities to psychopathology, resulting in increased problems or poor outcomes in the face of these stressors. In testing this model, the focus is typically on the identification and development of prior vulnerabilities to adjustment problems (e.g., poor emotion regulation skills, depressogenic cognitive styles, genetic markers). Ingram and Luxton (2005) note that these vulnerabilities must exist prior to the emergence of internalizing problems and as noted differ from risk factors of internalizing problems.

Cumulative or Simultaneous Events Models

Models of cumulative and simultaneous events posit that when individuals experience major events or transitions that typically occur during adolescence (e.g., school changes or pubertal development) either in close sequence (cumulatively) or simultaneously, they are more likely to have negative behavioral and emotional outcomes as a result of the confluence of events. Cumulative events may also be characterized by increasing numbers of stressful events that occur for adolescents commensurate with changes in peer groups, friendships, parental relationships, and school demands (e.g., the number of stressful events increases during early and mid-adolescence; Brooks-Gunn, 1991). Although conceptually the impact of simultaneously occurring events may differ from closely successive events, this is rarely, if ever, tested as most studies define events as simultaneous if they occur within a 6- to 12-month period (Graber & Brooks-Gunn, 1996). Of course, most adolescents effectively navigate the challenges and transitions of adolescence; however, in this model, coping resources may be overwhelmed by the experience of multiple changes in close proximity, and thus internalizing symptoms increase. In essence, the experience of stressful events and their timing, as well as the increased likelihood that certain events will occur during adolescence are the critical factors predicting internalizing problems in this model.

Accentuation Models

Accentuation models posit that major developmental transitions accentuate preexisting problems or vulnerabilities, resulting in increased problems and poor outcomes after the transition (e.g., Elder & Caspi, 1990). Drawing on Piagetian theory, the model suggests that individuals will assimilate new information and experiences into preexisting behavioral, emotional, or cognitive patterns of response to challenging situations (Graber & Brooks-Gunn, 1996). Studies supporting such a model have found that preexisting behavioral problems are accentuated by major transitional events (e.g., puberty and parenthood; Caspi & Moffat, 1991; Cowan, Cowan, Hemming, & Miller, 1991). However, most tests of this model have been made 2–3 years after the time of transition, and the question of how subsequent transitions are negotiated is still unanswered (Graber & Brooks-Gunn, 1996). Note that accentuation models focus on major transitions that exacerbate preexisting problems or vulnerabilities, in contrast to diathesis-stress models that focus on prior vulnerabilities that interact with any type of stressful event.

Differential Sensitivity

Contrary to the previous models discussed, differential or heightened sensitivity models emphasize the potential for vulnerability within an individual to vary depending on the developmental period. That is, periods of transition may present increased vulnerability to the individual in how stress is experienced. Heightened sensitivity models have arisen from studies of women's reproductive transitions (puberty, pregnancy, menopause) and the connections between these transitions and changes in mental and physical health (e.g., Graber &

Brooks-Gunn, 1996). Biological systems may be more sensitive to environmental or contextual influences during times of rapid change, as seen in reproductive transitions. Similarly, models of differential sensitivity, also like accentuation or diathesis-stress models, suggest that individuals with preexisting characteristics are potentially more sensitive to developmental transitions and challenges. In this case, the transition period results in heightened vulnerability to stress resulting in poorer mental or physical health, but vulnerability may be less significant in nontransitional periods. Notably, one concern is that individuals who experience heightened vulnerability and enter a path for poorer health may continue on that trajectory after the transitional period ends.

Clearly, these models share many similarities and the distinctions are predominantly in the area of emphasis (stressful events versus vulnerabilities) and the extent to which they focus on developmental transitions as distinct from other stressful events. (Table 19.2 provides an overview of the primary emphases of these models.) These models will be revisited in the following sections in cases where they have been used to explain findings in the literature.

VULNERABILITY AND RISK FACTORS ASSOCIATED WITH INTERNALIZING PROBLEMS

In general, the etiology of internalizing problems can be organized around several vulnerability and risk factors. These include: stress; psychological processing of stress including cognitive processes, personality, and related psychopathology; biology of stress, including markers of disorder, neurodevelopment, and hormones; genetics and gene environment interactions; and interpersonal relationships (e.g., Birmaher et al., 1996; Garber, 2000; Petersen et al., 1993). From a developmental perspective, such categorizations often fall short of examining integrative, transactional, or biopsychosocial processes; for example, genetic and parenting effects on depression are no doubt interactive (Collins, Maccoby, Steinberg, Hetherington, & Bornstein, 2000). Hence, consideration of how these factors influence each other and how they may be influenced by broader contextual factors (e.g., gender, transitional periods) will be taken into account. Also, within any particular study of adolescents, the subgroup experiencing an internalizing problem may be experiencing it for the first time or may have a recurrent or persistent problem. Recent longitudinal studies of depression find unique predictors of each (e.g., Lewinsohn et al., 1998). As noted, many more studies focus on the prediction and correlates of depression (MDD) and depressive symptoms, whereas much less in known about anxiety. Hence, the following sections most often apply to depressive problems and may not include literatures on all types of internalizing problems.

Stressful Events

As indicated, many models of the etiology of internalizing problems are based on the

TABLE 19.2 Models of the Development of Internalizing Problems

Model	Stress Associated With	Vulnerability
Diathesis-Stress	Stressful events	Traitlike characteristic that develops in childhood or during adolescence
Cumulative/Simultaneous Events	Developmental transition or multiple transitions	Conferred by timing of transition and overwhelming of psychological resources
Accentuation	Developmental transition or multiple transitions	Existence of prior internalizing problems
Differential Sensitivity	Stressful events	Develops during a transition due to some aspect of the transition

diathesis-stress model. Hence, stress or stressful events are critical risk factors for internalizing problems. Lazarus and Folkman (1984) define psychological stress as a "relationship between the person and the environment that is appraised by the person as taxing or exceeding his or her resources and endangering his or her well being," (p. 19). Garber (2000) defines stressful life events as "... circumstances characterized by either the lack or loss of a highly desirable and obtainable goal or the presence of a highly undesirable and inescapable event," (p. 475). As is demonstrated by these two highly cited definitions, numerous interpretations of stress exist within the psychological literature. Regardless of definitions, there is much consensus and evidence that events identified as stressful by the individual are linked to internalizing problems.

The adult and adolescent depression literatures frequently report retrospective associations between the experience of major life events and the experience of a depressive episode. Among adults, 60%–70% of individuals with MDD report a major stressful event in the preceding year, usually some type of loss; effects are more modest in studies of depressed children and adolescents (Birmaher et al., 1996). Traumatic loss, such as exposure to another's suicide, dramatically increases the risk for depression among adults and adolescents (Birmaher et al., 1996). Similarly, anxiety disorders may also be preceded by a stressful life event (Vasey & Ollendick, 2000). The events need not be associated with the anxiety problem directly, as in the case of PTSD, in which a precipitating event leads to the distress response, but instead, other anxiety disorders such as GAD may arise after family relocations, school changes, or other stressful life events.

The individual's assessments of the importance of the event, how negative the event is, what impact it has on other areas of the individual's life, and whether the event is controllable or uncontrollable are all factors affecting whether stressful events are associated with increased internalizing problems or disorder (Birmaher et al., 1996; Vasey & Ollendick, 2000). Compas Howell, Ledoux, Phares, and Williams (1989) report that major events in the family tend to increase the number of minor stressful events for family members, which leads to increased behavior problems (internalizing and externalizing symptoms) among young adolescents; thus, events in the broader context of an adolescent's life as well as immediately experienced events are salient to changes in internalizing symptoms. Furthermore, individual differences in terms of how events are interpreted or processed cognitively have been linked to onset and maintenance of internalizing problems (Kaslow, Adamson, & Collins, 2000; see subsequent section).

In general, such associations between stressful events and internalizing symptoms would apply to any point in the life course. As noted, cumulative or simultaneous event models suggest that internalizing symptoms increase during adolescence because adolescents are more likely to experience more stressful life events than individuals of other ages, given the nature of normative adolescent development; these events include puberty, school change, changes in family relationships, changes in peer relationships, and so on. Reports of stressful life events (both negative and positive events) have been found to increase during early to mid-adolescence, with higher rates of stressful events associated with increases in internalizing symptoms (Brooks-Gunn, 1991; Ge, Lorenz, Conger, Elder, & Simons, 1994).

In the few studies that have looked at the simultaneity or ordering of developmental events or transitions, the occurrence of peak pubertal development (as indexed by rapid change in physical growth) prior to school change was predictive of increased depressive symptoms several years later, at 12th grade (Petersen et al., 1991). Notably, this effect was found only among girls. Given the normative differences in pubertal timing between girls and boys, with girls showing physical changes of puberty earlier than boys, and the normative grades when young adolescents make

school transitions, only girls had significant pubertal changes prior to making a school change. Hence, gender differences in internalizing symptoms over adolescence may in part be explained by the timing of developmental events and transitions.

In addition to greater likelihood of experiencing synchronous events and transitions, girls may be at greater risk for increases in internalizing symptoms because of the types of events that normatively occur in adolescence and how girls respond to them. Specifically, during the middle school years, adolescents frequently experience changes in their close relationships, endorsing events such as breaking up with a friend, having a fight with a parent, and so on. Girls, in comparison to boys, may be more likely to experience negative emotions in response to events in relationships, report more events that are relationship focused, and perseverate about events that have happened with peers (Kessler & McLeod, 1984; Rudolf, 2002). Because of girls' tendencies to rely heavily on peer relationships for emotional support and intimacy, these events may elicit more frequent or prolonged internalizing responses in girls, especially girls with vulnerabilities in interpersonal relationships (Rudolph, 2002; see subsequent section).

Overall, stressful events may increase in number due to changes in adolescent experience, may increase in magnitude in association with major developmental transitions, and may have a more deleterious effect due to the timing of events. The impact of events may vary due to psychological factors or biological factors. Acknowledging that neural processes are the basis of psychological factors, each will be discussed separately.

Psychological Factors: Cognitions, Emotion Regulation, and Temperament

Cognitions

As not all adolescents who experience challenge or stress develop internalizing problems, individual characteristics, skills, and capacities are often vulnerabilities that interact with stress increasing the likelihood of internalizing symptoms and problems. Cognitive changes during adolescence are a foundation of changes in self-evaluation and processing of the other challenges of adolescence (Harter, 1998). While cognitive abilities increase in several domains during adolescence, allowing for more nuanced reflections on the self, one's future, and the world (Keating, 2004), one question that remains is the extent to which adolescent changes in cognition are predictive of changes in internalizing symptoms. A well-documented literature demonstrates that cognitive styles or cognitive attribution biases are associated with depressive symptoms and that these styles and biases are often established prior to adolescence (Kaslow et al., 2000; Nolen-Hoeksema, 1994). Hypothetically, some cognitive-based vulnerabilities may develop during adolescence commensurate with cognitive development, whereas others may be preexisting, interacting with the developmental challenges of adolescence to lead to internalizing problems. For individuals with preexisting vulnerabilities, adolescence may be the first time in development when sufficient stressors have occurred to result in problems.

Kaslow and colleagues (2000) identify three primary areas of cognitive processing that are associated with depression: negative self-schemas (negative views of the self), faulty information processing (attributional biases), and negative expectancies (helplessness and hopelessness). As noted, changes in thinking about the self are part of adolescent development. Self-consciousness also seems to increase during early adolescence. In addition, assessments of self-image or esteem tend to demonstrate moderate to strong associations with internalizing symptoms, making it difficult to consider negative self-evaluations separately from these symptoms.

Appraisals of physical development or body image are areas of self evaluation that are particularly salient to adolescents and their risk for internalizing problems. Although most youth

demonstrate increases in body esteem over adolescence, disturbances in body image are common during early adolescence (or at puberty) for both girls and boys (Graber, Petersen, & Brooks-Gunn, 1996), and in turn have been shown to prospectively predict increases in depressive symptomatology in adolescents (Paxton, Neumark-Sztainer, Hannan, & Eisenberg, 2006). At the same time, body image concerns seem to have a stronger impact on girls' internalizing symptoms as well as eating disorders and symptoms than on boys' symptoms (e.g., Allgood-Merten, Lewinsohn, & Hops, 1990; Rierdan, Koff, & Stubbs, 1989). In our own work in a longitudinal study of girls, many girls experienced a serious disturbance in body image at some point during adolescence (Ohring, Graber, & Brooks-Gunn, 2002). Body disturbance was concurrently associated with elevated internalizing and disturbed eating symptoms. However, girls with recurrent or persistent poor body image during adolescence not only had elevated depressive and eating symptoms during adolescence, but also reported more symptoms in young adulthood.

Nolen-Hoeksema (1994) postulated that gender differences in depression emerge during adolescence due to an interaction between how girls experience puberty and gender differences in the ways that adolescents experience and react to stress. Consistent with this proposition, we found that girls with persistent body dissatisfaction were also more likely to have gone through puberty earlier than their peers (Ohring et al., 2002). Thus, negative self evaluations (i.e., body dissatisfaction) were linked to puberty, and individual differences in puberty were important in determining who had continued negative self evaluations. In the limited literature on boys' body dissatisfaction, comparable individual differences are not found. Hence, the development during early adolescence of negative self-evaluations about the body may be an important factor explaining gender differences in depression, as well as explaining why some girls have more internalizing symptoms than other girls.

In contrast, faulty information processing and negative expectancies are cognitive vulnerabilities that are likely in place prior to adolescence. These factors are moderators of the experience of adolescent challenges in predicting not only internalizing symptoms, but also aggressive symptoms. In general, cognitive styles or faulty information processing usually reflect how social information or events are interpreted by the individual. Cognitive biases that attribute negative events to internal, stable, and global causes and biases that attribute positive events to external, unstable, and specific causes are linked to elevated depressive symptoms (Kaslow et al., 2000). The interpretation of the controllability of events in one's life has also been linked to both depressive and anxiety symptoms (Alloy & Abramson, 2007; Vasey & Ollendick, 2000). In particular, a sense of hopelessness (i.e., belief that events have internal, stable, and global causes that one can do little about) has been linked to depressive disorder.

Cognitive styles or attributions consistently differentiate youth with, versus without, disorders and those with elevated, versus normative, levels of internalizing symptoms (see Kaslow et al., 2000 for a review). However, a large meta-analysis did not find that attributional styles consistently interacted with life events to predict depression, as would be expected (Joiner & Wagner, 1995). Much of the research to date on this issue is cross-sectional, making it difficult to determine if cognitive styles develop or change substantially during adolescence. In analyses with the OADP, when youth were in high school, many adolescents demonstrated consistent cognitive styles over a 1-year period but subgroups of youth demonstrated change (Schwartz, Kaslow, Seeley, & Lewinsohn, 2000). Change in cognitive styles was associated with other cognitive factors; for example, better self-esteem at the initial assessment predicted change from maladaptive to an adaptive cognitive style over time.

Notably, certain attributions or cognitive styles may be more salient to the maintenance

and recurrence of depression than to onset. For example, individuals who ruminate on negative emotions tend to have longer and more severe depressive episodes. Importantly, Nolen-Hoeksema (1994) suggests that girls are more likely to be ruminators than are boys. In her model, girls are not only more likely to experience normative developmental experiences negatively (i.e., form negative body images in response to the changes of puberty) but also girls are more likely to demonstrate an attributional style (i.e., rumination) that magnifies those negative feelings. Such an interactive model would address the question of why more girls become depressed or have elevated internalizing symptoms and problems. Alloy and Abramson (2007) lend further support to this model, postulating that changes in cognitive functioning due to brain development during adolescence result in improvements in attentional executive functions, increases in working memory skills, improved hypothetical thinking, and future orientation. These neural–cognitive changes serve as prerequisites for generating negative cognitions about stressful events, experiencing feelings of hopelessness, and rumination.

Along with attributions about events and expectations about one's ability to influence events, individuals also differ in the approaches they use to cope with the events. These factors are often related, as seen with ruminating styles. That is, when individuals ruminate, they focus on negative emotions rather than engage in problem solving or distraction, and thus respond to the emotions with continued perseveration about the negative emotions or events. More generally, cognitive beliefs about the extent to which one's actions can influence events, what can be done about events, or why the events occurred are in part the result of attributional biases. However, these biases also lead to subsequent behavioral responses to events and feelings. Hence, coping behaviors and cognitive processing are interactive in predicting the development of internalizing symptoms (Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001).

Emotion Regulation

As noted, internalizing problems are defined as dysregulations in mood and affect, and hence, by definition, are indications of difficulties in regulating negative emotions. Emotion regulation skills tend to develop substantially during early childhood (Rothbart & Bates, 2006; Zahn-Waxler et al., 2000), but also continue to develop in response to new demands of adolescent emotional experiences. Deater-Deckard (2001) identified emotion regulation and social cognitive skills as particularly salient to the peer relations of children and adolescents. For example, among young adolescents, better abilities to understand others' feelings or thoughts are associated with greater peer acceptance (Bosacki & Astington, 1999).

In some sense, emotion regulation and coping are similar constructs in that both involve managing one's reactions to negative and potentially stressful situations. Lazarus and Folkman (1984) define coping as an effort to manage specific external and/or internal demands that are appraised as taxing or exceeding the resources of the person. In addition to these active coping strategies, some individuals have vulnerabilities that impact emotion regulation and hinder effective adaptation to stressful experiences. These vulnerabilities may include temperamentally based and conditioned cognitive, behavioral, and physiological reactions to stress that may or may not be within conscious awareness and are not under personal control, such as physiological arousal, intrusive thoughts, and rumination.

Coping strategies aimed at removing the stressor or resolving the emotional, physiological, or cognitive distress (e.g., problem solving, seeking support, expressing emotions, reevaluating the situation, etc.) are typically associated with more positive adjustment and fewer internalizing problems (Compas et al., 2001; Sontag, Graber, Brooks-Gunn, & Warren, 2008). However, studies with adults have demonstrated that the effectiveness of coping strategies is dependent on the type of stressor and the cultural background of the

individual (e.g., Compas et al., 2001; Utsey, Bolden, Lanier, & Williams, 2007). In recent work examining coping strategies, peer stress, and symptoms of psychopathology in young adolescent girls, we found that, compared to White girls, African American girls were less likely to use traditionally effective coping strategies, such as problem solving, emotional expression, and regulation (Sontag, Graber, & Brooks-Gunn, 2007). At the same time, exploratory analyses revealed that the tendency to use problem solving, emotional expression, and emotion regulation more often than other strategies mediated the association between stressful peer events and internalizing symptoms for African American girls only. Differences found between African American and White girls in use and impact of coping strategies suggest that standard measures of coping may not capture the full range of strategies used by adolescents from diverse ethnic groups.

Although it has been shown that more highly developed emotion regulation and coping skills in adolescents are associated with a lower incidence of internalizing problems, Zahn-Waxler and her colleagues (2000) have argued that promoting emotion regulation in early childhood may be linked subsequently to poor emotion regulation later in development. In particular, regulating externalizing behaviors (e.g., anger) is a primary task of early childhood. But individuals, particularly girls, who learn to overregulate these behaviors and emotions, may develop dispositions for internalizing emotions, such as fear and guilt. For example, parents can socialize children to be more or less anxious by encouraging regulation of fear via avoidance of the stimuli or situation versus encouraging a sense of control and self-efficacy (Vasey & Ollendick, 2000). Such findings speak to how attributional biases may develop with parents socializing children to develop different beliefs about control and efficacy in their environments (Zahn-Waxler et al., 2000). At the same time, attributions and emotion regulation skills may result from the interaction of biologically based (possibly with a genetic component) factors and socialization. Unfortunately, much of the research on emotion regulation effects on internalizing problems is based on cross-sectional comparisons of different age groups, and future longitudinal investigations are needed.

Temperament

As suggested in the discussion of attributional biases, many individuals have consistent ways of interacting with their social worlds, whereas for others, change in these constructs may occur across development. Temperament or personality may be one way of conceptualizing consistency in how individuals respond to their environments. A detailed discussion of how personality or temperament may be linked to internalizing problems over the course of development is beyond the scope of this chapter. However, a few brief examples support suggestions that these constructs may be important and understudied in their role in the development of psychopathology more generally.

Kagan and Fox (2006) view temperament as a stable way of responding to the environment, in particular, to unexpected events. Similarly, Block and Block (1980) describe ego control (impulse inhibition/expression) and ego resilience (flexibility to modify impulse control depending on the situation) as personality types, which reflect how an individual responds to environmental stress or frustration. Research based in each approach has found that children and adolescents who were inhibited or overcontrolled had higher rates of internalizing problems than those with other temperament or personality types (see Kagan and Fox, 2006, for a review). Perhaps the most compelling evidence in this area stems from Suomi's (1999) animal models of the interaction of temperament and rearing conditions. Selective breeding of highly reactive or inhibited monkeys produced offspring with these response patterns; however, rearing experiences had a profound impact on the behavioral adjustment of these monkeys over time.

Thus, compelling evidence from nonhuman primate and human studies indicate that temperament may be a vulnerability for internalizing problems, but that other factors mediate or moderate subsequent associations between this vulnerability and psychopathology. Such findings do not directly speak to why internalizing problems increase at adolescence in humans or why more girls than boys develop these problems. These factors likely play a role in answering these questions when temperament is examined along with developmental challenges and gender differences in stressful events. Furthermore, differential parenting practices of girls versus boys who are overcontrolled or inhibited/reactive may account in part for the gender differences in the development of particular attributional biases or coping responses.

"BIOLOGICAL MARKERS" OF DISORDER AND NEUROENDOCRINE PROCESSES OF STRESS

One scientific challenge has been to identify how experiences of stress translate into the behavioral and biological dysregulations observed in depressive and anxiety disorders. Investigations of biological mechanisms of disorder first focused on determining which neural and physiological systems differed between disordered and nondisordered individuals. More recently, process-oriented studies have considered how experiences interact with biological systems in the development of internalizing disorders.

Numerous studies of adults have documented altered or abnormal neuroendocrine functioning when comparing individuals with MDD or an anxiety disorder to individuals who do not have disorder, or have one or more nonaffective disorders (see Dahl & Ryan, 1996; Meyer, Chrousos, & Gold, 2001 for reviews). In comparable pediatric studies of MDD, several of these findings have not been replicated among children and adolescents. Such discrepancies were puzzling, as children, adolescents, and adults reported many similarities in the experience and symptoms of internalizing disorders. Discrepancies in neuroendocrine studies and pharmacological trials resulted in several tangential and occasionally nonproductive discussions as to whether children and adolescents were "really" depressed if they did not show biological markers of depression comparable to adults. For example, many of the drugs used to treat adulthood depression (e.g., tricyclics, lithium, and serotonin–norepinephrine reuptake inhibitors [SNRIs]) have been ineffective in treating MDD in children and adolescents. In contrast, efficacy trials of the present generation of drugs (primarily, selective serotonin reuptake inhibitors [SSRIs]), indicate that this class of drugs seems to be comparably effective in treating depression in children, adolescents, and adults (Singh, Pfeifer, Barzman, Kowatch, & DelBello, 2007). Notably, the neuroendocrine system is still undergoing substantial normative development in childhood and adolescence, making it likely that maturational factors account for some of the observed differences between adults versus children and adolescents. A comprehensive listing of which markers are or are not found in adolescents versus adults is beyond the scope of this chapter (see Dahl & Ryan, 1996; Meyer et al., 2001, for reviews). Most importantly, though, initial studies of differences between disordered and nondisordered individuals did not identify predictors of these disorders. Instead, such markers identify factors that need to be explained in the development of a disorder or possible systems that are disrupted by the disorder. Ultimately, the goal of neuroscience investigations is to link experiences to abnormal functioning of neurological or endocrine systems.

L-HPA and HPG Endocrine Systems

Two hormonal systems, the limbic–hypothalamus–pituitary–adrenal (L-HPA) axis and the hypothalamus–pituitary–gonadal (HPG) axis, appear to be central to the development of internalizing disorders, in part, stemming from studies of biological markers (see above), and

in part from an interest in behavioral change with puberty (both the L-HPA and the HPG systems control hormonal changes of puberty). In addition, human and animal research demonstrates gene–environment interactions in the development of L-HPA functioning (e.g., Caspi et al., 2003; Francis, Champagne, Liu, & Meaney, 1999). The following provides a brief review of how changes in each system may be linked to internalizing problems.

The L-HPA Stress System

Activation of the L-HPA system occurs in response to novelty and stress, with a particular sensitivity to social stressors. During or briefly after exposure to the stimulus, hormones in the brain stimulate the pituitary to secrete adrenocorticotropic hormone (ACTH), which in turn stimulates the adrenal gland to secrete cortisol (see Gunnar, 2007; Meyer et al., 2001). About 20–30 minutes after the stressor, peak concentrations of cortisol are found in the system. Following this peak, the negative feedback loop of the L-HPA axis begins to reestablish homeostatic functioning by reducing the level of circulating cortisol. In parallel, as the individual assesses the threat level or copes with the challenge, cortisol levels also tend to decrease. For individuals who follow a typical day–night schedule, cortisol production rises during the last few hours of sleep, peaks just after awakening, declines rapidly throughout the morning, and then remains at relatively low and stable levels in the late afternoon and evening, with increases again during periods of sleep (Gunnar & Vazquez, 2001).

Interestingly, acute elevations of cortisol in response to stressful situations may promote cognitive processing of emotions; that is, humans and animals remember experiences that activate stress responses (McEwen, 2000). However, chronic production of cortisol appears to damage brain structures. Note that chronic cortisol production is not always due to exposure to chronic stressors. Dysregulation in the cortisol response to minor stressors, which most individuals experience on a frequent basis, can also play a role. While the majority of people adapt quickly to a minor stressful event, some individuals display longer lasting elevations in cortisol levels. The result is a seemingly chronic cortisol response to daily stressors, such as social hassles. Major life events can lead to chronic or overall greater net cortisol exposure and their potential damaging effects via this mechanism, by increasing the number of daily stressors experienced by these individuals (e.g., Compas et al., 1989).

Animal studies and recent work in humans have demonstrated that early experiences may result in long-term alterations in the L-HPA system. In rodents, studies of maternal separation in early development have revealed interesting effects of maternal behavior on the L-HPA axis (Boccia & Pedersen, 2001). Specifically, following a short separation, mothers engaged in increased care of their offspring; when exposed to other stressors, these offspring demonstrated smaller hormonal stress responses and quick recovery to baseline in comparison to nonseparated pups. In contrast, following long maternal separations, mothers decreased care of their offspring; when exposed to stressors, these offspring had higher and prolonged hormonal stress responses and greater anxiety and fear. Subsequent studies demonstrated that some mothers provided more or less care in the absence of the experimental separation condition. Effects on offspring were quite similar in comparisons of naturally occurring variations in maternal behavior with experimentally induced variations in maternal care. As indicated previously, the seminal work of Suomi (1999) and his colleagues has documented the interaction of genetic-based vulnerabilities for reactive, particularly high or sustained, responses to stress and maternal behavior in rhesus monkeys. In this work, reactive monkeys (those who demonstrate heightened responses to novelty and challenge) reared with less responsive or less nurturing caregivers were likely to develop symptoms and behaviors indicative of depression in response to stressors. In contrast,

reactive monkeys who were reared by highly nurturing, patient mothers were likely to excel in the troupe social hierarchy, often becoming leaders in the group. The effects of parenting on the developing L-HPA system speak to how physiological vulnerabilities or perhaps invulnerabilities to stress may be established via early experience.

At present, there is a burgeoning of studies of the stress response in children, adolescents, and adults, including nonclinical samples. Across studies, adrenocortical activity as indicated by changes in basal levels and variations in response to stress have been associated with internalizing problems (Klimes-Dougan, Hastings, Granger, Usher, & Zahn-Waxler, 2001; Southwick, Vythilingam, & Charney, 2005). In one such study of young adolescents, Klimes-Dougan and colleagues (2001) found that maintaining an increased cortisol response to social challenges (i.e., public speaking task or conflict discussion task) was associated with both internalizing and externalizing symptoms; having a strong decrease in cortisol in response to the challenge was also associated with internalizing symptoms. However, within the child and adolescent literature, links between cortisol and internalizing problems have not been consistently found, especially in community rather than clinical studies (Klimes-Dougan et al., 2001). Hence, additional studies on how and why early vulnerabilities may eventually result in the dysregulations observed in depression among adults and whether dysregulations are specific to particular types of problems or disorders are warranted.

L-HPA and HPG Changes at Puberty and Depression As noted, the HPG and L-HPA axes are both involved in pubertal development. As studies of pubertal hormones and internalizing symptoms are reviewed in detail by Susman and Dorn (chapter 5, this volume) and elsewhere (e.g., Graber, 2008), the present discussion is limited to main findings. Substantial maturation of these systems during puberty results in dramatically increasing hormonal levels, with some hormones (e.g., estradiol, DHEAS) linked to changes in internalizing symptoms and disorders. Adrenarche, or maturation of the adrenal glands, involves the production of dehydroepiandrosterone (DHEA) and its sulfate (DHEAS) as well as other hormones, including testosterone. A different area of the adrenal gland is responsible for the secretion of cortisol. Walker, Walder, and Reynolds (2001) have found that the adrenal-cortisol areas mature linearly and continue to mature in the postpubertal period. They have also suggested that this maturation may be linked to expression of symptoms and disorder, most likely among individuals with vulnerabilities to these problems.

Initial studies of puberty and depression compared hormone profiles of depressed prepubertal and postpubertal individuals (Dahl & Ryan, 1996). Again, the physiological dysregulation observed in adult patients, was not observed in depressed children and adolescents. However, recent studies have identified altered diurnal patterns of cortisol secretion among children with anxiety disorders and adolescents with depression (Forbes, Silk, & Dahl, in press). Whereas the neuroendocrine dysregulation observed with MDD in adulthood is not fully present in children with MDD, diurnal alterations in cortisol may be an indication that pubertal maturation of the L-HPA is necessary before adult-type neuroendocrine concomitants of depression are observed. Of course, it should be remembered that even though neuroendocrine profiles of MDD differ between adolescents and adults, few differences in the experience of symptoms of depression are found from childhood into adulthood (e.g., Birmaher et al., 2004).

Along with puberty-related changes in the L-HPA system, evidence for links between HPG axis changes and internalizing problems has also been found. In particular, periods of puberty characterized by rapid increases in hormones as indexed by estradiol have been linked to higher levels of depressive symptoms among girls (Brooks-Gunn & Warren, 1989). Furthermore in the GSM, higher rates of

MDD were observed in girls who had attained a threshold level of estradiol, typical of the later stages of puberty (Angold, Costello, Erkanli, & Worthman, 1999). One problem with such findings is that ultimately all girls will attain the hormonal levels typical of adult reproductive functioning, but not all girls will become depressed. Subsequent analyses by Angold in the GSM indicate that other factors (i.e., maternal mental health) moderate these associations (Angold, Worthman, & Costello, 2003). It may be that depression is less likely to manifest prior to advanced or postpubertal development, at least in girls, an issue that will be discussed more fully in our review of the role of genetic factors. Notably, in the GSM, girls with estrogen levels indicative of later maturation were not more sensitive to depressogenic effects of life events than girls with lower levels of estrogen (Angold et al., 2003). In contrast, it may be that individual differences in postpubertal estrogen are more important than differences in estrogen as a function of pubertal stage. That is, some individuals do, in fact, have elevated gonadal hormonal levels postpubertally and in adulthood. A few studies (e.g., Lai, Vesprini, Chu, Jernström, & Narod, 2001) have reported that women who experienced menarche at earlier ages may have higher levels of estrogen in adulthood. In this case, propensity for elevated estrogen levels in women may be a vulnerability for depressive problems over the life course.

Of course, it is important to remember that the L-HPA and HPG systems are interconnected and that hormones do not function in isolation of one another. Interactions between these systems may result in experiencing the pubertal transition as a time of heightened sensitivity to environmental events or stressors. Walker (2002) has suggested that increases in gonadal hormones play an important role in the activation and regulation of the stress response and that the association between baseline hormone levels and behavior is nonlinear, emerging only after a certain threshold level of gonadal hormones has been reached. Lending support to this hypothesis, Netherton, Goodyer, Tamplin, and Herbert (2004) found distinct gender differences in morning salivary cortisol levels in mid- to postpubertal adolescents, with girls exhibiting higher levels than boys; this difference did not emerge for pre- to beginning-pubertal boys and girls. In our lab, we have also found evidence that aspects of puberty (i.e., pubertal timing) and stress responses were associated in girls; in this case, early-maturing girls who also had high emotional arousal or high-for-development DHEAS levels had the highest levels of internalizing symptoms. Hence, the interaction of L-HPA and HPG systems may be most salient for understanding initial changes in internalizing symptoms in the early adolescent years (Graber, Brooks-Gunn, & Warren, 2006).

McEwen (1994) points out that gonadal hormones have effects in the brain beyond reproductive systems, including in the hippocampus, where many effects of stress are also observed. Taylor and her colleagues (2000) also identify behavioral responses to stress that may be more representative of female responses to stress, especially social stressors. Moreover, the physiological mechanisms underlying such responses involve female reproductive (gonadal) hormones and oxytocin, a hormone that is often associated with maternal caregiving behaviors as well as lactation. Advances in understanding gender differences in internalizing problems may be afforded by considering multiple "stress" systems and interactions across systems.

Pubertal Timing and Internalizing Problems

In addition to the role that hormones may play, increases in internalizing problems have also been linked to other aspects of puberty. Over the past 15 years, several studies have found that pubertal timing (going through puberty earlier, at about the same time, or later than one's peers) is associated with psychopathology during adolescence. In particular, earlier maturation among girls

is associated with higher rates of depressive symptoms and disorders (e.g., Graber, Seeley, Brooks-Gunn, & Lewinsohn, 2004; Hayward et al., 1997; Stice et al., 2001), as well as externalizing (e.g., Graber, Lewinsohn, Seeley, & Brooks-Gunn, 1997; Sontag et al., 2008), and eating disorders and symptoms during adolescence (e.g., Graber, Brooks-Gunn, Paikoff, & Warren, 1994; Graber et al., 1997).

Findings are most consistent for early maturation effects in girls when looking at the disorder end of the continuum, although several studies find effects for subclinical symptoms (Graber, 2008). Of the studies that have examined disorder, three report an effect of early maturation on MDD or general internalizing disorders (Graber et al., 1997; Hayward et al., 1997; Stice et al., 2001); only one study did not find an effect (Angold, Costello, & Worthman, 1998). This inconsistency likely resulted from an unusual truncation of range in pubertal timing in the analyses (Graber, 2008). Of note is that these studies used a range of methods for assessing pubertal maturation and timing as well as diagnostic methods for assessing psychopathology.

More recently, we have also demonstrated that pubertal timing effects are maintained into young adulthood. Specifically, in the OADP, we found that early maturation and late maturation in girls were both associated with higher rates of MDD during the high school years. Notably, early-maturing girls also had elevated rates of many symptoms linked to depression at that time (Graber et al., 1997). In young adulthood, although late maturation was no longer associated with higher prevalence of MDD, early maturation effects were maintained, such that women who had been early maturers continued to have higher lifetime prevalence rates of MDD compared to on-time or late maturers (Graber et al., 2004).

For boys, pubertal timing has more often been linked to elevated symptoms rather than disorders, with early maturation associated with higher internalizing symptoms (e.g., Ge, Conger, & Elder, 2001; Graber et al., 1997), as well as externalizing behaviors (e.g., Ge et al., 2001). Late maturation for boys also seems to confer some risk for psychopathology as reflected in higher rates of disruptive behavior disorders and increased alcohol use and/or abuse in young adulthood (Andersson & Magnusson, 1990; Graber et al., 2004).

Specific links between timing and adjustment within racial groups are just beginning to be studied. For example, Ge, Brody, Conger, and Simons (2006) reported similar effects of early maturation among both boys and girls on internalizing and externalizing behaviors and symptoms in a community sample of African American young adolescents. Recent studies have found comparable effects of early maturation on externalizing behaviors in African American and Latino youth (e.g., Lynne, Graber, Nichols, Brooks-Gunn, & Botvin, 2007). While limited, the emerging evidence indicates that early maturation is a risk for adjustment problems for European American, African American, and Latino youth and possibly other groups. Given the more pervasive and severe risk for internalizing problems among early-maturing girls, understanding the mechanisms or pathways for these effects may be particularly important in understanding why gender differences in internalizing disorders emerge in adolescence.

The aforementioned tests of simultaneous transition models have found that early maturing girls were at risk for increases in depressive symptoms across adolescence, as they were more likely than boys to experience rapid pubertal change at the same time as a school transition. In addition, two main hypotheses have been the foundation for these studies (Brooks-Gunn, Petersen, & Eichorn, 1985). An early maturation hypothesis suggests that being earlier than one's peers results in individuals entering into more "adultlike" behaviors commensurate with their physical appearance but prior to developing the skills or competencies needed to negotiate these situations. The result is that early maturers, potentially both girls and boys, may engage

in more problem behaviors and experience greater distress during adolescence. However, no studies have actually examined this hypothesis; that is, no studies of pubertal timing have intensively assessed social cognitive abilities, social skills, and emotion regulation skills. Yet, as previously discussed, research suggests that these types of skills (and deficiencies in them) are important in the development of internalizing problems.

A separate hypothesis suggests that being "out-of-sync" with one's peers results in poor adjustment; in this case, early maturing girls and late maturing boys should be at risk for negative outcomes as their development is the most off-time given the relative gender difference in the timing of puberty. A caveat of this general hypothesis is that early maturing girls will seek out individuals more like themselves (e.g., older peers) and engage in problem behaviors at young ages (Stattin & Magnusson, 1990). As we and our colleagues have discussed previously (Graber & Sontag, 2006), tests of this hypothesis have focused on early maturing girls' associations with deviant peers or associations with boys, presumably romantic partners, both early maturing girls' and boys' association with deviant peers, and effects on externalizing behaviors (e.g., Ge, Brody, Conger, Simons, & Murry, 2002). For example, Marín, Coyle, Gómez, Carvajal, and Kirby (2000) found that girls with older boyfriends had more unwanted sexual advances, had more friends who were nonvirgins, and were more likely to have had intercourse than other girls. These experiences may or may not lead to internalizing problems (see subsequent section).

In the OADP, along with the striking rates of depressive and substance use disorders found among early maturing girls, these girls also report lower levels of social support from family and friends in mid-adolescence and young adulthood (Graber et al., 1997; Graber et al., 2004). Women who had been early maturers had higher rates of traits reflective of antisocial personality disorder at age 24, a disorder associated with serious impairments in interpersonal relationships. Deficits in social interactions may be a pathway for girls to develop internalizing problems and possibly other problems; early maturation may be one component of this pathway. This hypothesis requires more detailed examinations of the quality of relationships among early-maturing girls as well as the type of individuals with whom they have relationships.

In fact, hypothetically, much of the effect of early timing on disorder in girls may be accounted for by exposure to unhealthy family relationships. Specifically, several studies have now demonstrated that stressful home environments and family interactions actually predict earlier maturation in girls (Belsky et al., 2007; Ellis & Garber, 2000, to name a few). These same family factors have been associated with the development of vulnerabilities for depression (Graber, 2008). Furthermore, youth who enter adolescence with poorer quality family relationships are likely to continue to have low-warmth and high-conflict relationships and higher risk for psychopathology during adolescence (Steinberg, 2001). It may be that both pubertal timing and family relationships have unique contributions as well as a combined contribution to the development of internalizing problems. As yet, no studies have fully examined family factors in childhood and adolescence in order to determine if early timing effects on depression are mediated by family relationships or other factors (Graber, 2008).

Genetic Factors and Family Aggregation

As with many disorders, genetic factors have been identified as important risk factors for internalizing problems. Note that genetic factors (vulnerabilities) confer greater risk for disorder via their interaction with environmental forces. Birmaher and colleagues (1996) suggest that, looking across twin and adoption studies, about 50% of the variance in mood disorders (e.g., MDD, dysthymic disorder, and bipolar disorder) is accounted for by genetic

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similarity. And children of depressed parents are about three times more likely to have a lifetime history of MDD even accounting for differences across study designs (Strober, 2001). Fewer family history, twin, or adoption studies have focused on anxiety problems and disorders in childhood or adolescence (Vasey & Ollendick, 2001); however, twin studies of adults find genetic similarity constitutes risk for GAD and other anxiety disorders (see Kendler, 2001, for a review). Interestingly, the impact of having a depressed parent is not confined to MDD among their offspring but is also associated with increased rates of anxiety and other disorders in offspring. Kendler (1995) has hypothesized that such findings may be explained by a genetic factor that is shared by depression and anxiety such that other environmental factors interact with or exacerbate this risk, resulting in one or the other disorder. Again, this inference is consistent with the position that anxiety and depression may not be distinct phenomena but rather have substantial shared characteristics, in terms of both their symptoms and their underlying genetics.

Genetic similarity and other familial factors may play a unique role in timing of onset of disorder, in particular, onset prior to adulthood (Jaffee & Poulton, 2006). In part, family context or parental behaviors may be more salient when individuals live with family members. At the same time, genetic similarity may be predictive of a vulnerability that results in earlier development of disorder. Drawing on the Virginia Twin Study of Adolescent Behavioral Development, Silberg and her colleagues (1999) found that genetic similarity was not important in explaining variance in symptoms in prepubertal children. Rather, genetic similarity was only significantly associated with depressive symptoms of pubertal and postpubertal adolescents. Consistent with other studies, Silberg et al. (1999) reported that negative life events were predictive of depressive symptoms in both boys and girls. But, among girls, depressive symptoms increased with age, even among girls who did not experience negative life events. Interestingly, genetic similarity accounted for variance in the reporting of negative life events. Thus, there is evidence for genetic similarity in twins with respect to vulnerability to report or to experience negative life events that covaries with depressive symptoms. Although genetic similarity likely plays a role in depression across the life span, this study points to the salience of such similarity in increases in depression at puberty, especially among girls.

Much of the literature on genetic factors has relied on family aggregation and twin studies which provide estimates of genetic influences but have several limitations. In contrast, with advances in the field of molecular genetics and drawing on animal models, recent studies have identified specific gene–environment interactions that may be linked to different disorders (Moffitt, Caspi, & Rutter, 2006). In the Dunedin project, a recent study examining the impact of gene–environment interactions on depression found that a functional polymorphism in the promoter region of the serotonin transporter (5-HTT) gene moderated the influence of stressful life events on depression (Caspi et al., 2003). This particular gene is thought to moderate serotonergic responses to stress. In this study, childhood maltreatment was more strongly linked to subsequent disorder in adolescence and young adulthood among individuals with the short allele of the gene in comparison to individuals homozygous for the long allele. Several, but not all, studies have replicated links between depression and the 5-HTT transporter gene (Moffitt et al., 2006).

The notion that genetic factors play a role in vulnerabilities via gene–environment interactions does not fit with a compartmentalized approach to predictors of disorder. Indeed, environmental influences have genetic components, especially when the environments include parents. Characteristics of parents, such as their own temperament or vulnerabilities to environmental stressors, influence not only the parent's likelihood of developing an

internalizing problem but also their parenting behaviors. A fuller delineation of the processes through which genetic factors translate into vulnerabilities for internalizing symptoms and problems is needed and will likely emerge in the next several years (Collins et al., 2000).

Family Relations and Context

As indicated beyond linear genetic processes, family relationships, events, and interactions, as well as broader family contextual factors, have all been linked to changes in internalizing symptoms in childhood and adolescence. As noted, maltreatment in the family context interacts with genetic risk, and socialization behaviors of parents may influence the development of cognitive attributions and emotion regulation. The nature of parent–child interactions and attachments has been the focus of numerous theories of internalizing disorders (Bowlby, 1980; Zahn-Waxler et al., 2000). Freudian psychoanalytic theory identified the source of both anxiety and depression among adults as problems in early childhood relationships with parents.

In the work of Bowlby and Ainsworth on attachment, early parent–child interactions, as tapped by caregiver sensitivity and consistency, shape the child's expectations for the behavior of others and are the basis for a working model of relationships (see Thompson, 2006, for a review). Working models are not unchanging during development; subsequent changes in parental behaviors will also influence the child's working model over the course of development. During adolescence, nonfamilial relationships take on greater importance with initiation of romantic relationships and increasing intimacy in friendships (see chapter 3, vol. 2 of this *Handbook*); hence, working models may play a more significant role in social interactions at this time. In particular, working models defined by insecurity have been associated with depression in adolescents and adults (Garber, 2000). As mentioned, Suomi (1999) found that reactive monkeys reared with less responsive or less nurturing caregivers were more likely to develop internalizing-like symptoms in response to stress. These findings suggest an interaction between parenting behaviors and temperament in predicting offspring behavior. Interestingly, there is no indication that a "working model" was a necessary conduit for this association. Specifically, it is unlikely that rhesus monkeys are cognitively capable of forming working models of relationships; rather, poor parenting behaviors influenced the stress system or emotion regulation skills, resulting in a vulnerability for internalizing problems.

Both increases in internalizing symptoms and depressive disorders in adolescence have been associated with increased family conflict, lower family warmth, parental rejection, and prior and concurrent maltreatment or abuse (Birmaher et al., 1996). Notably, Rueter, Scaramella, Wallace, and Conger (1999) examined the impact of parent–child conflict on internalizing symptoms and disorder over time. Both internalizing symptoms and parent–child conflict in early adolescence (age 12–13) predicted changes in internalizing symptoms and reports of history of disorder by young adulthood (ages 19–20). Specifically, prior parent–child disagreement influenced internalizing disorders via their indirect effect on subclinical symptoms rather than via a direct path to later disorder. In this study, gender differences in pathways were not assessed, although the rates of anxiety and depressive disorders demonstrated expected gender differences.

Other longitudinal projects have demonstrated that maternal adjustment and marital discord or divorce result in increases in internalizing symptoms over early to late adolescence in rural and urban youth (Crawford, Cohen, Midlarsky, & Brook, 2001; Forehand, Biggar, & Kotchick, 1998). Forehand and colleagues looked at the impact of multiple family risk factors (e.g., divorce, maternal depressive symptoms, mother–child relations) and found that when more than three risk factors were present during early

adolescence, depressive symptoms were dramatically higher in late adolescence/young adulthood. However, the presence of more than three family risk factors also predicted poorer academic achievement or attainment by young adulthood. Steinberg and Avenevoli (2000) argue that findings such as these indicate a lack of specificity in the link between contextual stress and psychopathology. Also, gender was not consistently associated with cumulative risks; hence, risks were not explanatory of gender differences in this study. In contrast, Crawford and colleagues (2001) focused on the effects of maternal distress, as indexed by internalizing symptoms and marital discord. In this case, expected gender differences in internalizing symptoms emerged around age 13–14 and were predicted by maternal distress.

As noted, links between parental distress and adolescent internalizing symptoms may, in part, be accounted for by genetic similarity. At the same time, socialization and other factors may also play a role. Kessler and McLeod (1984) have suggested that women are more vulnerable than men to "network events," that is, stressful events that happen in one's network of close relationships. Gender differences in sensitivity to interpersonal stress have been attributed to biologically driven changes in affiliative relationship patterns (i.e., females display a stronger preference for intimacy and responsiveness within relationships). Specifically, research with animal models has shown that increases in the production of oxytocin triggered by female pubertal hormones are associated with the development of female affiliative behaviors (Cyranowski, Frank, Young, & Shear, 2000). This potential developmental process would be consistent with the age when gender differences emerged in the Crawford et al. (2001) study and with the finding that stressful life events influence depressive symptoms and have a genetic component (Silberg et al., 1999). Perhaps genetic similarity in the disposition to experience or to report stressful events is limited to stressful events within close social networks, such as family and friends. Alternatively, vulnerability to network events may in part be associated with who is in one's network, in that adolescent girls who develop internalizing problems may be more likely to have female network members who share a genetic vulnerability to internalizing problems (i.e., mother, sister, etc.) and hence more distress in the network.

One concern about family influences on adolescent internalizing problems is that effects may not be unidirectional. Rather, having an adolescent with serious internalizing problems places additional strains on families and may result in increased conflict between parents and children, changes in expressions of warmth, communication problems among family members, and strain in the marital relationship of parents (Birmaher et al., 1996; Garber, 2000). Interestingly, Reuter and colleagues (1999) examined bidirectional influences of family conflict and internalizing symptoms over time and did not find support for internalizing symptoms predicting family conflict; however, they did find that conflict had an indirect effect on symptoms and disorder over time. In research on symptoms of eating disorders, quality of family relations predicted increased symptoms in young adolescents (Archibald, Graber, & Brooks-Gunn, 1999), and increasing symptoms of disturbed eating predicted declines in quality of family relations among mid- to late adolescents in separate studies that tested bidirectional influences (Archibald, Linver, Graber, & Brooks-Gunn, 2002). Quality of family relations may be more salient to younger adolescents or may have a greater impact on internalizing emotions during this developmental period. Additional studies modeling simultaneous change in family interactions and relationships and internalizing problems across developmental periods are needed.

Finally, stemming from ecological models (e.g., Bronfenbrenner, 1977), studies of family influences on internalizing problems have also examined how broader contextual factors influence children and families. Several studies have found that economic strain and persistent

parental unemployment are predictive of internalizing symptoms among rural, White, and urban African American adolescents, usually via effects on parental mental health or parenting behaviors in parent–child interactions (Conger, Ge, Elder, Lorenz, & Simons, 1994; McLoyd, Jayaratne, Ceballo, & Borquez, 1994). Perceptions of economic hardship by youth, themselves, are also a factor in this process; adolescents who perceive that economic problems exist for the family are more likely to evince increasing internalizing symptoms over time (Conger, Conger, Matthews, & Elder, 1999). As with other family factors, contextual events and experiences do not uniformly predict internalizing symptoms in adolescence. Individual characteristics, such as the use of more problem-solving coping strategies or mastery, may be protective against perceiving strain in the first place (i.e., mastery predicted the likelihood of perceiving economic hardship; Conger et al., 1999) or may mediate effects of strain and family conflict on internalizing symptoms (Wadsworth, Raviv, Compas, & Connor-Smith, 2005).

Often, studies of economic influences on changes in internalizing symptoms during adolescence explain general effects on symptoms, usually well within the normal range of functioning, and have not shed light on why more girls develop these problems or even why increases happen during adolescence. To clarify, many of these studies have documented factors that predict changes for some individuals but not others in internalizing symptoms during adolescence. However, models might fit equally well in predicting changes in internalizing symptoms in childhood.

Peers

The impact of peer relationships on adjustment has been much less studied in adolescence than in childhood. In addition, much of the focus on peer relationships has been on aggression and externalizing problems. At the same time, withdrawal from social relationships and activities is often used as an indicator of impairment among youth experiencing internalizing problems: for example, the diagnostic criteria for GAD and for MDD in the DSM-IV-TR both require that the anxious or depressive symptoms result in significant impairment in social or occupational functioning. Peer interactions and internalizing symptoms likely have bidirectional or transactional influences on each other; that is, poor relationships lead to poor emotional functioning, which in turn leads to poorer quality social interactions, and so on.

With entry into adolescence comes an increased focus on peer relationships and the greater importance of peers in one's daily life. Moreover, the quality of peer relationships changes, with greater focus on intimacy and trust (see chapter 3, vol. 2 of this *Handbook*; Cairns & Cairns, 1994; Rudolph, 2002). As noted, the report of stressful life events increases with age and is predictive of in-creased internalizing symptoms (Brooks-Gunn, 1991; Ge et al., 1994; Silberg et al., 1999). Peer-related events (e.g., a fight with a friend, breaking up with a friend, making a new friend) may account for a significant number of the events that adolescents experience (Brooks-Gunn, 1991), and appear to be more salient to girls than boys as part of the greater selience of social networks. Examination of the emotional impact of negative peer experiences includes victimization, rejection, and poor-quality peer relationships.

Victimization has been linked to poor emotional adjustment, poor school adjustment, and poor relationships with peers in community studies and cross-national research (Deater-Deckard, 2001; Nansel et al., 2004). Because of girls' tendencies to rely heavily on peers for emotional support and intimacy, victimization targeted at damaging one's social reputation and interpersonal relationships (i.e., relational aggression) is likely to create higher levels of stress in girls compared to boys, which in turn may contribute to higher rates of depression in girls (Rudolph, 2002). For example, Storch, Nock, Masia-Warner, and Barlas (2003) found that overt peer victimization was positively associated with depressive symptoms and

aspects of social anxiety for both boys and girls, whereas, relational victimization was predictive of internalizing problems for girls only.

Certainly, prior relationship skills and patterns influence how well the challenges of new relationship demands are met by adolescents and may help to identify who will have greater difficulties during adolescence. Rejection by peers is often an indicator of skills. That is, peers may reject other children because the child is aggressive or because the child is withdrawn; however, rejection is also a source of stress and internalizing symptoms. Cross-sectional studies have indicated that, during childhood, peer rejection resulting from social withdrawal, rather than from aggression, has been linked to increased depressive symptoms and possibly disorder (Hecht, Inderbitzen, & Bukowski, 1998; Rubin & Burgess, 2001). In a longitudinal study from childhood into adolescence, Coie, Terry, Lenox, Lochman, and Hyman (1995) reported that boys who were rejected and aggressive in childhood increased in both externalizing and internalizing symptoms during early and mid-adolescence. In contrast, girls who were rejected in childhood regardless of the reason for rejection (withdrawal or aggression) had higher reports of internalizing symptoms in childhood and in early and mid-adolescence. These girls did not show increases in internalizing symptoms, but rather entered adolescence with already elevated symptoms. One implication of this finding is that within any group of adolescent girls with elevated internalizing symptoms, some will have had persistent internalizing problems and others will not have had preadolescent difficulties.

In other longitudinal work, London, Downey, and Bonica (2007) found that peer rejection predicted an increase over time in both anxious and angry expectations about rejection during early adolescence; expectations for future rejection, in turn, were associated with risk for internalizing and externalizing problems. Interestingly, London and colleagues (2007) found that anxious expectations of rejection predicted increases over time in social anxiety and withdrawal, whereas angry expectations predicted decreases in social anxiety. Way (1996), in an ethnographic study of urban high school students, found that negative experiences with peers often resulted in negative emotions (e.g., distrust) about peers and close relationships. Significant periods of isolation or withdrawal from relationships with peers were also reported by several youth. Hence, both negative events and rejection shape adolescents' expectations about relationships, influencing subsequent relationship experiences and internalizing symptoms.

An extensive cross-sectional literature on bullying and victimization also suggests that children without peer supports who are victimized or rejected are more likely to have higher internalizing symptoms (Deater-Deckard, 2001). Both in childhood and in adolescence, better quality of a close friendship buffers negative effects of having few friends or difficulties in larger peer groups (Berndt, Hawkins, & Jiao, 1999). However, adolescents seem to associate with peers who have similar levels of internalizing symptoms (Hogue & Steinberg, 1995); in such cases, friends may reward less effective coping strategies for dealing with problems and maintain elevated symptoms or problems. Research has shown that friend-reported depressive symptoms are associated with increases in adolescent girls' self-reported depressive symptoms over time (Stevens & Prinstein, 2005). In addition, social anxiety and best friend's peer popularity moderate the association between best friend's and self-reported internalizing problems, such that only those adolescents high on social anxiety and those adolescents with popular friends exhibited the peer contagion effect (Prinstein, 2007).

Close peer relationships and peer group membership change frequently during the middle school years, with a tendency for greater stability by late high school (Cairns & Cairns, 1994). The turbulence and quality of peer relationships likely play a role in increases in internalizing problems during adolescence,

especially in early to mid-adolescence. Also, emotion regulation and social cognitive skills influence competence in peer relationships, and are one source of individual differences in who does and does not experience poor peer relationships, and subsequent internalizing problems in the face of changing interpersonal demands. How peer events are experienced also varies based on these factors. Individuals with better skills may have fewer negative peer events (e.g., fight less often) or may deal with the emotional impact of negative events more effectively, either through better regulation skills or the buffering effect of having other positive relationships. Again, the salience of relationships to the emotional experience of adolescent girls coupled with instability of relationships during early adolescence may be important factors in explaining gender differences in internalizing problems (e.g., Rudolf, 2002).

Romantic and Sexual Relationships

The importance of romantic relationships in adolescent development has undergone a surge of attention both in understanding developmental process and in understanding problems and challenges faced by adolescents (e.g., Davila, 2008; Furman, Brown, & Feiring, 1999; Shulman & Collins, 1997). In the case of internalizing problems, the impact of romantic relationships, like peer relationships, often has been considered in terms of stressful life events, such as breaking up with a romantic partner, beginning dating, unrequited interest, and the like.

Although young adolescents, especially girls, strongly endorse the notion that it is desirable to be in love and have a romantic relationship (Simon, Eder, & Evans, 1992), experiences such as breaking up with a partner are stressful, particularly for young adolescents who may have difficulty regulating the strong emotions often coincident with the event. In turn, these negative experiences and emotions may lead to depressive symptoms, possibly even episodes (Joyner & Udry, 2000; Larson, Clore, & Wood, 1999). Although some studies have suggested that positive romantic relationships may protect adolescents against feelings of social anxiety (La Greca & Harrison, 2006), Joyner and Udry (2000) found that initiating romantic relationships during adolescence was predictive of increased depressive symptoms over time among both boys and girls. In addition, becoming involved in a relationship had a larger negative effect on girls' than on boys' depressive symptoms. Negative effects were highest for younger girls who had repeated or continuous involvement in relationships. For boys, declines in school performance accounted for part of the effect of relationships on depressive symptoms, whereas for girls decreases in the quality of relationships with parents did so. The number and stability of relationships reported over time were also important in explaining the negative impact of involvement in romantic relationships, with more relationships and less stability leading to increases in depressive symptoms. Again, a distinctive feature of changes in symptoms for girls was their link to interpersonal factors (in the domain of partners and parents).

In general, gender differences in relationship goals, with girls being more likely than boys to focus on intimacy, make it particularly challenging for young adolescents to have relationships that meet their needs (Maccoby, 1998). Much like their tendencies in peer relationships, girls experience greater stress and internalize their experiences with romantic partners to a greater extent than do boys (Rudolf, 2002). Although empirical evidence on the impact of romantic involvements on adolescent adjustment is limited, parallel research on sexual behaviors sheds some light on this issue (see chapter 14, this volume).

Even though it is frequently noted that managing sexual feelings and interactions may tax emotion and behavior regulation skills, little attention has been given to this aspect of sexual behavior. (Actually, there is an enormous literature on the sexual activity of adolescents, but only recently has that literature been linked to relationships or with

internalizing problems.) The emotional challenges of regulating sexual feelings and interactions may, in part, be embedded within the other emotional challenges of romantic relationships. However, it is likely that sexual experiences outside of relationships may also impact emotions and adjustment.

Adolescent girls are more likely than boys to have sexual partners who are older than they are at first intercourse, and are more likely to report that their first sexual experience was unwanted (Terry-Humen, Manlove & Cottingham, 2006). Individuals, especially girls, who have intercourse at younger ages are thought to be at greater risk for a number of sexual health–related problems and are more likely to transition to the next sexual partner more quickly than older girls (AGI, 1994). Again, higher number of relationships and relationships at younger ages have been linked to higher reports of depressive symptoms (e.g., Joyner & Udry, 2000).

As discussed previously, the negative effects reported for early-maturing girls may, in part, be due to their association with older or deviant peers. In fact, Halpern, Kaestle, and Hallfors (2007) reported that having a romantic partner was an important link between early maturation and risk for numerous internalizing symptoms and externalizing behaviors in both girls and boys. However, having an older partner was a unique risk for early maturing girls. As with prior research (e.g., Ge et al., 2002; Stattin & Magnusson, 1990), peers moderated the association between timing and adjustment, and the negative impact was not specific to internalizing problems but to externalizing problems as well.

Managing sexuality, relationships, or the interplay of both may all play a role in increases in internalizing symptoms during adolescence, but unique effects of each factor are difficult to delineate. For example, is the breakup of a relationship more stressful, eliciting more sadness, if the relationship included intercourse? The romantic relationships of adolescents are increasingly likely to include intercourse the longer the relationship lasts (Bearman, 2001). Another example of the challenges posed by relationships can be seen in studies of adolescent responses to sexual infidelity and betrayal in romantic relationships (Feldman & Cauffman, 1999; Thompson, 1994). In some cases, infidelity or betrayal leads to breakups and hence potential increases in internalizing symptoms. In fact, experience of breakups has been linked to suicide attempts among adolescents (Monroe, Rohde, Seeley, & Lewinsohn, 1999). But not all relationships that involve infidelity or betrayal result in breaking off the relationship. In either situation, many adolescents experience strong negative emotions such as sadness, despondency, and disillusionment in response to infidelities (Feldman & Cauffman, 1999; Thompson, 1994). In the narratives of an ethnically diverse sample of girls, Thompson (1994) found that girls who idealized romantic relationships described more fear of rejection and despondency at being "dumped."

Thus, rejection or betrayal in romantic and sexual relationships influences emotion, but as noted in peer relationships, these experiences also impact beliefs about relationships and future behaviors in relationships. In outlining the development of rejection sensitivity, Downey and colleagues (Downey, Bonica, & Rincon, 1999) note that past childhood experiences of rejection, initially from parents and then from peers, result in heightened sensitivity to rejection from romantic partners. Testing this model with young adolescent girls, Purdie and Downey (2000) found expectations of rejection from ambiguous peer and teacher interactions prospectively predicted heightened rejection fears from romantic partners. Rejection-sensitive individuals may shy away from romantic involvements out of increased fearfulness about close relationships or may find themselves seeking such involvements in order to gain acceptance that has previously been lacking. Individuals who seek acceptance may be overly willing to acquiesce in the relationship or place high value on it. In a

study of adolescent couples, Harper, Dickson, and Welsh (2006) found that ability and comfort in voicing one's feelings or opinions partially mediated the association between rejection sensitivity and depressive symptoms in both boys and girls, suggesting that the fear of being rejected encourages adolescents in romantic relationships to stifle their emotions and fear voicing issues or concerns with the relationship. This type of dynamic in a romantic relationship puts both girls and boys at risk for internalizing problems. In addition, rejection-sensitive individuals tend to be highly jealous and hostile in their relationships; these feelings often lead to more conflict in the relationship, which subsequently leads to breakup (Downey et al., 1999). Whereas Downey and colleagues have suggested that rejection-sensitive women may be more likely to experience internalizing problems after a relationship breakup and rejection-sensitive men may be more likely to respond with aggression, recent studies have found that both boys and girls high in rejection sensitivity display greater levels of internalizing problems (Harper et al., 2006).

Our discussion has been based on findings across several literatures that seem to suggest that romantic and sexual relationships may be particularly challenging to adolescents and may be a source of increased internalizing problems for some youth. A recent increase in the number of empirical studies examining not only sexual behaviors but also more psychosocially based aspects of romantic relationships (i.e., rejection sensitivity, attachment, personality, pubertal development, peer relations, etc.) has helped the literature move away from focusing on adolescent romantic relationships as simply a risk for sexual promiscuity, pregnancy, and disease (see chapter 4, vol. 2 of this *Handbook*; Graber & Sontag, 2006). Unfortunately, many studies of sexual behaviors still focus heavily on teen pregnancy and health risks, such as sexually transmitted diseases, without considering interconnections with psychopathology. For example, poorer body image has been associated with fear of abandonment, low rates of condom use during intercourse, and engaging in casual sex among African American girls (Wingood, DiClemente, Harrington, & Davies, 2002). Hence, health risks would be better understood if embedded in a broader developmental context.

Of course, emerging sexual feelings and behaviors as well as romantic interest and relationships are a normative part of adolescent development, and most youth meet these challenges with only temporary emotional upheaval. The importance of romantic and sexual experiences, the new emotion regulation challenges elicited by these experiences, the dramatic increase of these experiences around the time that internalizing problems increase (especially rates of MDD), and the potential for relationship experiences to account for gender differences in internalizing problems would all suggest that more nuanced investigations of how adolescents regulate relationships are warranted. Models, such as that of Downey on rejection sensitivity, that integrate childhood experiences, development of gender differences in response to these experiences, and interactions of vulnerabilities with challenges at adolescence are likely to be informative for understanding who is at risk for internalizing problems during adolescence.

TREATMENT OF INTERNALIZING DISORDERS

Given the severity and chronicity of internalizing problems and disorders during adolescence and the extension of these problems into adulthood, a large body of research has examined the efficacy of treatment for MDD in children and adolescents. We provide a brief overview here. Based on findings from efficacy studies, there are currently three empirically supported treatment methods for child and adolescent MDD—namely, interpersonal therapy, cognitive behavioral therapy (CBT), and antidepressant treatment (Wagner, 2003; Zalsman, Brent, & Weersing, 2006). Research examining treatment efficacy has found high response rate to

placebo or brief supportive treatment in less clinically severe cases of MDD among adolescents, suggesting that it would be reasonable to begin treatment of mild depression with family education, supportive counseling, case management, and problem solving (Zalsman et al., 2006). However, for more persistent or severe depression, antidepressants, CBT, and interpersonal therapy have been shown to be more effective (Zalsman et al., 2006).

Interpersonal psychotherapy focuses on developmentally appropriate issues for adolescents, such as separation from parents, authority conflicts, peer pressures, and interpersonal relationships. In efficacy studies, interpersonal therapy has been effective at reducing depressive symptoms and improving social functioning and problem solving in adolescents (Wagner, 2003). At the same time, the majority of research has focused on the efficacy of CBT and antidepressants. CBT focuses on modifying cognitions, assumptions, beliefs, and behaviors as a way to reduce symptomatology. Despite being hailed as one of the more effective treatments for MDD in adolescents (Wagner, 2003), a recent meta-analysis of the effects of psychotherapy for child and adolescent treatment found that CBT trials were no more effective than non-cognitive-based treatments (e.g., relaxation training; Weisz, McCarty, & Valeri, 2006). In addition, a comparison of the SSRI fluoxetine, CBT, and their combination in the Treatment for Adolescent Depression Study (TADS, 2004) found that adolescents treated with CBT alone fared no better than those in the placebo condition, whereas adolescents treated with fluoxetine alone demonstrated outcomes superior to those in both the placebo and CBT alone condition. Using a combination of fluoxetine and CBT produced the most positive treatment response, indicating that combination treatments may be a very potent treatment tactic.

A particular challenge for parents and youth is that in many communities, specialized treatment of child and adolescent depression, such as CBT or interpersonal therapy, is not readily available. Although these communities often have treatment resources available for adults, the more generic types of psychotherapy that are often practiced with adults may not be helpful in the treatment of youth depression (Zalsman et al., 2006). When specialized psychotherapy is not available, an antidepressant is often recommended as a first-line treatment.

In fact, the first-line treatment for children with moderate to severe depressive symptoms or with impairment in functioning is pharmacotherapy, either alone or with CBT or interpersonal therapy. In general, SSRIs are effective for treating both children and adolescents with MDD as previously noted (Singh et al., 2007). Although there are a variety of SSRIs available in the market, fluoxetine is the only Food and Drug Administration (FDA)-approved antidepressant to treat MDD in children younger than 7 years of age (NIMH, 2008). SSRIs are well tolerated in adolescents with fewer side effects than the previous generation of drugs (e.g., tricyclic antidepressants) commonly used to treat MDD in adults. However, SSRIs have been shown to increase the risk for suicidal thoughts/behaviors, suggesting to clinicians and parents that these drugs be prescribed with caution to adolescents prone to these symptoms and that careful monitoring should ensue (Singh et al., 2007); that is, simply prescribing drugs with periodic monitoring by a psychiatrist or pediatrician may be risky in comparison to drug therapy plus regular psychotherapy sessions. With respect to other antidepressant drug options often employed in adult populations, studies find no difference in response rates between other antidepressants and placebo for the treatment of MDD in adolescents (Singh et al., 2007).

As noted, although studies have shown that SSRIs produce decreases in depressive symptoms in adolescents, the fact that many of the other drugs used to treat adult depression do not produce similar results in children suggests that the biology of depression (i.e., the role of neurotransmitters, dysregulations in brain systems) may differ between adolescents

and adults. In addition, while studies have been conducted on treatment efficacy, especially clinical trials for medication, for MDD, very few studies have examined treatment protocols for less common disorders, such as PBPD, even though pharmacological treatments are actively prescribed for children and adolescents by a range of health practitioners.

Another concern for effective intervention is that engaging adolescents with mild or severe depression in treatment has proven to be difficult. The National Institute of Mental Health (NIMH) estimates that approximately 1 in 10 individuals under the age of 18 suffer from mental illness severe enough to cause impairment, but only 1 in 5 of these children and adolescents receives treatment (NIMH, 2004). Often, adolescents or their parents are reluctant to seek treatment, or, as noted, specialized treatment for youth is not available.

Because of the large number of adolescents who do not receive treatment for internalizing problems and the high costs associated with pediatric depression (i.e., increased concurrent occurrence of substance abuse, academic problems, aggression, physical health problems, etc.), interest in prevention programs has grown rapidly in the past 10–15 years (Horowitz & Garber, 2006). Programs designed to prevent the emergence of symptomatology typically fall within three broad categories: universal, selective, or indicated programs. *Universal* programs are administered to all members of a target population and typically involve large-group presentation or curricula that focus on cognitive restructuring, anxiety management, relaxation, problem-solving skills, emotion-focused coping, anticipating consequences, and assertiveness. Generally, universal programs are thought to be advantageous because of their ability to avoid the stigma of singling out individuals for treatment and their relatively low dropout rates. *Selective* interventions target individuals at elevated risk for depression (e.g., adolescents from divorced families, low SES, etc.) and often target multiple outcomes in addition to reducing depression. Finally, *indicated* interventions are targeted at individuals who already demonstrate subclinical symptoms of depression. Similar to universal programs, most selective and indicated programs utilize some form of cognitive behavioral techniques.

While studies of prevention programs have shown a reduction in depressive symptoms in the short term, few studies have shown a reduction in the persistence of depressive disorder or symptoms (Horowitz & Garber, 2006; Merry, 2007). Unfortunately, most prevention programs do not follow up with participants beyond a 6- or 12-month period. In addition, many of the studies rely on self-report measures of depressive symptoms, which may not correspond to clinical assessment. Research comparing universal programs to more targeted programs has found that more targeted programs (both selective and indicated) are generally more effective at maintaining low levels of depression or reducing existing symptoms (Horowitz & Garber, 2006; Merry, 2007). However, Horowitz and Garber (2006), as well as others, caution that the apparent superiority of more targeted programs over universal may be partly explained by the differences in level of symptoms found in the control groups. That is, because universal programs target a normative sample with generally low initial levels of depression, effect sizes of change will ultimately be smaller.

Despite limitations of the programs, research suggests that more targeted prevention programs may be more cost-effective, practical, and beneficial in the long run than universal programs. Notably, Post, Leverich, Xing, and Weiss (2001) identify several reasons that treatment and prevention of internalizing disorders during adolescence should be a focus in research and practice. First, as we have noted, episodes of MDD are frequently untreated among adolescents. Second, there is evidence that recurrent episodes of disorder become harder to treat. And, finally, early treatment of disorders may be protective against recurrent episodes later in development. Thus, better

identification of disordered youth and better access to services for them would substantially improve mental health in subsequent cohorts of adults.

IMPLICATIONS AND CONCLUSIONS

In the course of this chapter, the goal has been to consider the nature of internalizing symptoms, problems, and disorders during adolescence, as well as variations on the diathesis-stress model that explain why internalizing problems occur, why they increase during adolescence, and why more girls than boys experience these problems. As noted previously, it has been questioned whether subclinical problems are of importance. Clearly, in the developmental perspective taken here and elsewhere, subclinical problems are important as a pathway to disorder for some individuals. However, the literature on disorder and subclinical problems is incomplete in several areas. The extent to which findings regarding subclinical problems also apply to the develop of disorder or the extent to which dysregulations observed in disordered individuals also occur to a lesser, or even the same, extent in individuals with subclinical problems has often not been assessed.

Although rates of internalizing problems, especially MDD, change dramatically during adolescence, examination of development prior to adolescence in order to understand vulnerabilities and risk factors that influence the trajectory for adjustment is essential. Internalizing problems occur across the life span and exhibit much continuity, impacting later development in adulthood and being highly predictive from childhood symptomatology and experiences. The present discussion was limited to a single segment of the life course (albeit an important one). Internalizing symptoms, in particular depressive symptoms, seem to follow a curvilinear trend over the life span with highest symptom reports in adolescence and young adulthood, and rates of disorder being lowest among older adults (over age 65; Karel, 1997).

There is compelling evidence that preexisting biological and psychological vulnerabilities are an important factor affecting who develops internalizing problems during adolescence. Notably, socializing experiences and genetic factors interact in the development of vulnerabilities, and both contribute to the establishment of vulnerabilities prior to and during adolescence. Whereas different models place different emphasis on vulnerability versus stress, more attention has been given to unique stressors that may emerge during adolescence rather than determining how vulnerabilities change at this time. This trend is shifting, though, as evidenced by volumes devoted to specific constructs, how they develop, and potentially become vulnerabilities for internalizing problems; for example, Allen and Sheeber (2008) focused on emotional development and regulation as vulnerabilities for depression during adolescence.

Explaining why more girls experience internalizing problems has long been a focal point in research in this area and the mechanisms underlying this effect are emerging. Certainly, as Nolen-Hoeksema (1994) has asserted, there are unique challenges of adolescence and puberty that may exist for girls more so than for boys. In addition, studies of adolescent relationships have been particularly relevant to the issue of gender differences. Emotional responses to relationships and regulation of emotion or coping are also particularly salient to girls' experiences. Additional studies of relationships, how they are experienced (both psychologically and physiologically), and why some individuals may have particular difficulties with interpersonal relationships (e.g., early-maturing girls) would likely illuminate not only why more girls have internalizing problems but also why some boys experience these problems as well.

Across the correlates and possible predictors of internalizing problems discussed, it is clear that recent studies have been more integrative and have included multiple factors. Such approaches have made it feasible to compare

relative influences of predictors on internalizing problems and their relative impact on other problems. No single study will be able to examine in depth each of the factors discussed in this review, but focused investigations that study pieces of different models and make comparative tests of models will continue to advance this area of research. Whereas several new findings have emerged in the past few years and certainly since the original studies of adolescence and adolescent disorder were begun, there are still numerous gaps in the literature, especially in terms of integration of constructs.

In concluding, it is probably useful to highlight what was not considered in this review. This review did not include all studies of internalizing problems and disorders but rather drew upon prior reviews and recent studies that seemed to advance the areas of interest here (i.e., those most salient to adolescence and individual differences). More importantly, this chapter provided only a brief discussion of prevention and treatment of problems and disorders. Certainly, the primary reason for understanding why these problems occur is to prevent them or treat them more effectively. Fortunately, several reviews consider the advances that have been made in treatment and prevention (e.g., Horowitz & Garber, 2006; Lewinsohn et al., 1998). Unfortunately, little or no research focuses on what treatments are actually being used in real-world settings versus those that are studied via clinical trials, especially for understudied disorders such as PBPD.

From the wealth of empirical evidence that has amassed in recent years, internalizing problems and disorders among adolescents are certainly a serious health concern, and these problems merit the attention they have been given and will likely continue to receive. At the same time, a fuller understanding of vulnerability and stress during adolescence would provide specific information for prevention programming. Whereas problems such as substance use initiation, use, and abuse have widespread prevention programming often mandated by state or federal law, internalizing problems receive much less policy attention. Prevention measures focused on internalizing problems would likely yield societal benefits equal to or greater than those hoped for in other health domains.

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