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Book

CHAPTER 5

Child and Adolescent Depression

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DEFINING CHILD AND ADOLESCENT DEPRESSION

Joey is a 10-year-old boy whose mother and teacher have shared their concerns about his irritability and temper tantrums displayed both at home and at school. With little provocation, he bursts into tears, yells, and throws objects. In class, he seems to have difficulty concentrating and seems easily distracted. Increasingly shunned by his peers, he plays by himself at recess—and at home, spends most of his time in his room watching TV. His mother notes that he has been sleeping poorly and has gained 10 pounds over the past couple of months from constant snacking. A consultation with the school psychologist has ruled out learning disabilities or attention-deficit/hyperactivity disorder (ADHD); instead, she says, he is a deeply unhappy child who expresses feelings of worthlessness and hopelessness—and even a wish that he would die. These experiences probably began about 6 months ago when his father—divorced from the mother for several years—remarried and moved to another town, where he spends far less time with Joey.

Diagnostic Criteria

The *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013) provides essentially the same criteria for major depressive disorder (MDD) for both adults and children. The criteria are shown in Table 5.1. Persistent depressive disorder (dysthymic disorder) is a diagnosis of chronic, mild to moderate depressive (or for children, irritable mood) symptoms, with a duration of at least 1 year (in adults, duration is at least 2 years). With the new category of persistent depressive disorder, DSM-5 differs from DSM-IV, which defined chronic major depression and dysthymic disorder in separate sections; DSM-5 now emphasizes *persistence* rather than severity. Even the fairly mild symptoms typical of persistent depressive (dysthymic) disorder beginning in childhood or adolescence do not mean that it is a relatively benign condition, however. Such chronic symptoms commonly predict the development of major depressive episodes (MDEs), and may predict a long-term course with significant psychosocial impairment—especially if the symptoms are associated with familial depression and poor parent–child relationships, as is often the case (Klein, Shankman, & Rose, 2008).

In recognition that irritability is a common expression of distress in depressed youngsters (as shown in the case of Joey), DSM-5 specifies that irritable mood may be substituted for depressed mood. However, irritability occurring in MDEs or persistent depressive (dysthymic) disorder is to be distinguished from a new DSM-5 depressive disorder, disruptive mood dysregula-

TABLE 5.1. DSM-5 Diagnostic Criteria for Major Depressive Disorder

A. Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.
Note: Do not include symptoms that are clearly attributable to another medical condition.

1. Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad, empty, hopeless) or observation made by others (e.g., appears tearful). (**Note:** In children and adolescents, can be irritable mood.)
2. Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation).
3. Significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day.
(**Note:** In children, consider failure to make expected weight gain.)
4. Insomnia or hypersomnia nearly every day.
5. Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down).
6. Fatigue or loss of energy nearly ever day.
7. Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick).
8. Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others).
9. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.

B. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The episode is not attributable to the physiological effects of a substance or to another medical condition.

Note: Criteria A–C represent a major depressive episode.

Note: Responses to a significant loss (e.g., bereavement, financial ruin, losses from a natural disaster, a serious medical illness or disability) may include the feelings of intense sadness, rumination about the loss, insomnia, poor appetite, and weight loss noted in Criterion A, which may resemble a depressive episode. Although such symptoms may be understandable or considered appropriate to the loss, the presence of a major depressive episode in addition to the normal response to a significant loss should also be carefully considered. This decision inevitably requires the exercise of clinical judgment based on the individual's history and the cultural norms for the expression of distress in the context of loss.

D. The occurrence of the major depressive episode is not better explained by schizoaffective disorder, schizophrenia, schizophreniform disorder, delusional disorder, or other specified and unspecified schizophrenia spectrum and other psychotic disorders.

E. There has never been a manic episode or a hypomanic episode.
Note: This exclusion does not apply if all of the manic-like or hypomanic-like episodes are substance-induced or are attributable to the physiological effects of another medical condition.

Coding and Recording Procedures

The diagnostic code for major depressive disorder is based on whether this is a single or recurrent episode, current severity, presence of psychotic features, and remission status. Current severity and psychotic features are only indicated if full criteria are currently met for a major depressive episode. Remission specifiers are only indicated if the full criteria are not currently met for a major depressive episode.

In recording the name of a diagnosis, terms should be listed in the following order: major depressive disorder, single or recurrent episode, severity/psychotic/remission specifiers, followed by as many of the following specifiers without codes that apply to the current episode.

Specify:

With anxious distress
With mixed features
With melancholic features

(continued)

TABLE 5.1. *(continued)*

With atypical features
With mood-congruent psychotic features
With mood-incongruent psychotic features
With catatonia
With peripartum onset
With seasonal pattern

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tion disorder, which was intended to provide an alternative to the excessive diagnosis of bipolar disorders in children when the presentation is marked by severe and persistent temper outbursts and irritability rather than classic episodic mood changes (for further discussion, see Youngstrom & Algorta, Chapter 6, this volume). Disruptive mood dysregulation disorder in children is defined by pronounced and frequent temper outbursts, with rage, aggression, and persistently angry mood (see Table 5.2). Evidence of its validity as a depressive disorder is scant at present, however, and critics have argued that it may not be either distinguishable from oppositional defiant disorder or conduct disorder, or predictive of a depressive course (e.g., Axelson et al., 2012).

TABLE 5.2. DSM-5 Diagnostic Criteria for Disruptive Mood Dysregulation Disorder

A. Severe recurrent temper outbursts manifested verbally (e.g., verbal rages) and/or behaviorally (e.g., physical aggression toward people or property) that are grossly out of proportion in intensity or duration to the situation or provocation.
B. The temper outbursts are inconsistent with developmental level.
C. The temper outbursts occur, on average, three or more times per week.
D. The mood between temper outbursts is persistently irritable or angry most of the day, nearly every day, and is observable by others (e.g., parents, teachers, peers).
E. Criteria A–D have been present for 12 or more months. Throughout that time, the individual has not had a period lasting 3 or more consecutive months without all of the symptoms in Criteria A–D.
F. Criteria A and D are present in at least two of three settings (i.e., at home, at school, with peers) and are severe in at least one of these.
G. The diagnosis should not be made for the first time before age 6 years or after age 18 years.
H. By history or observation, the age at onset of Criteria A–E is before 10 years.
I. There has never been a distinct period lasting more than 1 day during which the full symptom criteria, except duration, for a manic or hypomanic episode have been met.
Note: Developmentally appropriate mood elevation, such as occurs in the context of a highly positive event or its anticipation, should not be considered as a symptom of mania or hypomania.
J. The behaviors do not occur exclusively during an episode of major depressive disorder and are not better explained by another mental disorder (e.g., autism spectrum disorder, posttraumatic stress disorder, separation anxiety disorder, persistent depressive disorder [dysthymia]).
Note: This diagnosis cannot coexist with oppositional defiant disorder, intermittent explosive disorder, or bipolar disorder, though it can coexist with others, including major depressive disorder, attention-deficit/hyperactivity disorder, conduct disorder, and substance use disorders. Individuals whose symptoms meet criteria for both disruptive mood dysregulation disorder and oppositional defiant disorder should only be given the diagnosis of disruptive mood dysregulation disorder. If an individual has ever experienced a manic or hypomanic episode, the diagnosis of disruptive mood dysregulation disorder should not be assigned.
K. The symptoms are not attributable to the physiological effects of a substance or to another medical or neurological condition.

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Developmental differences in the expression of depressive symptoms have been noted, even if these are not codified in the formal diagnostic criteria. As summarized by Avenevoli, Knight, Kessler, and Merikangas (2008; see also Rao & Chen, 2009), somatic complaints are more common among younger samples, who also express less subjective dysphoria and hopelessness; hypersomnia increases during adolescence, and appetite decreases (in girls). Depressed boys are at the greatest risk of suicidal behaviors in late adolescence, whereas girls are at the highest risk during middle adolescence. Yorbik, Birmaher, Axelson, Williamson, and Ryan (2004) compared the symptoms of nearly 900 depressed children and adolescents, and found that depressed adolescents exhibited significantly more fatigue, hypersomnia, suicidal thoughts and attempts, hopelessness/helplessness, and weight loss than children.

It is possible that additional research on developmental expressions of depression will suggest further age-appropriate modifications of the diagnostic criteria. For example, a longitudinal study of MDD in preschoolers found that although the DSM-IV criteria validly defined a group of young children with depression and homotypic continuity into early childhood (Luby, Si, Belden, Tandon, & Spitznagel, 2009), the minimum duration and frequency criteria might not necessarily apply. Children who met full symptom criteria but not duration and frequency did not differ in severity, impairment, or risk of MDD 2 years later (Gaffrey, Belden, & Luby, 2011). Further study of developmentally relevant modifications is needed, and potential alterations may be especially important for significant but subclinical cases that might otherwise not be identified.

Like adult depression, childhood depression sometimes includes psychotic symptoms and endogenous (melancholic) features indicative of severe depression. However, the symptom manifestations that have attracted most attention among children and adolescents are suicidal thoughts and actions, which are commonly but not exclusively associated with depressive disorders (see Cha & Nock, Chapter 7, this volume). The topic has attracted widespread attention for two reasons. First, the U.S. Food and Drug Administration issued a "black box" warning of suicidality as an alleged side effect of antidepressant medications among children, adolescents, and young adults; a discussion of this is beyond the scope of this chapter (but see *www.nimh.nih.gov/health/topics/child-and-adolescent-mental-health/antidepressant-medications-for-children-and-adolescents-information-for-parents-and-caregivers.shtml*). Second, suicidality certainly underscores the severity and lethality of depressive disorders, reminding us that these disorders are serious problems and not merely expressions of youthful turmoil. A review by Bridge, Goldstein, and Brent (2006) notes that suicidal ideation is very common in adolescence with reported point prevalence rates of 15–25%, whereas actual suicide attempts occur in 1–4% of adolescent males and 1.5–10% of females. Completed suicides increase in frequency from childhood to older adolescence, and are considerably higher in males than in females (e.g., 17% for males vs. 3% for females among older adolescents in the United States). Depressive disorders appear to be present in approximately 40% of completed suicides, and even higher rates are seen in youth for whom depression is comorbid with substance use and disruptive behavior disorders (Bridge et al., 2006). Rates of depression have been reported as 40–80% among those who attempt suicide (Cash & Bridge, 2009). In samples of clinically referred youth with depressive disorders, 85% report suicidal ideation, and 32% make a suicide attempt during adolescence or young adulthood (Kovacs, Goldston, & Gatsonis, 1993).

Continuity of Depression Severity

Depression in its "clinical" forms is represented by diagnostic categories as discussed thus far, but DSM classifications, despite their value in improving reliability and communicability, have the disadvantage of implying that individuals either do or do not "have" the disorders in question. In the context of depression, taxometric analyses of the latent structure of DSM-IV MDD symptoms in a sample of 845 youth (ages 9–17) suggested that depression is continuously, rather than categorically, distributed for both children and adolescents and boys and girls (Hankin, Fraley, Lahey, & Waldman, 2005). These authors recommend dimensional assessment of the severity of depression in order to fully capture the phenomena. Importantly, subsyndromal or subclinical depressions that fall short of full diagnostic criteria may nevertheless predict negative outcomes and commonly warrant intervention. Among adults, for example, subclinical levels of symptoms and minor depression portend degrees of functional impairment and use of services often approximating those of individuals with MDD (e.g., Backenstrass et al., 2006; Cuijpers, de Graaf, & van Dorsselaer, 2004). In a youth sample, subclinical depression at ages 17–18 predicted

elevated rates of MDD and depressive symptoms and other disorders (as well as treatment seeking) in two subsequent follow-ups to age 25, compared to those without depressive symptoms (Fergusson, Horwood, Ritter, & Beautrais, 2005; see also Shankman et al., 2009).

Many of the studies of depression in children and adolescents reported in this chapter do not rely on diagnostic assessments, but instead are based on elevated scores on a continuous measure of depression severity covering various symptoms of the syndrome. Commonly used self-report scales are the Children's Depression Inventory (Kovacs, 1980) and the Center for Epidemiologic Studies Depression Scale (Radloff, 1977), both well-validated measures of severity of depressive symptoms, although questions arise about their specificity to depression versus more general negative affect. Similar to studies of clinical compared to subclinical diagnoses, a youth's high scores on self-report measures may portend significant clinical and functional impairment even if the person is not diagnosable (e.g., Gotlib, Lewinsohn, & Seeley, 1995). Use of the self-report Patient Health Questionnaire for Depression (Kroenke & Spitzer, 2002), which assesses the presence of the nine MDD symptoms and has been adapted for children and adolescents, has been recommended in DSM-5 as a supplement to diagnostic evaluation, providing a continuous score of severity of depression.

Clinical Course of Depression

Age of Onset

The prototypical depression is MDD with adolescent onset. In the National Comorbidity Survey Replication—Adolescent Supplement (NCS-A), the median age of onset for DSM-IV mood disorders was 13 (Merikangas et al., 2010). Retrospective assessment among community adults typically indicates that middle to late adolescence is the most common age of onset for a first episode of MDD or significant symptoms (e.g., Burke, Burke, Regier, & Rae, 1990; see also Kessler, Berglund, Demler, Jin, & Walters, 2005). In their community sample, Lewinsohn, Pettit, Joiner, and Seeley (2003) reported mean onset of MDD at around 14 years and mean onset of dysthymic disorder at around 11 years for both boys and girls. As discussed in the section on gender and depression, early adolescence is the point at which the rates of major depression increase sharply for girls and exceed depression rates in boys.

Age of onset appears to be an important potential marker for the course of a depressive disorder and the possibility of etiologically different subtypes. Compared to childhood onset, adolescent onset predicts greater homotypic continuity, whereas childhood onset is more commonly associated with heterotypic continuity. For example, Weissman, Wolk, Wickramaratne, and colleagues (1999) followed a clinically ascertained group of prepubertally depressed youngsters for 10–15 years into adulthood, and found that the majority did not go on to have adult depressive experiences. These youngsters had high rates of psychological disorders and significant maladjustment, but there was poor specificity for depressive disorders. Similar results were reported by Harrington, Fudge, Rutter, Pickles, and Hill (1990) in a follow-back study of the adult functioning of individuals who had been treated for depression as children or adolescents. Thus, across these studies, *childhood* onset of depression may predict significant disorder but not specifically recurring depression, except in subsamples characterized by less comorbidity, recurrent MDDs, and family history of depression. Many children presenting with depression plus externalizing disorders may have an etiologically different depression, or actually may not have depressive disorder as such, but rather suffer from marked emotional and behavioral dysfunction that eventually coalesces into nondepressive psychopathology. As reported later in the section on genetic factors in depression, studies of heritability typically find much stronger evidence of heritability for adolescent-onset depression (similar to adult depression) than for childhood onset (e.g., Rice, 2010).

Recurrence

Data on continuity of *adolescent* depressive disorders into adulthood are strongly consistent and underscore the premise that much depression seen in adults is actually recurrent adolescent-onset depression. Several large-scale prospective community samples reported on the outcomes in young adulthood of those who had been found to have a diagnosis of MDD during adolescence. The Queensland High Risk Study (Hammen, Brennan, Keenan-Miller, & Herr, 2008), the Dunedin (New Zealand) Multidisciplinary Health and Development Study (Bardone, Moffitt, Caspi, Dickson, & Silva, 1996), the Ontario Child Health Study (Fleming, Boyle, & Offord, 1993), the Oregon Adolescent Depression Project (Lewinsohn, Rohde, Klein, & Seeley, 1999),

and the Upstate New York study (Pine, Cohen, Gurley, Brook, & Ma, 1998) all reported high rates of recurrence of MDD in young adulthood (approximately 25–45% within 4 years). Recurrence rates of 40–60% are typical of clinically ascertained samples. A large-scale 10-year study of clinic-referred adolescents followed up to a mean age of 26 found that only 37% survived without an episode of MDD in adulthood (Weissman, Wolk, Goldstein, et al., 1999).

Among those with childhood onset of depression, true childhood-onset unipolar depression is relatively rare, but appears to be associated with a high risk of recurrence; many with unipolar depression have been found to have early-onset dysthymic disorder followed by MDEs (so-called "double depression"), as well as a high degree of depressive disorders in relatives (Birmaher et al., 2004; Kovacs, Akiskal, Gatsonis, & Parrone, 1994; Kovacs, Devlin, Pollock, Richards, & Mukerji, 1997; Weissman, Wolk, Wickramaratne, et al., 1999). Several studies have found a strong association between childhood anxiety symptoms and inhibited or withdrawn behavior, and later anxiety and depressive disorders (e.g., Goodwin, Fergusson, & Horwood, 2004; Katz, Conway, Hammen, Brennan, & Najman, 2011).

It should be noted that a significant minority of children initially (mis)diagnosed with unipolar depression in clinical settings eventually evidence hypomania or mania, which enables a bipolar disorder to be diagnosed (reviewed in Kovacs, 1996; see Youngstrom & Algorta, Chapter 6, this volume). For instance, Kovacs, Akiskal, Gatsonis, and Parrone (1994) found that 13% of their sample initially diagnosed with depression "switched" to bipolar disorder if followed long enough. Geller, Fox, and Clark (1994) found that among a clinical sample of severely depressed children (ages 6–12), 32% switched to bipolar I or II during a 2- to 5-year follow-up. Although symptom predictors of switching have yet to be validated, having a family member with a bipolar disorder increases the likelihood that a child's depression may be an early manifestation of a bipolar disorder. Biederman and colleagues (2009) found that depressed children and adolescents with comorbid ADHD or conduct disorder at baseline also had an increased likelihood of eventual bipolar diagnoses.

Comorbidity

The co-occurrence of disorders has attracted considerable attention in recent years, and comorbidity has now become widely recognized as the rule rather than the exception among depressed youngsters. Community studies permit the best tests of comorbidity rates, inasmuch as clinical populations may be biased because treatment seeking is more common among those with multiple conditions, which in turn are associated with greater impairment of functioning. In a large British community survey, children ages 5–15 with depression were most likely of those with any disorder to have at least one current comorbid diagnosis (66%) (Ford, Goodman, & Meltzer, 2003). Depressed children and adolescents are especially likely to experience anxiety disorders, but also conduct/behavioral disorders, as well as substance use disorders (in adolescents). Angold, Costello, and Erkanli (1999) conducted a meta-analysis of comorbidity in community studies of youngsters and reported a median odds ratio (degree of association) of 8.2 for depression and anxiety disorders, 6.6 for depression and conduct/oppositional defiant disorder, and 5.5 for depression and ADHD. It should be noted that patterns of comorbidity and timing of disorders may differ somewhat by developmental stage and gender (see O'Neil, Conner, & Kendall, 2011; Zahn-Waxler, Shirtcliff, & Marceau, 2008).

To a considerable extent, depression usually occurs after an earlier-onset disorder. A particularly striking case is that of anxiety disorders. Rohde (2009) reported that anxiety disorders occurred first in 85% of youth with comorbid depressive/anxiety disorders (see also Essau, 2003), although depression also may be followed by anxiety disorders in some cases. Externalizing disorders often have earlier onset than comorbid depressive disorders, although the pattern is variable across studies (Rohde, 2009). Kessler, Avenevoli, McLaughlin, and colleagues (2012) retrospectively evaluated temporal patterns of disorders in the NCS-A: They factor-analyzed disorders into classes labeled Fear (e.g., social and specific phobia, panic disorder), Distress (generalized anxiety or separation anxiety disorders and depressive disorders), Behavior (e.g., oppositional defiant disorder, conduct disorder, ADHD), and Substance disorders. The investigators determined that within-class associations were significantly stronger than cross-class associations (e.g., Distress disorders predicted other Distress disorders). Fear disorders were the strongest cross-class predictors, consistent with the common observation of early-onset anxiety disorders preceding depressive disorders.

The magnitude of depression comorbidity raises important clinical, conceptual, and methodological questions. Clinically, the presence of comorbid conditions

with depression predicts greater impairment of functioning, sometimes elevated rates of suicidal behavior, and greater treatment utilization, but less successful treatment outcomes (reviewed in Rohde, 2009). Conceptually, extensive comorbidity means that research findings attributed to depression may sometimes reflect effects due to unreported comorbid conditions or to the greater severity/impairment typically associated with comorbidity. The high rates of comorbidity have been variously hypothesized to arise from deficiencies in the diagnostic system, such as overlapping symptoms, shared etiological factors, or a functional relationship between disorders (e.g., disruptive behavior disorders may cause stressful consequences that provoke depressive reactions). Notably, there are significant bodies of research on shared etiological features of depression and anxiety disorders (e.g., genetic, personality/temperament, and neurotransmitter/neurocognitive factors), and on differentiating shared and unique predictive factors (e.g., Anderson & Hope, 2008; Clark & Watson, 1991).

A full discussion of the origins and meaning of depression comorbidity is beyond the scope of the present chapter. However, it is worth noting the emergence of analytic and assessment strategies to deal with the joint problems of the heterogeneity of the depression phenotype and diagnostic comorbidity, which are barriers to precision in the understanding of depression and its unique risk factors and consequences. Of relevance to depression is the use of quantitative approaches to aggregating manifest DSM diagnoses into superordinate categories, based on the assumption that disorders within the superordinate category reflect a common cause. Numerous studies mostly on adults have supported general internalizing and externalizing factors (e.g., Eaton et al., 2012; see Krueger & Markon, 2006), but Kessler, Avenevoli, McLaughlin, and colleagues (2012) also demonstrated the broad internalizing factor among adolescents with diagnoses may be subdivided into Fear and Distress factors. Such transdiagnostic approaches to the study of child and adolescent depression/anxiety might yield new insights beyond those of studies more narrowly focused on specific DSM diagnoses. In addition, the National Institute of Mental Health has developed the Research Domain Criteria (Sanislow et al., 2010) as a research strategy intended to study specific functions across multiple units of analysis, to cut across diagnostic boundaries, and to try to translate basic research into an improved and integrative understanding and treatment of psychopathology. This strategy is somewhat similar to the search for "endophenotypes" or "intermediate phenotypes." An endophenotype is the more specific representation or element of a disorder that is between the disease and likely distal heritable aspect of the mechanism of the disorder but is not the same as the diagnostic entity. In youth depression, for example, bias toward negative emotions (negative mood), or impaired reward functioning (anhedonia), or a particular biological function such as amygdala reactivity to emotional stimuli are a potential endophenotype that might provide a more focused target of study independent of the diagnostic heterogeneity of MDD and comorbidity (e.g., Hasler, Drevets, Manji, & Charney, 2004). Further developments in our understanding of youth depression are likely to require assessment strategies and case identification methods that go beyond use of DSM categories.

Summary

Depressive disorders and significant symptoms in children and adolescents often portend serious psychological and functional adjustment problems—sometimes recurring depression, but other times different forms of maladjustment into later adolescence and adulthood. Adolescent-onset depression is virtually the "prototype" of what we mean by MDD in adults. However, the differences among presentations of depression by age, clinical features, comorbidities, and outcomes are obstacles to research. Thus further study of developmentally appropriate diagnostic and assessment methods, as well as both transdiagnostic and endophenotypic approaches to characterization of the phenomena, are warranted.

EPIDEMIOLOGY

Prevalence/Incidence

Recent years have seen an increase in epidemiological surveys of child and adolescent disorders using diagnostic interviews and representative samples, although variations in assessment and informant methods have precluded precise comparability across studies. A review of 28 U.S. and international surveys using standardized diagnostic criteria was reported by Avenevoli and colleagues (2008), who found a range of 2–13% in 6- or 12-month prevalence of MDD among adolescents (approximately 13–18 years), and about 1–3% among

school-age children (7–12 years). The NCS-A, the largest and most nationally representative U.S. diagnosis-based survey, reported a 12-month prevalence rate of 8.2% for MDD or dysthymia in the 13- to 17-year-old sample (Kessler, Avenevoli, Costello, Georgiades, et al., 2012); about one-third of adolescents with depression were characterized as having "severe" cases, as defined by functional impairment represented by a score of 50 or less on the Children's Global Assessment Scale (Kessler, Avenevoli, Costello, Green, et al., 2012). Merikangas and colleagues (2010) reported a lifetime rate of 11.7% with MDD or dysthymia in the NCS-A, including 8.7% with severe depression. Avenenvoli and colleagues (2008) found that rates of dysthymia are typically are higher than those of MDD among children, but lower than those of MDD among adolescents.

Epidemiological samples also report markedly high rates of elevated depressive symptoms. A U.S. school-based survey of 11-to 15-year-olds obtained self-reports of DSM criteria for MDE in the past 12 months, and found that 18% of youth overall met the criteria (25% females, 10% males; Saluja et al., 2004). In the National Longitudinal Study of Adolescent Health of youth in grades 7–12 (AddHealth), 29% of youth reported depressive symptoms for the past week meeting the "moderately severe" cutoff of 16 on the Center for Epidemiologic Studies Depression Scale, and 9% met the "severe" cutoff of >24 (Rushton, Forcier, & Schectman, 2002). One year later, 44% of youth with severe symptoms continued to report the same high levels.

Gender, Socioeconomic, and Race/Cultural Differences in Depression

Besides evidence for higher rates of depression among adolescents than children, other notable epidemiological issues concern distributions by gender and additional sociodemographic factors. Most studies indicate that boys and girls have largely similar rates of depression in childhood, but by early adolescence, girls' rates of depressive disorders accelerate dramatically to approximately twice the rates as for boys, and the female–male gender difference remains throughout adulthood and occurs cross-nationally (e.g., Kessler, Avenevoli, Costello, Georgiades, et al., 2012; Merikangas et al., 2010; Nolen-Hoeksema & Girgus, 1994).

The female preponderance of depression is a significant challenge to theories about the origins of these disorders. Several theoretical perspectives highlight the interactive contribution of sex-linked differences in hormonal and biological functioning, stress-related processes, and interpersonal relatedness to the emerging sex difference during adolescence (e.g., Cyranowski, Frank, Young, & Shear, 2000; Hilt & Nolen-Hoeksema, 2009; Rudolph, 2009). Collectively, these perspectives suggest that complex associations among puberty-linked gonadal hormones and brain neurotransmitters affect mood and biological processes in response to stressful circumstances during adolescence in vulnerable individuals. Risk is also thought to be intensified in girls relative to boys due to girls' greater exposure and reactivity to social challenges during this time (Rudolph, 2002; Shih, Eberhart, Hammen, & Brennan, 2006), which in turn are believed to result from both biological sex differences in affiliative needs and socialization experiences that create a heightened focus on interpersonal connectedness and social-evaluative concerns (Cyranowksi et al., 2000; Hilt & Nolen-Hoeksema, 2009; Rudolph, 2009). Girls also are exposed more often to traumatic sexual abuse experiences, which can further affect their biological and psychological reactivity to social stressors (Hilt & Nolen-Hoeksema, 2009). There are additional differences between the genders in the ways they cope with stressful life events and depressed mood, with women tending to adopt a more passive, internalized, ruminative style that amplifies depressive symptoms, compared to males' more active/distracting and instrumental coping that dissipates negative emotionality (Nolen-Hoeksema, 2000). During the transition through puberty, interpersonal vulnerability, social risk, and normative developmental challenges (e.g., physical-maturational, cognitive-developmental, and social-contextual changes) collectively contribute to the emerging sex difference in depression (Rudolph, 2009). Moreover, this developmental context of risk is particularly salient in girls who progress through puberty earlier than their peers—as reflected in prospective links between early maturation and heightened depressive symptoms—suggesting that puberty and its timing may be more important predictors of the emerging sex difference than chronological age per se (Rudolph, 2014). Clearly, complex, integrative models are necessary to account for the emergence of marked sex differences in rates of depression across adolescence.

Sociodemographic variables that represent relatively adverse environmental conditions also are generally associated with higher rates of depression in adult samples, but the evidence is less consistent for youth (e.g., Kessler, Avenevoli, Costello, Georgiades, et al., 2012).

Poverty was not associated with lifetime depressive disorders in youth in the NCS-A study (Merikangas et al., 2010). In their meta-analysis of depressive symptomatology in adolescent samples, Twenge and Nolen-Hoeksema (2002) found no association between socioeconomic status and depression. Similarly, evidence of systematic differences in depression by race/ethnicity has been mixed, with some studies showing elevated rates of depressive disorders and symptoms in African American, Hispanic/Latino, and Asian American samples compared with European American samples, but other studies showing no differences (e.g., Anderson & Mayes, 2010; Latzman et al., 2011). Higher rates of mood disorders were seen in Hispanic adolescents than in non-Hispanic European American adolescents in the NCS-A lifetime rates (Merikangas et al., 2010). Twenge and Nolen-Hoeksema (2002) also found that Hispanic samples scored higher levels of depressive symptoms than did African American or European American samples. Further studies are needed to explore race and ethnicity effects by addressing methodological shortcomings, and to separate out effects that might be caused by different cultural expressions of depressive symptoms and adverse conditions that could be associated with ethnic status (e.g., Anderson & Mayes, 2010).

Birth Cohort Effects

Earlier reports of birth cohort effects showing higher rates of major depression in those born more recently (e.g., Klerman et al., 1985) have been replicated in the United States and internationally by the Cross-National Collaborative Group (1992), indicating growing rates of childhood or adolescent onset of depression among those born in more recent decades. Results from the original National Comorbidity Study also showed evidence of increasing prevalence of MDEs in those born since 1960 (Kessler, Avenevoli, & Merikangas, 2001). Various analyses of the sources of such increasing rates generally have argued against methodological artifacts as explanations, such as memory or increasing willingness to admit to depressive experiences. Most of the research has been based on retrospective accounts; obviously, longitudinally collected information is needed to examine the issue more directly. Twenge and Nolen-Hoeksema (2002) examined longitudinal studies of depressive symptoms (controlling for age and period effects) and found evidence of decreasing symptoms for boys and no changes for girls, contrary to the findings of earlier retrospective studies. It is likely that rates of depressive disorders did increase among youth in recent years—clearly accompanied by higher rates of treatment seeking, impairment, and suicidality—but whether the effect has now diminished, or was in fact due in significant part to changes in perceptions and awareness of depression, remains unresolved.

ETIOLOGY OF DEPRESSION

Biological Vulnerability to Depression

Brain Structures and Neural Circuitry

Efforts to understand the neural underpinnings of depression have commonly focused on brain structures associated with detecting, responding to, and regulating emotional information—mostly in limbic and cortical circuits, including prefrontal cortex (PFC), amygdala, hippocampal, ventromedial striatum, and related areas. The amygdala is involved in detection of stimuli that are salient for the individual's immediate well-being. Meta-analyses of structural findings have largely confirmed reductions in amygdala *volume* in adults with depression (especially those who are unmedicated), compared with controls (Hamilton, Siemer, & Gotlib, 2008). According to a review of imaging studies of depressed children and adolescents, there is similar but not entirely consistent evidence of amygdala volume differences in depressed and nondepressed youth (Hulvershorn, Cullen, & Anand, 2011); however, certain cortical areas, such as the PFC, orbitofrontal cortex (OFC), and anterior cingulate cortex (ACC), more consistently show volume abnormalities similar to those seen in adult depression.

Smaller amygdala volume is associated with greater responsivity to emotional stimuli. Studies of amygdala *activation* commonly present stimuli such as emotional faces. Depressed adults who were scanned while viewing fearful faces displayed greater amygdala activation compared to nondepressed controls (e.g., Monk, 2008). Similar paradigms generally yield parallel findings in youth, although with small samples and some inconsistencies between child and adolescent samples (Hulvershorn et al., 2011). Yang and colleagues (2010) presented an emotional face-matching task to adolescents with depression and to matched controls, and observed abnormally hyperactive left amygdala in the depressed youth. Increased amygdala activation also has been observed in adolescents at risk for depression due to maternal depression (Monk et al., 2008). Using a neu-

ral activation paradigm, Joormann, Cooney, Henry, and Gotlib (2012) demonstrated that at-risk girls were less successful in cognitive control (i.e., using positive autobiographical memories to "repair" sad mood induced by a film). Compared to the control daughters, the at-risk daughters showed less activation of dorsal areas of the PFC to recruit positive memories to reduce sad mood, and showed sustained greater amygdala activation. The authors speculate that these neural patterns reflect a trait marker stemming from difficulty in regulating negative affect, potentially portending development of depression (especially in the face of stressors). Emotion regulation processes based in limbic–cortical interactions also may be disrupted in youth with very early-onset MDD. Pagliaccio and colleagues (2012) studied school-age children who had experienced preschool onset of depression, using a version of the sad mood elaboration task of Joormann and colleagues, and found similar hypoactivity in areas of the PFC.

It should be noted that research has yet to determine the origins of depression-related cortical–limbic abnormalities—whether they are acquired, genetic, or both. Several studies have demonstrated the role of gene variants in neural correlates of disorder. Studies of both adults and children show increased activation of the amygdala to negative emotional stimuli among those with the short alleles of the serotonin transporter gene (5-HTTLPR; e.g., Furman, Joormann, Hamilton, & Gotlib, 2011; Hariri et al., 2002). Lau and colleagues (2010) found greater amygdala activation to emotional faces among brain-derived neurotrophic factor (BDNF) Met-allele carriers compared to Val/Val homozygotes in a sample of adolescents with depressive or anxiety disorders. Pagliaccio and colleagues (2012) found that severity of initial preschool depression was associated with later dysfunctional brain activity, and hypothesized that the depression symptoms may play a causal role in decreasing ability to effectively exert cognitive or prefrontal control over one's emotions.

Another element of cortical, limbic, and striatal brain regions implicated in emotion-processing neural circuits that are believed to be dysregulated in MDD is the hippocampus (HC), adjacent to the amygdala. The HC plays a major role in consolidation of information into long-term memory as well as in emotional responding, and is an important regulator of PFC function. It contains high levels of glucocorticoid receptors and is involved in the regulation of the hypothalamic–pituitary–adrenocortical (HPA) axis through its projections to the hypothalamus. It is vulnerable to stress-related steroids, which have been speculated to cause HC atrophy under conditions of severe and prolonged stress. Considerable evidence has shown reduced HC volume in depressed adult patients (e.g., Kempton et al., 2011; MacQueen & Frodl, 2011), and HC dysfunction contributes to sustained dysregulation of the stress response. Child and adolescent studies have generally found similar reduced HC volumes (Hulvershorn et al., 2011). Rao and colleagues (2010) found lower HC volumes in both depressed adolescents and at-risk adolescents (due to parental depression), and lower HC volume was associated with higher levels of early life adversity. Chen, Hamilton, and Gotlib (2010) similarly found lower HC volumes in girls ages 9–15 at risk for depression due to maternal depression. Several studies have noted potential genetic and environmental effects on reduced HC volume. For example, Frodl and colleagues (2010) found that adults' reports of childhood stress interacted with the presence of short alleles of the 5-HTTLPR gene to predict hippocampal volume. They speculated that genetic processes predictive of depression partly affect the extent of HC changes in response to stress.

Another conceptual paradigm for studying emotional mechanisms underlying depressive disorders has focused on biological bases of reward processing, involving affective, motivational, and decisional components, behaviorally reflected in depressed individuals' low mood and reduced experiences of pleasure, and biased perceptions of and attention to negative outcomes. Considerable evidence has emerged from task-based imaging studies of abnormalities in the striatum, amygdala, and OFC in depressed adults (e.g., Diekhof, Falkai, & Gruber, 2008). Studies of the reward behaviors and neural patterns of depressed children and adolescence also indicate abnormalities such as blunting of reward-related activation (e.g., Forbes et al., 2009). Similarly, even before the development of depression, girls at risk due to maternal depression displayed abnormal patterns under conditions of reward and loss in the striatum and the dorsal ACC, compared to a no-risk comparison group (Gotlib et al., 2010).

Reward circuitry also has been discussed in terms of asymmetries in PFC function associated with approach- and withdrawal-related mood and emotion, with left-sided hypoactivation associated with depression and reduced perception and pursuit of positive incentives, and right-sided hyperactivation associated with inhibition and anxiety (Davidson, Pizzagalli, Nitschke, & Putnam, 2002). Both infants of depressed

mothers and depressed adolescents display relatively reduced left frontal activation measured by electroencephalogram (reviewed in Davidson et al., 2002). These authors speculate that the interconnections of the PFC and other cortical and subcortical structures represent a dysfunctional circuit in which there is deficient regulation of the amygdala, potentially resulting in prolonged processing of negative affect, and insufficient modulatory control by other cortical functions. These ideas are bolstered by a decade of neuroimaging research, as noted above. However, it is important to acknowledge not only that the childhood/adolescent literature on brain functions is relatively small, but that the origins of abnormal neural structures and circuits are matters of speculation. There also is a paucity of longitudinal research clarifying the developmental course of functional brain abnormalities and their clinical consequences. The potential role of gonadal hormones in the development of depression, for example, is enormously complex, in part due to the crucial links among hormones, brain structures and functions, and neurotransmitters (Blanton et al., 2012).

Hypothalamic–Pituitary–Adrenocortical Axis

Dysregulation of the HPA axis is one of the most robust biological correlates of adult depression, with evidence of elevated cortisol levels, elevated corticotropin-releasing hormone (CRH), and impaired negative feedback control of the HPA axis. It is generally hypothesized that exposure to stressful events and chronic circumstances triggers the development of depression in part through the HPA axis and its associated brain connections, with the supposition that severe and/or early stress exposure may alter neural and HPA axis functioning (e.g., Heim, Newport, Mletzko, Miller, & Nemeroff, 2008). Some individuals also are presumed to have preexisting genetically or environmentally mediated abnormalities in the stress response system, which make them vulnerable to depressive reactions to stress (e.g., Halligan, Herbert, Goodyer, & Murray, 2007).

Earlier studies of child and adolescent depressed samples provided supportive but inconsistent evidence of HPA axis dysregulation, but more recent meta-analyses and reviews have drawn different conclusions. Guerry and Hastings (2011; see also Lopez-Duran, Kovacs, & George, 2009) examined studies based on different methods: dexamethasone suppression, basal cortisol, CRH infusion, psychological challenges, and children of depressed parents. Noting numerous methodological deficiencies, Guerry and Hastings (2011) and Lopez-Duran and colleagues (2009) nevertheless found that when studies were grouped by methods, there was fairly consistent evidence of abnormalities in HPA axis functioning in depressed or at-risk children and youth, including elevations in basal levels of cortisol, greater cortisol response to psychological stressors, and a predictive association between elevated cortisol and later development of depression. Moreover, differences between depressed and nondepressed youth generally appeared to be smaller in scale than between depressed and nondepressed adults, suggesting the need for developmentally informed hypotheses and methods. Indeed, research indicates that baseline cortisol levels and reactivity to stress increase across adolescence and pubertal development (e.g., Gunnar, Wewerka, Frenn, Long, & Griggs, 2009).

Guerry and Hastings (2011) suggested that cortisol differences are particularly observed under stressful conditions, such as laboratory stressors and both acute and chronic stress exposure (including having a depressed mother). Examining cortisol in the context of stressful situations, rather than naturally occurring levels, may improve prediction of future depressive symptoms. In one study, Susman, Dorn, Inoff-Germain, Nottelmann, and Chrousos (1997) found that cortisol reactivity to a stressful situation (blood draw) predicted depressive symptoms 1 year later. In another study, Rudolph, Troop-Gordon, and Granger (2011) found that exposure to the stressful experience of actual peer victimization interacted with heightened anticipatory cortisol while awaiting a laboratory peer-related stressor to predict depressive symptoms 1 year later. These findings suggest that sensitivity of the HPA axis to ongoing stressors, particularly in youth with a history of stress exposure, may serve as a risk factor for subsequent depression.

Adam, Sutton, Doane, and Mineka (2008) also emphasized the need for longitudinal studies of youth prior to the development of depression, and accentuated the importance of the "cortisol awakening response" (CAR, which occurs approximately 40 minutes after waking up) as a particularly potent predictor of future depression onset. Adam and colleagues (2010) found that elevated baseline CAR, but not other measures of cortisol such as bedtime or daily slope, predicted a significantly increased rate of depressive disorder over the next year in a community sample of late adolescents. It was suggested that the CAR measure is uniquely pre-

dictive of future depression, whereas other measures of cortisol functioning covary with current depression. Because the CAR marks the highest cortisol level of the day, Adam and colleagues speculate that over time high levels contribute to changes in brain glucocorticoid receptors involved in the negative feedback regulation of the HPA axis, including changes in the hippocampus and amygdala and their dysfunctional effects in the cortical–limbic circuits underlying emotion regulation. As to the origins of elevated CAR, these authors note the possibility of genetically transmitted characteristics, but emphasize the likelihood that adverse experiences in childhood modify the developing brain and its HPA axis characteristics.

Genetics

There have been enormous empirical and methodological advances in genetic research in recent years, and these offer a wide array of approaches to the study of genetic contributions to psychological disorders. Depression, while one of the most prevalent of all public health issues, unfortunately offers a substantially heterogeneous phenotype for study, varying in symptomatology, comorbidity, severity, age of onset, course, and impairment. The topic of child and adolescent depression contributes its own unique issues and questions: Do genetic features apply similarly to child, adolescent, and adult populations? How are we to characterize nature–nurture questions? What is the nature/mechanism of genetic effects on depression in youth?

It has been well established in reviews and meta-analyses that depression runs in families (e.g., Rice, Harold, & Thapar, 2002), but the contributions of heritability and environment are obviously confounded, due to the psychological and environmental effects of depression and its vulnerabilities and risk factors for other family members. Quantitative genetic analyses in the form of twin studies provide methods of partitioning the variance contributed by genetic and environmental factors. In general, studies of adults report heritability estimates of around .4, with more variance accounted for by environmental factors, particularly that which is unique to individuals and not "shared" in family contexts (reviewed in Lau & Eley, 2010). When applied to children and adolescents, however, twin studies have provided more variable heritability estimates. Rice (2010) notes that heritability is nonsignificant in childhood samples (with high rates of environmental contribution), whereas heritability is significant among adolescents, similar to rates found in adult depression. The majority of twin studies of children and adolescents have been based on symptom measures of depression, with some differences depending on whether self-ratings or parental ratings are employed. Thus caution is warranted until further studies with more diagnostic-based ascertainment are conducted. The finding that adolescent but not childhood depression has a significant heritable component is consistent with several follow-up studies of depressed child patients into adulthood, which found relatively low rates of continuity of depression (although those who were not depressed as adults nonetheless had severe behavioral disorders and impaired functioning; see, e.g., Harrington et al., 1990; Weissman, Wolk, Wickramaratne, et al., 1999). It should be noted that although questions remain about heritability of depression across different ages, analyses of gender differences tend to find little evidence of different genetic contributions to males and females, despite the greater incidence of depressive disorders in females arising in adolescence (Franic, Middledorp, Dolan, Ligthart, & Boomsma, 2010).

Important developmental information has emerged from *longitudinal* twin studies. For example, Lau and Eley (2006) examined depressive experiences three times during adolescence and early adulthood in the G1219 study, finding evidence of "new" genetic and nonshared environmental influences emerging over time. The investigators speculated that such changes might contribute to the increasing rates of depression in adolescence, and that such changes might mutually influence each other (such as increasing selection into stressful situations, which themselves could trigger other genetically driven vulnerabilities toward depression). A longitudinal study of Swedish twins at four points between ages 8 and 20 by Kendler, Gardner, and Lichtenstein (2008) examined changes in genetic and environmental risk factors for mixed depression/anxiety symptoms. They found evidence for changes in genetic influences, with new influences coming "online," but also for attenuation of earlier influences on later symptoms. They conclude that genetic factors show a dynamic course over development, possibly contributing to the low continuity of symptoms from childhood to adolescence.

Although classical quantitative genetic analyses of twin samples permit the partition of variance in depression into genetic and environmental factors, newer methodologies have also yielded important leads. For example, the Children of Twins study of twin sets of parents and their children permits analysis of genetic effects separate from family (environmental) condi-

tions. Investigators Silberg, Maes, and Eaves (2010) examined whether genetic or family environmental factors (or both) provided the best explanation for the association between parent and offspring depression. The authors concluded that the best predictor of the associations between parent and offspring depression was family environment, whereas offspring conduct disorder was predicted by both genetic and environmental factors.

Research in psychopathology in general has increasingly developed techniques and devoted resources to the molecular genetic approaches to gene finding, including various studies of depression in adults. However, such strategies have not been applied to depressed child and adolescent samples, and probably need to be deferred until clearer resolution of age and developmental issues is attained. Nevertheless, there has been considerable interest in addressing the question of how genetic factors exert their effects—in both adult and child/adolescent depression—with increasing focus on candidate genes. Specifically, studies of gene–environment interactions (G×E), and to a lesser degree gene–environment correlations (*r*GE), have employed the same candidate genes as those studied in adult depression. It appears that, as in adults, depression in youth is associated with genetic risk for increased exposure to adverse environments (*r*GE) as well as greater reactivity to such environments (G×E). Notably, a polymorphism in 5-HTTLPR has been associated with adult depression, particularly under stressful conditions (reviewed in Karg, Burmeister, Shedden, & Sen, 2011). Several studies have found similar relevant patterns in depressed or "at risk" (due to maternal depression) children (e.g., Gibb, Uhrlass, Grassia, Benas, & McGeary, 2009; Hayden et al., 2008), and adolescent or mixed child/adolescent samples (Eley et al., 2004; Goodyer, Croudace, Dudbridge, Ban, & Herbert, 2010; see also Hankin, Jenness, Abela, & Smolen, 2011, for a longitudinal analysis). Other studies of candidate genes in adult depressed samples also have yielded evidence of gene–environment interactions (e.g., BDNF—Goodyer et al., 2010; Kaufman et al., 2006; dopamine D2 receptor gene—Hayden et al., 2010). Increasingly, investigators are integrating brain circuit activation with genetic analyses in youth samples. For example, as noted in the review of brain reactivity, Lau and colleagues (2010) found that adolescent depressed or anxiety patients who were BDNF Met carriers showed greater amygdala and hippocampal activations than those with Val/Val homozygotes, although many inconsistencies in findings across neuroimaging (and candidate gene–environment) studies are evident, suggesting needs for replication, larger and better-characterized or homogeneous samples, and measurement precision.

Studies of *r*GE are particularly needed for understanding risk for depression due to parental depression, as depression is strongly correlated with various environmental circumstances, such that children "inherit" not only genes but environments including dysfunctional parenting (a possible case of "passive" *r*GE). Evocative and active *r*GE may also occur, as when a child's heritable traits lead to behaviors that provoke reactions in others, which in turn contribute to depression (e.g., dependency may provoke rejection), or lead to selection into adverse environmental conditions (e.g., low self-esteem may contribute to the selection of dysfunctional romantic partners). *r*GE with respect to the occurrence of stressful life events is well known; Hammen (1991), for example, used the phrase "stress generation" to refer to the tendency of individuals with a history of depression (not just current depression) to contribute to the occurrence of stressful life events—a trend also observed in children with depression and children of depressed women. Starr, Hammen, Brennan, and Najman (2012) found that the presence of short alleles of 5-HTTLPR interacted with depression to predict stressful life events in a high-risk sample of adolescents. Lau and Eley (2008) applied quantitative genetic analyses to twins and siblings in the G1219 longitudinal study to test for different effects of *r*GE and G×E; they incorporated two measures of the environment—negative life events that had been at least partly caused by a participant, and participants' reports of maternal punitive discipline. The complex findings implicated both G×E and *r*GE on the two measures, and suggested that adolescent depression's genetic risks are due in part to exposure to these two adverse conditions, and that the adverse conditions themselves may activate genetic effects and increase the probability of depressive reactions. The authors call for further studies of the mechanisms by which genetic effects are mediated, including brain circuits such as amygdala reactivity, as well as cognitive and personality processes.

Although the future of genetic approaches to understanding depression in adults and youth will doubtless lead in exciting directions with the expansion and further development of traditional quantitative and molecular paradigms, *epigenetic* processes that determine the where, when, and how much of fundamental protein components of DNA will also increasingly expand our understanding of genetic mechanisms of behavior. As reviewed by Lau and Eley (2010), animal research on

how gene expression is affected by environmental experiences, such as maternal care, will invariably open up new findings and questions about the origins, developmental course, and stability of behavioral and emotional outcomes relevant to depression.

Summary

Undeniably, the past decade has seen a surge in interest and methodological advances in the study of biological characteristics associated with adult depression—and, by extension, child and adolescent depression. Considerable interest focuses on emerging evidence in depressed children and adolescents on dysfunctional neural circuits underlying emotional processing and their associations with brain regions and mechanisms of information processing and cognitive control, as well as the effects of dysregulated biological stress processes in the HPA axis, and their associations with neurocognitive mechanisms. Similarly, research has underscored the importance of genetically mediated influences. However, major questions remain about the origins and mechanisms of neural abnormalities, and the transactions among experiential and biological factors. Developmentally sensitive models, studied in longitudinal designs, are needed for the field to advance in the clarity of our understanding of depression as it appears in youth.

Emotional Vulnerability to Depression

Mood disruptions are fundamental to depression; consequently, developmental theory and research have sought to uncover the unique pathways through which emotional functioning contributes to risk for depression in youth. In addition to the biological and cognitive processes related to emotions reviewed in other sections of this chapter, investigations of emotional vulnerability to depression have explored stable, trait-like aspects of emotionality (i.e., temperament), and situational, state-like aspects of emotional processing, responses, and regulation.

Theoretical Models

One group of theoretical perspectives focuses on temperament, as reflected in stable individual differences in self-regulation and affect that are presumed to result from the interplay between biology and experience (Rothbart & Posner, 2006). Theorists have highlighted three dimensions of temperament with relevance to depression (for a review, see Rothbart & Posner, 2006): high negative emotionality (NE; a tendency toward experiencing frequent, intense, and lasting negative affect); low positive emotionality (PE; a tendency toward experiencing low levels of positive emotions such as joy and pleasure); and poor effortful control (EC; difficulty inhibiting undesired impulses and effectively regulating attention). According to the tripartite model, the combination of high NE and low PE differentiates depression from internalizing disorders such as anxiety (Clark & Watson, 1991). Together, NE, PE, and EC account for children's' dispositional emotionality and responsivity to emotionally evocative stimuli.

Theorists typically conceptualize temperament as a diathesis for depression (e.g., Hyde, Mezulis, & Abramson, 2008; Yap, Allen, & Sheeber, 2007). According to these models, temperament may serve as a vulnerability to depression directly, by fostering symptoms (e.g., depressed mood, anhedonia, irritability), or indirectly, by fostering emotional vulnerability (e.g., rumination, emotion dysregulation) or eliciting negative experiences (e.g., stress) that in turn contribute to depression. Temperament also may act as a moderator, shaping children's emotional reactivity to environmental risks for depression, consistent with broader developmental theories such as the biological sensitivity to context model (Boyce & Ellis, 2005). Importantly, these theoretical models predict that direct, indirect, and moderated pathways are complementary rather than mutually exclusive.

Other perspectives focus on individual differences in emotional responses, emotion regulation, and emotion processing as precursors to depression. Response style theory posits that individuals who tend to engage in unproductive emotion-related rumination sustain and amplify negative moods rather than resolve them, such that a ruminative response style contributes to the onset and maintenance of depression (for a review, see Rood, Roelefs, Bogels, Nolen-Hoeksema, & Schouten, 2009). Emotion regulation perspectives propose that a failure to effectively regulate emotional responses is fundamental to mood disorders including depression, and that poor emotion regulation early in development will set the stage for later symptoms (e.g., Compas, Jaser, & Benson, 2009). Building on emotion regulation perspectives, others have proposed that deficits in processing emotions (e.g., identifying and understanding emotions, regulating attention to emotions) may underlie emotion regulation difficulties, which in turn

are expected to heighten vulnerability to depression (e.g., Flynn & Rudolph, in press). Some aspects of executive functioning with relevance to emotion regulation, such as problem solving, response selection, and regulation of attention, could also play a role in shaping vulnerability to depression (e.g., McClintock, Husain, Greer, & Cullum, 2010), although research is needed in youth populations (see later discussion of cognitive vulnerabilities to depression). In recent years, emotion processing and regulation perspectives have increasingly focused on ways that developmental changes in emotional functioning, particularly related to biological maturation (e.g., puberty, neurological maturation), may contribute to the higher prevalence of depressive symptoms in adolescence (e.g., Forbes, Phillips, Ryan, & Dahl, 2011).

Empirical Evidence

TEMPERAMENT

Empirical investigations of temperament and depression generally support theoretical predictions. Prospective research supports direct associations between high NE and depressive symptoms in adolescence (e.g., Krueger, 1999). Furthermore, heightened rumination (Mezulis, Simonson, McCauley, & Vander Stoep, 2011) and generation of stressful life events (Barrocas & Hankin, 2011) mediate the prospective association between NE and depression in youth; emotion dysregulation mediates the prospective link between EC and depression (Zalewski, Lengua, Wilson, Trancik, & Bazinet, 2011). Temperament also moderates the impact of environmental factors on depression. In some cases, temperamental vulnerability (e.g., high NE, low EC) enhances the contribution of environmental factors—such as negative parenting (Kiff, Lengua, & Bush, 2011) and peer victimization (Sugimura & Rudolph, 2012)—to depression. However, temperament also may promote resilience; for example, Gartstein and Bateman (2008) found that low NE attenuated the link between maternal depression in infancy and depression-like symptoms in toddlerhood.

EMOTION PROCESSING

A number of emotion-processing deficits are associated with vulnerability to depression in youth. Depressive symptoms are correlated with youth's inaccurate identification of parents' emotions in parent–child interactions (Ehrmantrout, Allen, Leve, Davis, & Sheeber, 2011) and with perceptions of more anger and less joy in low-intensity facial stimuli (van Beek & Dubas, 2008). Regarding one's own emotions, low emotional clarity, or difficulty identifying and distinguishing between one's emotions, and perceptual asymmetry in processing of emotional faces (i.e., reduced posterior right-hemispheric bias) predict subsequent depressive symptoms in youth. Importantly, maladaptive responses to stress (e.g., low levels of engagement strategies, such as emotion regulation; high levels of dysregulated, automatic responses, such as rumination and emotional numbing) mediated these associations, supporting the idea that poor emotion regulation is a mechanism through which emotion-processing deficits increase vulnerability to depression (Flynn & Rudolph, 2010a, 2010b, in press).

EMOTION REGULATION

Depressed youth also exhibit compromised emotional responses and regulation. Depressive symptoms are correlated with parent and self-reports of poor emotion regulation (for a review, see Durbin & Shafir, 2008). Corroborating these findings, cross-sectional research indicates that compared to nondepressed youth, depressed youth exhibit more dysregulated expressions of negative affect as indexed by experience-sampling methods (Silk et al., 2011) and observations (Sheeber et al., 2009). Some longitudinal research supports the idea that poor emotional functioning sets the stage for later depression in youth. Consistent with response style theory, emotion-related rumination is a robust prospective predictor of heightened depressive symptoms, particularly among adolescents (for a meta-analysis, see Rood et al., 2009). Limited research reveals prospective links between aspects of poor emotion regulation and youth depression, including high emotional inertia (i.e., temporally persistent moods; Kuppens et al., 2011) and poor regulation of sadness and anger (Feng et al., 2009).

Origins and Development of Emotional Vulnerability

Both environmental and biological factors play a role in shaping the development of emotional vulnerability to depression in youth. Considerable evidence links early adversity (e.g., maltreatment, parent depression) to disruptions in emotional processing and regulation (at both biological and behavioral levels) among youth consid-

ered at risk for depression (for a review, see Abàied & Rudolph, 2014). Proximal environmental factors, such as maladaptive parent socialization of emotion regulation and coping, also contribute to deficits in emotional functioning and depression (e.g., Abaied & Rudolph, 2010b, 2011). However, additional research is needed to test directly whether youth emotional functioning accounts for prospective contributions of adversity and parent socialization to depression.

Developmental neuroscience research suggests that emotional vulnerability to depression also has neurological underpinnings. Researchers have proposed that reduced neural response to rewards may underlie the low levels of PE common to depression, and that biological maturation may exaggerate this pattern in adolescence (for a review, see Forbes & Dahl, 2012). Youth at risk for depression (e.g., daughters of depressed mothers) also show maladaptive patterns of neural activity during emotion regulation tasks (e.g., Joormann et al., 2012). These processes may be affected by endocrinological changes associated with pubertal maturation; Forbes and colleagues (2011) found that pubertal maturation rather than age predicted less activation in brain regions associated with emotion regulation in response to social threats. Outside the context of neuroscience research, Silk and colleagues (2011) found that depressed adolescents experienced higher levels of negative emotions than nondepressed adolescents, and that this difference was amplified among those with advanced pubertal status. These puberty-related changes in emotional vulnerability may help to explain the higher rates of depression onset in adolescence than in childhood.

Summary

Emotional vulnerability to depression operates though both state- and trait-like processes. Prospective designs provide ample empirical support for temperament models of depression and response style theory, such that youth's dispositional emotionality and style of emotional responses are implicated in the development of depression. Although research supports emotion processing and regulation deficits among depressed youth and youth at risk for depression (i.e., those exposed to early adversity), additional longitudinal investigations are needed to more clearly differentiate emotion regulation deficits as antecedents versus consequences of depression. Future research also should seek to elucidate the pathways though which multiple levels of emotional functioning (i.e., biological, cognitive, behavioral) combine and interact to shape vulnerability to depression.

Cognitive Vulnerability to Depression

Cognitive models implicate negative belief systems and maladaptive information processing in the onset and course of depression. According to these models, cognitive vulnerability serves as a stable predisposition that interacts with life stress to predict depression. This vulnerability is reflected in characteristic biases in attention, interpretation, and recall of information. More specifically, cognitive theories often suggest that a key determinant of depression is the *match* between a particular cognitive vulnerability and a particular stressor. That is, stressful events or circumstances would induce depression to the extent that they precipitate a loss of self-worth in an individual's specific area of cognitive vulnerability. In this regard, the most common distinction focuses on individual differences in the tendency to base one's self-worth either on success in interpersonal relationships (as reflected in sociotropy or dependency) or on individual achievement and independence (as reflected in autonomy or self-criticism) (Beck, 1987; Blatt & Zuroff, 1992; Coyne & Wiffen, 1995).

Theoretical Models

Beck's (1967, 1987) cognitive theory of depression elucidates three aspects of disrupted cognitive functioning in depression. First, depressed individuals possess core dysfunctional attitudes and negative cognitive schemas (characterized by themes of loss, failure, and inadequacy) that guide information processing. Second, these schemas drive systematic biases in thinking, which create idiosyncratic interpretations of events (e.g., negative automatic thoughts, cognitive errors). Third, depression is associated with the "negative cognitive triad," or a tendency to possess negative views of the self as worthless or inadequate, the world as mean or unfair, and the future as hopeless. The theory maintains that these cognitive styles heighten susceptibility to depression, especially when activated by external stressors. Because the rigid nature of cognitive schemas renders them highly resistant to change, depressed individuals may be vulnerable to persistent difficulties.

A second set of cognitive theories involves reformulations of Seligman's (1975) "learned helplessness" model. The original version posited that depression

stems from the experience of uncontrollable, noncontingent events. A revision of this model (Abramson, Seligman, & Teasdale, 1978) introduced the notion of a "depressive attributional style," or a predisposition to attribute negative outcomes to internal, global, and stable factors, and positive outcomes to external, specific, and unstable factors. In the most recent version of this model, Abramson, Metalsky, and Alloy (1989) described a subtype of "hopelessness" depression, which evolves from the interaction between exposure to negative events and a depressogenic inferential style involving pessimistic inferences about the causes, consequences, and self-implications of events.

Related self-regulatory theories (Rehm, 1977; Weisz, Sweeney, Proffitt, & Carr, 1994) suggest that one's expectations about outcomes (e.g., perceptions of control and competence, outcome contingencies) and one's personal investment in outcomes (e.g., goals, standards, values) jointly confer vulnerability to depression. Competence-based models focus in particular on the perceived competence aspect of self-regulation (Cole, Martin, & Powers, 1997). Appraisal-based models emphasize maladaptive appraisals about the meaning of events (a tendency to appraise challenging events as threatening, harmful, or stressful, rather than as opportunities for learning, mastery, and growth; Lazarus & Folkman, 1984).

Response style theory (Nolen-Hoeksema, 1991) proposes that depression arises from individual differences in self-focused attention. According to this theory, the tendency to "ruminate"—rather than distract oneself—in response to negative affect determines susceptibility to persistent and severe depression. Rumination involves perseverating on depressive symptoms and the possible causes and consequences of symptoms. More recent elaborations of this theory distinguish two dimensions of rumination (Treynor, Gonzalez, & Nolen-Hoeksema, 2003). Whereas "brooding" involves passively focusing on symptoms, "self-reflection" involves actively attempting to gain insight into one's problems. It is thought that brooding, but not self-reflection, serves as a specific vulnerability for depression.

Empirical Evidence

Over the past decade, researchers have increasingly used prospective designs to evaluate the etiological significance of cognitive vulnerability, as well as to test cognitive vulnerability–stress interactions. This research has yielded significant support for cognitive vulnerability–stress models of depression in youth, although there are exceptions, with some studies yielding qualified support (e.g., by age, type of outcome, interactions with other vulnerabilities) or no support for these models (for reviews, see Abela & Hankin, 2008; Gibb & Coles, 2005; Jacobs, Reinecke, Gollan, & Kane, 2008).

SELF-REPORTED BELIEFS AND STYLES

Most longitudinal research uses self-report questionnaires to examine explicit aspects of cognitive vulnerability, such as dysfunctional attitudes (e.g., perfectionism, need for social approval), negative automatic thoughts (e.g., catastrophization, overgeneralization), negative inferential style (e.g., stable, global attributions for failure), self-critical thoughts, low perceived control, and ruminative response styles. Overall, this research provides compelling evidence for the idea that self-reported cognitive vulnerability alone and, in particular, vulnerability–stress interactions prospectively contribute to subsequent depressive symptoms and disorders in youth.

Examining Beck's theory, a few studies provide partial or full support for the idea that dysfunctional attitudes prospectively interact with stress to predict depressive symptoms (e.g., Abela & Skitch, 2007; Hankin, Abramson, Miller, & Haeffel, 2004; Lewinsohn, Joiner, & Rhode, 2001). Examining the hopelessness theory, a growing number of studies reveal that depressive attributions about the causes of events interact with stress to predict depressive symptoms (e.g., Abela et al., 2011; Bohon, Stice, Burton, Fudell, & Nolen-Hoeksema, 2008; Carter & Garber, 2011; Hankin, 2008). A few studies provide partial or full support for the predictive contribution of depressogenic inferences about the consequences and self-implications of events (e.g., Abela, 2001, 2002). Building on these findings, Abela and colleagues (e.g., Abela & Sarin, 2002) have shown that it may be important to identify youth's "weakest link"—namely, their most negative cognitive style—in research examining cognitive vulnerability–stress interactions. Research also supports the predictive contribution of perceived competence (Tram & Cole, 2000) and control (Rudolph, Kurlakowsky, & Conley, 2001) to depressive symptoms. Finally, ruminative response style alone (Nolen-Hoeksema, Stice, Wade, & Bohon, 2007) and in interaction with stress (Abela & Hankin, 2011) predicts subsequent depression. When subtypes are distinguished, effects hold

for brooding rather than reflection; moreover, brooding accounts for stability in depressive symptoms in girls but not in boys (Burwell & Shirk, 2007). Despite evidence for cognitive vulnerability–stress models, it is noteworthy that some studies provide only partial or no support for these theories (for reviews, see Abela & Hankin 2008; Gibb & Coles, 2005; Jacobs et al., 2008); moreover, research often supports a reciprocal association wherein depressive symptoms predict subsequent maladaptive cognitions (e.g., LaGrange et al., 2011).

Less research has tested the validity of cognitive specificity models in youth. Some research supports self-criticism × achievement stress contributions (Abela, Sakellaropoulo, & Taxel, 2007) and dependency × interpersonal stress contributions (Little & Garber, 2000) to depressive symptoms over time; however, other research has not supported the predictive role of cognitive vulnerability–stress match (for a review, see Abela & Hankin, 2008). Research also reveals that depressogenic interpersonal beliefs and schemas (negative beliefs and biased processing about interpersonal relationships) confer vulnerability to depression in the face of interpersonal stress (Hammen et al., 1995; Shirk, Boergers, Eason, & Van Horn, 1998). These preliminary findings indicate the need for further pursuit of longitudinal research on domain-specific cognitive vulnerability and cognition–stressor match.

INFORMATION-PROCESSING BIASES

Researchers have used two approaches to examine the role of implicit information-processing biases in depression. The first approach involves experimental assessments of selective attention and memory. Relative to nondepressed youth, depressed youth show an attentional bias toward sad faces (Hankin, Gibb, Abela, & Flory, 2010) and idiosyncratic processing of self-referent (Hammen & Zupan, 1984; Neshat-Doost, Taghavi, Moradi, Yule, & Dalgleish, 1998) and other-referent (Rudolph, Hammen, & Burge, 1997) information. This research reveals that depressed youth show either more of a bias toward negative stimuli, or less of a bias toward positive stimuli, than do nondepressed youth, although there are exceptions (for a review, see Jacobs et al., 2008).

The second approach involves examining the accuracy of cognitive appraisals in depressed youth. Most investigations of cognitive vulnerability assess decontextualized belief systems (e.g., generalized dysfunctional attitudes) or interpretations of hypothetical events (e.g., negative inferential style), making it difficult to determine the accuracy of depressogenic cognitive styles. Although we might surmise that extreme negative beliefs (e.g., catastrophization) are at least somewhat biased, it is clear that depressed youth do, in fact, experience significant competence deficits and environmental adversity (for a review, see Rudolph, Hammen, & Daley, 2006). Thus characteristic negative cognitions could, at least in part, reflect realistic appraisals of such disturbances. To resolve this issue, a few studies have examined the accuracy of appraisals by assessing cognitions within the context of actual life experiences.

Examining self-appraisals of competence, research reveals that depressed youth underestimate their competence relative to objective ratings (e.g., Brendgen, Vitaro, Turgeon, & Poulin, 2002), supporting the presence of a depressive bias in self-appraisal. Examining appraisals of naturally occurring life events, one study confirms that depressed youth overestimate event stressfulness (the degree of negative impact associated with events) and event dependence (the extent to which the youth contributed to event occurrence) relative to objective ratings (Krackow & Rudolph, 2008), confirming the presence of a depressive bias in appraisals of event meaning, causes, and consequences.

Despite evidence for information-processing biases in depressed youth, this research is limited by several methodological constraints. First, studies of information processing primarily use concurrent designs, precluding strong conclusions regarding the temporal precedence of cognitive vulnerability. Indeed, a few studies (Cole, Martin, Peeke, Seroczynski, & Hoffman, 1998; McGrath & Repetti, 2002; Pomerantz & Rudolph, 2003) suggest that depressive symptoms foster biased self-appraisals (i.e., underestimations of competence) over time. Second, research reveals that depressed youth are sensitive to actual deficits in their competence (Rudolph & Clark, 2001), suggesting some realistic basis for negative appraisals and the need for future research to distinguish realistic versus distorted perceptions of reality. Third, much of this research overlooks the contextual component of cognitive theories—namely, that depressogenic cognitive schemas may remain latent until activated by negative mood states or events. To address this concern, future research needs to assess information-processing biases under conditions of cognitive activation, such as following negative mood induction (e.g., Taylor & Ingram, 1999), or as a diathesis that interacts with stressful life events to predict depression.

Origins and Development of Cognitive Vulnerability

For a full understanding of the role cognitive vulnerability plays in depression, it is important to elucidate how it emerges and develops over time. Original cognitive theories, developed in adults, view cognitive vulnerability as an early-emerging and persistent predisposition stemming from adverse experiences. This perspective assumes that cognitive vulnerability is a relatively stable, latent personality trait that is activated by mood-related or environmental triggers (Joormann, 2009). Prompted by research suggesting developmental differences in the stability and predictive validity of cognitive vulnerability (for a review, see Abela & Hankin, 2008), as well as research suggesting that depressive symptoms may leave a cognitive "scar" (Nolen-Hoeksema, Girgus, & Seligman, 1992; Pomerantz & Rudolph, 2003), the field of developmental psychopathology increasingly views cognitive vulnerability as a dynamic construct that crystallizes over the course of development, perhaps in response to maturational and experiential changes during the transition through adolescence.

INDIVIDUAL-DIFFERENCE ORIGINS

There is some evidence for a genetic liability to cognitive vulnerability, including negative attributional style (Lau, Rijsdijk, & Eley, 2006), information-processing biases (Beevers, Wells, Ellis, & McGeary, 2009; Pérez-Edgar et al., 2010) and rumination (Beevers, Wells, & McGeary, 2009). Research also has investigated the neural and biological basis of cognitive vulnerability. For example, differential patterns of brain activation (Monk et al., 2008) and heightened cortisol activation to stress (Rudolph, Troop-Gordon, & Granger, 2011) are linked to attentional biases and rumination, respectively. Temperament or personality traits may play a role in the development of cognitive vulnerability. One study revealed that low levels of positive emotionality in early childhood predicted subsequent information-processing biases (less recall of positive self-referent information; Hayden, Klein, Durbin, & Olino, 2006). Another study revealed that depressive personality traits were associated with girls' tendency to overestimate the stressfulness of events and their contribution to events, even after the researchers adjusted for lifetime history of depression (Rudolph & Klein, 2009). Increasing interest also has emerged in how core executive functions (cognitive processes that guide planning, decision making, and self-regulation) influence cognitive vulnerability to depression. For example, deficits in cognitive inhibition interfere with efficient updating of working memory (i.e., focusing attention on relevant information and ignoring irrelevant information), setting the stage for rumination, information-processing biases (e.g., elaboration of and difficulty disengaging from negative material), and other forms of cognitive vulnerability (for reviews, see Gotlib & Joormann, 2010; Joormann, 2009). Given these links, it is not surprising that executive function deficits are associated with depression in adults (for a review, see Gotlib & Joormann, 2010); research with youth is more limited and typically relies on adult reports of temperamental dimensions associated with executive functions, such as EC and attention regulation, rather than cognitive tasks (for a review, see Eisenberg, Smith, Sadovsky, & Spinrad, 2004).

SOCIAL-CONTEXTUAL ORIGINS

Several theories suggest that early exposure to chronic or severe adversity (e.g., trauma, family disruption, life stressors, maladaptive parent socialization) is internalized in the form of cognitive vulnerability (e.g., Gibb & Coles, 2005; Rose & Abramson, 1992). Consistent with this idea, mounting evidence reveals that such forms of adversity predict the emergence of cognitive vulnerability over time in youth (for reviews, see Abela & Hankin, 2008; Gibb & Coles, 2005). Research also documents information-processing biases in the offspring of depressed mothers. In one study, never-disordered daughters of depressed mothers selectively attended to negative emotional information, while never-disordered daughters of never-disordered mothers selectively attended to positive emotional information (Joormann, Talbot, & Gotlib, 2007). In another study, maternal depressive symptoms were associated with a negative bias in youth's processing of mother-relevant information (specifically for youth with heightened emotional reactivity to stress; Flynn & Rudolph, 2012). Offspring of mothers with a history of depression also show patterns of psychophysiological functioning suggestive of deficits in selective attention (Pérez-Edgar, Fox, Cohn, & Kovacs, 2006). Collectively, these studies support the idea that stressful life contexts foster the development of cognitive vulnerability, which may contribute to the intergenerational transmission of depression. Of course, it also is possible that childhood adversity and maternal depression reflect a genetic liability that is in-

stantiated in the form of cognitive vulnerability. Additional research is needed to determine the mechanisms through which adverse contexts and maternal depression heighten cognitive vulnerability.

DEVELOPMENTAL CHANGES

A quantitative review (Lakdawalla, Hankin, & Mermelstein, 2007) reveals that the cognitive vulnerability–depression link strengthens with age; this finding indicates the importance of understanding changes in the stability, consolidation, and predictive power of cognitive vulnerability across development. Several cognitive transformations during the adolescent transition may set the stage for increasing cognitive vulnerability (for reviews, see Abela & Hankin, 2008; Gibb & Coles, 2005; Jacobs et al., 2008). Cognitive vulnerability may emerge as children develop the capacity to engage in abstract reasoning, integrate information across situations and time, and make stable attributions about behavior. Cognitive processes also become more rigid across development, making it less likely that individuals will flexibly integrate schema-incongruent information; this rigidity may be intensified by normative increases in self-consciousness during adolescence. At the same time, a maturational gap emerges between emotional reactivity and cognitive regulatory capacity (Dahl, 2004), laying fertile ground for the cultivation of emotionally driven difficulties in regulatory focus (e.g., rumination) and unchecked negative inferences about stressful events. These maturational changes may intersect with increasing life stress during adolescence (Rudolph & Hammen, 1999) to set the stage for heightened cognitive vulnerability. Shifts also may occur in the association between cognitive vulnerability and stressors. Whereas cognitive vulnerability may emerge from stressors (Tram & Cole, 2000) or prior depressive symptoms (LaGrange et al., 2011; Pomerantz & Rudolph, 2003) earlier in development, it may interact with stressors later in development. Moreover, cognitive vulnerability contributes to the generation of stress (e.g., Eberhart, Auerbach, Bigda-Peyton, & Abela, 2011; Shih, Abela, & Starrs, 2009), highlighting the dynamic association between cognitions and stress.

Given evidence for distinct dimensions of cognitive vulnerability in youth (Ginsburg et al., 2009), it is possible that these dimensions coalesce into stable traits at different stages of development. Supporting this idea, research using an advanced quantitative modeling approach suggests that a reliable time-invariant (trait-like) component of negative attributional style does not emerge until early adolescence (Cole et al., 2008), whereas stable components of other negative cognitions (negative automatic thoughts, the negative cognitive triad) emerge during middle childhood (LaGrange et al., 2011). These findings underscore the need to distinguish various aspects of cognitive vulnerability, some of which may be more accessible and well developed earlier in childhood (and thus serve as predictors of depression), and others of which may involve more complex cognitive processes (e.g., making inferences about events) and do not stabilize until youth show certain cognitive advances during adolescence. Once the various components reach a certain level of stability (i.e., become trait-like), they may consolidate into a single set of interrelated vulnerabilities (Abela & Hankin, 2008).

Summary

Early research often failed to test key aspects of cognitive vulnerability models, such as the temporal precedence and stability of dysfunctional cognitive styles, the activation of cognitive vulnerability by negative mood states or stressors, and the accuracy of negative cognitions. Notable advances in the use of rigorous, prospective, multiwave, and experimental designs over the past decade address many of these limitations. As a result, the field has witnessed significant progress in addressing some of the ongoing controversies and affirming the position of cognitive theories as useful conceptual frameworks for understanding the etiology and persistence of depression. At the same time, recent advances illustrate the need to embed cognitive theories of depression within the context of dynamic developmental frameworks that explain the emergence, consolidation, and crystallization of cognitive vulnerability over time. Future efforts to refine and validate cognitive theories of depression must elucidate the independent, transactional, and interactive contributions of cognitive vulnerability and other risk factors (e.g., genetic, biological, emotion-regulating, and social processes) with the goal of developing integrative multilevel models.

Interpersonal Vulnerability to Depression

Interpersonal approaches to understanding depression posit that depression is fundamentally an interpersonal disorder (Coyne, 1976; Joiner & Timmons, 2009). Interpersonal difficulties are robust predictors and consequences of depression, and many other forms of vulnerability to depression (e.g., cognitive, emotional)

are expressed in interpersonal contexts. Furthermore, interpersonal vulnerability to depression may be of particular import for youth, whose relationships evolve dramatically over the course of childhood and adolescence. In recent years, substantial progress has been made in theory and research seeking to understand interpersonal vulnerability to depression in youth.

Theoretical Models

According to interpersonal theories of depression, originally developed to understand adult depression, depressed individuals both *react* and *contribute* to interpersonal difficulties. Specifically, impairment in social skills (e.g., excessive reassurance seeking, social withdrawal) and relationship disturbances (e.g., unsupportive or conflictual relationships, interpersonal stress) heighten vulnerability to depression. In turn, characteristics and behaviors of depressed individuals contribute to stress in relationships, aversive interpersonal encounters, and rejection, which maintain or promote depression over time (Coyne, 1976; Gotlib & Hammen, 1992; Joiner & Timmons, 2009). This cyclical process may help to explain high stability and recurrence of depression.

Extensions of these theories to youth provide developmentally sensitive accounts of the early origins of interpersonal disruption, as well as the continuously evolving interplay between interpersonal disruption and depression over time and across critical developmental stages (e.g., Cyranowski et al., 2000; Rudolph, Flynn, & Abaied, 2008). According to these models, early exposure to social adversity may set the stage for proximal interpersonal vulnerabilities in youth. These models take into account key developmental transitions, paying particular attention to ways in which biological (e.g., puberty, sex), cognitive (e.g., executive functioning, abstract reasoning), and social (e.g., increasing importance of peers and romance) development may exacerbate preexisting interpersonal vulnerability and contribute to the sharp increase in depression beginning in midadolescence. Finally, developmental perspectives on the interpersonal context of depression posit that interpersonal vulnerabilities may manifest themselves in a variety of relationship contexts, including family, peer, and romantic relationships.

Empirical Evidence

Longitudinal research supports both directions of influence—interpersonal impairments and problems predicting youth depression, and youth depression predicting incompetence and dysfunction in relationships—in multiple interpersonal domains. We first discuss research covering interpersonal stress across different domains and then focus on specific relationships, including those with family, peers, and romantic partners, focusing on links between proximal aspects of youth's relationships and depression.

INTERPERSONAL STRESS

Exposure to interpersonal stressors (pooled across multiple types of relationships) predicts subsequent depression in youth (e.g., Carter & Garber, 2011; Hankin, Mermelstein, & Roesch, 2007). Furthermore, the process of interpersonal stress generation, in which depressive characteristics and behaviors (e.g., excessive reassurance seeking, negative conceptions of relationships) disrupt relationships and create new interpersonal stressors, also contributes to the maintenance of depression over time (for a review, see Hammen, 2009a). Consistent with the idea that depression is particularly damaging to relationships, depressed youth are more likely to generate interpersonal than noninterpersonal stress (Hammen, 2009a; Rudolph, Flynn, Abaied, Groot, & Thompson, 2009).

FAMILY RELATIONSHIPS

Drawing from family systems (Cox & Paley, 1997) and attachment theory (Bowlby, 1969) perspectives, much of the research on interpersonal vulnerability to depression in youth focuses on the family context. Youth who are exposed to stressful and unsupportive family environments and maladaptive parenting are at heightened risk for developing depression. Families of depressed youth are more conflictual and less cohesive compared to families of nondepressed youth, and parents of depressed youth are more negative (i.e., unsupportive, hostile, or intrusive) in parent–child interactions compared to parents of nondepressed youth (for a review, see Abaied & Rudolph, 2014). Aspects of the family environment (e.g., high parent–child conflict, coercive and emotionally negative interactions among family members) and parenting behavior (e.g., low parental warmth, parent hostility, and psychological control) forecast subsequent depression in youth (e.g., Schwartz et al., 2011; Soenens et al., 2008; Stice, Ragan, & Randall, 2004), and a highly critical parenting style predicts the onset (Silk et al., 2009) and maintenance (McCleary & Sanford, 2002) of youth depression.

As predicted by interpersonal theories of depression, youth depressive symptoms also disrupt family environments. Youth depression predicts more stress in parent–child relationships (Raposa, Hammen, & Brennan, 2011), lower perceived family relationship quality (Lewinsohn, Rohde, et al., 2003), and lower perceived support from parents (Needham, 2007) over time. Furthermore, depressed youth perceive their parents as increasingly hostile, harsh, and inconsistent over time (Kim, Conger, Elder, & Lorenz, 2003). Observational research has revealed more negative interchanges, less positive reciprocity, and more negative reciprocity between depressed children and their parents than in nondepressed families (for a review, see Abaied & Rudolph, 2014). Thus depressed children's symptoms or dysfunctional behavior may evoke negative responses from their parents and perpetuate negative parent–child interactions over time. Providing some support for transactional associations, some research documents reciprocal effects between low perceived parent support and depressive symptoms (Allen et al., 2006; Branje, Hale, Frijns, & Meeus, 2010; cf. Stice et al., 2004). Therefore, substantial evidence supports problematic family relationships as both predictors and outcomes of youth depression.

PEER RELATIONSHIPS

Learning to build and maintain positive relationships with peers, with whom most youth spend a large portion of their time, is a key developmental task in childhood, and peer relationships become more central to self-worth and emotional well-being during the transition to adolescence (Laursen, 1996). Thus, in recent years, researchers have paid increasing attention to peer relationships as a context of vulnerability to depression. Building on early cross-sectional work identifying a variety of impairments in depressed children's relationships with peers (e.g., Rudolph & Clark, 2001; for a review, see Gotlib & Hammen, 1992), investigators have demonstrated that deficits in interpersonal behaviors within peer relationships, including excessive reassurance seeking, negative feedback seeking, social withdrawal, and ineffective responses to peer stressors contribute to subsequent depression (e.g., Agoston & Rudolph, 2011; Borelli & Prinstein, 2006; Prinstein et al., 2005). Furthermore, exposure to social difficulties such as peer rejection, exclusion, victimization, and poor-quality friendships predict heightened depressive symptoms over time (Burton, Stice, & Seely, 2004; Nolan, Flynn, & Garber, 2003; Rudolph, Troop-Gordon, Hessel, & Schmidt, 2011). Supporting early (Coyne, 1976) and updated (Joiner & Timmons, 2009) interpersonal theories of depression, depressive symptoms also are contagious within peer groups, such that depressive symptoms in one's peers predict increases in one's own depression over time, with stronger effects generally emerging for girls compared to boys (e.g., Conway, Rancourt, Adelman, Burk, & Prinstein, 2011; van Zalk, Kerr, Branje, Stattin, & Meeus, 2010). One mechanism of depression contagion may be "co-rumination" (i.e., extensively discussing problems and negative feelings). Co-rumination among friends, although associated with higher friendship quality, predicts subsequent heightened depressive symptoms among girls (Rose, 2002; Rose, Carlson, & Waller, 2007). Together, these findings indicate that impaired functioning within peer relationships constitutes a substantial risk factor for subsequent depression.

Depression also interferes with children's subsequent interpersonal functioning. Depressed children have difficulty negotiating peer conflicts, and they elicit negative affect and aversive responses from unfamiliar peers (e.g., Rudolph, Hammen, & Burge, 1994), suggesting that characteristics of depressed children undermine the quality of their interactions. Perhaps as a result, children with the highest levels of depressive symptoms are most likely to lose friends over time (van Zalk et al., 2010), and depressive symptoms prospectively predict less stable and poorer-quality friendships (Oppenheimer & Hankin, 2011; Prinstein, Borelli, Cheah, Simon, & Aikins, 2005). Providing insight into two pathways through which depression can undermine peer relationships in youth, Agoston and Rudolph (2013) found that socially helpless behavior (e.g., lack of social initiative and persistence in the face of social challenge) and aggressive behavior accounted for the prospective contribution of depressive symptoms to low social status over time. In sum, disruptions in peer relationships may be antecedents or consequences of depression in youth.

ROMANTIC RELATIONSHIPS

Despite the developmentally salient and normative nature of adolescent romantic attraction and involvement (Collins, 2003), a small but growing literature suggests that romantic involvement in adolescence represents a substantial risk factor for subsequent depression (Davila et al., 2009; Starr, Davila, et al., 2012). Davila (2008) proposes that many youth lack the resources

to cope effectively with the challenges associated with romance, leaving romantically involved youth vulnerable to depression; for example, romance often involves intense and potentially novel emotions (e.g., sexual attraction, passion, romantic love) and introduces a variety of stressors (e.g., rejection, breakups, initiation of sexual behavior).

Consistent with this view, stress in romantic relationships (Daley & Hammen, 2002), negative interactions with partners (La Greca & Harrison, 2005), and low levels of intimacy (Williams, Connolly, & Segal, 2001) are associated with vulnerability to depression above and beyond involvement in romance. Romantic breakups constitute a particularly robust predictor of depressive symptoms and the onset of depressive episodes (Joyner & Udry, 2000). Supportive, low-conflict family relationships protect romantically involved youth from depression (Steinberg & Davila, 2008), whereas excessive reassurance seeking (Starr & Davila, 2008) and a preoccupied relational style (Davila, Steinberg, Kachadourian, Cobb, & Fincham, 2004) exacerbate this association. Thus lack of access to social support for coping with romantic stress may help to explain the link between romance and depression. Some studies have found stronger links between romantic involvement and depression or internalizing symptoms among girls compared to boys, and among younger compared to older adolescents (e.g., Joyner & Udry, 2000; Zimmer-Gembeck, Siebenbruner, & Collins, 2001).

Depression also may foster dysfunction in romantic relationships. In adolescence, depression is associated with generation of subsequent stress in romantic relationships (Hankin et al., 2007) and with dysfunction in romantic relationships, such as partner relationship dissatisfaction and physical coercion (Rao, Hammen, & Daley, 1999). Similarly, Daley and Hammen (2002) found that late adolescent women's depressive symptoms predicted lower levels of emotional support from their partners over time. Thus preliminary evidence supports disruption in romantic relationships as not only an antecedent but also a consequence of depression.

Summary

Supporting interpersonal theories of depression, research indicates that functioning within multiple domains of relationships may both contribute to and result from depression in youth. Relevant aspects of interpersonal functioning include dysfunctional social behaviors, poor-quality or unsupportive relationships, negative interactions with others, and interpersonal stress. Despite the transactional nature of interpersonal theories of depression, the majority of longitudinal studies in this area have focused on unidirectional pathways, and those that have tested transactional pathways have yielded inconsistent results; some only found support for one direction of effect or the other (Agoston & Rudolph, 2013; Borelli & Prinstein, 2006; Oppenheimer & Hankin, 2011; Prinstein et al., 2005; Stice et al., 2004). Unexamined moderators known to contribute to depression (e.g., genetic risk, cognitive style, or pubertal development) may have masked transactional effects in these studies. Future efforts to uncover transactional pathways should focus on moderators that are highly relevant to the aspects of interpersonal vulnerability being studied. In addition, most research in this area focuses on one relationship, and research is needed to examine the relative impact of different interpersonal contexts on depression in youth. Finally, long-term longitudinal studies will provide much-needed empirical tests of the core predictions stemming from developmental theories of interpersonal vulnerability to depression, including the notion that proximal interpersonal vulnerability is a mechanism through which early social adversity sets the stage for depression in childhood and adolescence.

Early Social Adversity and Depression

Moving beyond the proximal interpersonal context, developmental psychopathology theories of depression increasingly consider the adverse long-term consequences of early social adversity. Both prospective and retrospective research implicates early exposure to adverse social environments—through parental depression, trauma, loss, maltreatment, insecure attachment, or family disruption—as a precursor to youth depression (e.g., Garber & Cole, 2010; Hazel, Hammen, Brennan, & Najman, 2008; for a review, see Goodman & Brand, 2009). To achieve a better understanding of the relevant explanatory mechanisms, recent efforts have focused on articulating how early adversity undermines youth development in ways that heighten subsequent risk for depression.

Models of Risk

Developmental scientists have proposed several nonspecific models of early risk with potential relevance for youth depression. O'Connor (2003) distinguishes

three models accounting for the long-term impact of early experience. Most relevant to understanding the effects of early adversity on depression are the "experience-adaptive" or "developmental programming" model and the "cumulative-effects" model. An experience-adaptive model holds that biological systems adapt to environment input, particularly during sensitive periods of development; moreover, this malleability is developmentally constrained, such that systems have difficulty readjusting to later changes in the environment (although they may be altered through direct intervention). A cumulative-effects model holds that early experiences have a long-term impact to the extent that these effects are reinforced or maintained by later events; this model includes both an additive-effects variant (the effects of later adversity add to the effects of earlier risks) and an interactive-effects variant (the effects of later adversity depend on the history of earlier risks). Boyce and Ellis's (2005) "biological sensitivity to context" model combines elements of the experience-adaptive and cumulative-effects models; specifically, this model suggests that early adversity calibrates the stress response system, such that youth exposed to stressful early social environments show heightened biological reactivity to later stress. In something of a departure from other risk models, this sensitivity is thought to exert risk-augmenting effects under subsequent stressful conditions, but risk-protective effects under subsequent supportive conditions.

Focusing more specifically on interpersonal processes and depression, Rudolph and colleagues (2008) present an integrative model wherein early family adversity (e.g., insecure parent–child attachment, parental depression) interferes with the development of adaptive interpersonal behaviors and fosters maladaptive interpersonal behaviors. These social-behavioral deficits cause youth to generate further disturbances in their relationships, which serve as proximal precursors of depression. Depressive symptoms further undermine interpersonal functioning, leading to the perpetuation or exacerbation of depression and risk for recurrence. More specific models of risk explain the contribution of particular forms of adversity, such as maternal depression (e.g., Hammen, 2009b) and maltreatment (Alink, Cicchetti, Kim, & Rogosch, 2009), to youth depression.

In recent years, efforts to understand long-term risk have considered how early adversity interacts with recent stress to confer vulnerability to depression (similar to the interactive cumulative-effects model). According to a "stress amplification" model, childhood adversity amplifies depressive reactions to recent stress; that is, youth with a history of adversity are presumed to demonstrate higher levels of depression than those without a history of adversity when exposed to severe but not mild recent stress. According to a "stress sensitization" model, childhood adversity reduces an individual's threshold for depressive reactions to recent stress; that is, youth with a history of adversity are presumed to require only mild stress to trigger depression, whereas youth without a history of adversity require more severe stress to trigger depression. Mounting evidence supports stress sensitization models, indicating that youth exposed to early adversity (e.g., parental abuse/neglect, multiple forms of family disruption) are more likely than those with no history of adversity to become depressed following exposure to mild or moderate levels of stress (e.g., Harkness, Bruce, & Lumley, 2006). One interesting study revealed stress amplification effects in prepubertal girls, but stress sensitization effects in girls progressing through puberty (Rudolph & Flynn, 2007), suggesting changes in sensitization across the adolescent transition. Of note, the stress sensitization effect (at least when declines in the strength of association between life stress and the onset of depression across episodes are examined) appears to be most pronounced in those at low genetic risk for depression, perhaps because those with high genetic risk are already sensitized to an initial onset of depression even in the presence of low levels of life stress (Kendler, Thornton, & Gardner, 2001).

Pathways of Risk

Building on research documenting stress sensitization and stress amplification effects, contemporary efforts have explored specific pathways through which these processes unfold (for a review, see Goodman & Brand, 2009). One line of research focuses on the idea that a history of prolonged or severe adversity sensitizes developing brain systems so as to create heightened reactivity to stress later in life (for a review, see Gunnar & Loman, 2011). Another line of research suggests that early adversity induces risk through exposure to maladaptive parent socialization patterns and stressful interpersonal contexts that undermine the normative development of sense of self, coping skills, and interpersonal competencies, leaving youth vulnerable in the face of future stress or even causing them to generate stressful events and circumstances (for reviews, see Hammen, 2009b; Rudolph et al., 2008).

Supporting biological risk pathways, exposure to early adversity predicts future dysregulation of the HPA axis (e.g., Cicchetti & Rogosch, 2012; Rao, Hammen, Ortiz, Chen, & Poland, 2008), deviant neural processing of emotional stimuli (Cicchetti & Curtis, 2005; Parker, Nelson, & The Bucharest Early Intervention Core Group, 2005), atypical patterns of frontal lobe activity (Dawson et al., 2003), and compromised immune system functioning (Shirtcliff, Coe, & Pollak, 2009). In addition to these functional variations, recent research reveals that lifetime exposure to adversity predicts differences in brain structure, specifically smaller volumes in specific regions of the PFC; moreover, reduced PFC volumes mediate the link between lifetime adversity and poorer executive function (spatial working memory) in adolescents (Hanson et al., 2012). In turn, individual differences in brain structure and function and poorer executive function are linked to depression, thereby supporting the idea that disruption in the systems underlying emotion regulation, stress reactivity, and cognitive self-regulation can serve as one mechanism through which early adversity contributes to future affective risk (for a review, see Forbes & Dahl, 2005).

Supporting psychosocial risk pathways, early adversity (e.g., maternal depression, maltreatment insecure attachment) exposes youth to several forms of interpersonal disruption, such as maladaptive socialization of emotion, problematic parent–child relationships, and stressful contexts (for reviews, see Abaied & Rudolph, 2014; Hammen, 2009b; Rudolph et al., 2008). For example, depressed, maltreating, and insecurely attached parents show less adaptive responses to their children's stress and expression of emotion (e.g., Abaied & Rudolph, 2010a; Edwards, Shipman, & Brown, 2005; Shaw et al., 2006) and engage in more hostile and less positive parenting (for a review, see Abaied & Rudolph, 2014). It is not surprising, therefore, that these youth suffer from a spectrum of functional deficits linked to depression, such as ineffective regulation and expression of emotion, social-behavioral deficits (e.g., seeking of negative feedback, avoidant responses to stress), relationship disturbances (e.g., poor friendships, peer victimization), and a tendency to generate interpersonal stress (for reviews, see Abaied & Rudolph, 2014; Rudolph et al., 2008). Providing explicit support for psychosocial pathways, research supports models in which disrupted parent–child relationships, heightened stress exposure, and social competence deficits mediate the contribution of early adversity, such as maternal depression, to subsequent youth depression (Garber & Cole, 2010; Hammen, Shih, & Brennan, 2004). However, because these studies have not used genetically informed designs, it is difficult to tease apart the extent to which the transmission of risk is due to socialization versus shared genetic vulnerability.

Resilience in the Face of Risk

Exposure to adverse conditions early in life, including parental depression, clearly does not uniformly disrupt the successful outcomes or healthy development of all children. Although studies of risk and vulnerability factors and processes predominate in studies of depression and dysfunctional outcomes, some research has examined predictors of resilient outcomes (including absence of depressive and other disorders), as well as protective processes. The classic study by Masten, Best, and Garmezy (1990) reported a resilience profile not specific to depression outcomes that generally has been replicated in studies of children of depressed mothers: "[They have] a positive relationship with a competent adult, they are good learners and problem-solvers, they are engaging to other people, and they have areas of competence and perceived efficacy valued by self or society" (p. 425). Masten and colleagues (2004) also found that along with parenting quality, IQ and positive personal characteristics and coping styles were predictive of resilient outcomes in a longitudinal study of high-risk youth. In a sample of youth at risk for depression due to maternal depression, Brennan, LeBrocque, and Hammen (2003) found that positive quality of parenting was a significant predictor of resilient outcomes by age 15. Pargas, Brennan, Hammen, and LeBrocque (2010) also found that positive maternal parenting, higher IQ, and personal qualities of high self-esteem and social competence with peers were protective factors in youth resilience at age 20. In the same high-risk sample, Hammen and colleagues (2008) found that youth with early-onset depression who "desisted" from further depression during the follow-up of 5 years to age 20 had significantly more positive peer and family relationships and higher self-esteem at age 15 than youth who had early-onset depression and also went on to have a recurrent/chronic course to age 20. Southwick, Vythilingam, and Charney (2005) reviewed a variety of positive personal traits and attributes, as well as genetic, neurobiological, and neuroendocrine factors also likely to play a role in protection against depression in the face of stressful experiences.

Summary

Emerging theory and research implicate early adversity as a pervasive and potent contributor to depression, propelling youth along a risky pathway characterized by significant intrapersonal and interpersonal disturbances that both heighten sensitivity to and increase the likelihood of future stressful experiences, thereby promoting risk for depression. These psychological, biological, and interpersonal disruptions may help explain the intergenerational transmission of risk. As noted, however, early adversity and consequent risks could, in part, reflect a shared genetic liability that accounts for vulnerability to depression. Understanding the intersection between genetic and environmental risks associated with early adversity will be a key direction for future research. Moreover, it will be important to determine whether the negative consequences of early social adversity depend on the timing of exposure, or whether they emerge during particular developmental stages. Given research implicating the adolescent transition as a pivotal interpersonal context of risk for depression (Rudolph, 2009; Rudolph et al., 2008), the long-term legacy of early adversity may be intensified during this stage, perhaps crystallizing into more severe and/or persistent forms of depression. Further research should build on initial studies of resilience to study both resistance to depression and deflection of the course of depression to less severe trajectories.

AN INTEGRATIVE DEVELOPMENTAL MODEL OF YOUTH DEPRESSION, AND DIRECTIONS FOR FURTHER STUDY

This chapter acknowledges the significant expansion of empirical research, using improved and innovative methodologies, in child and adolescent depression. There has been an increasing focus on developmentally sensitive models, although significant questions remain about the applicability of adult approaches to diagnosis and conceptualization, how childhood- and adolescent-onset depressions may differ, and especially, how development and experiences alter the neural and neuroendocrine systems relevant to depression. Because adolescent-onset depression is the "prototype" of most adult depression that is studied and treated, further understanding of developmental considerations will be fundamental to understanding recurrent adult depression—in short, a reversal of the typical "downward extension" models of adult depression applied to children. Three key methodological goals are encouraged for future studies:

1. Greater refinements in definitional issues of "depression" that address the challenges raised by heterogeneity, comorbidity, and the arbitrary lines between clinical and nonclinical depression, including transdiagnostic and endophenotype strategies. Such strategies also will assist in addressing the important question of the specificity of depressive outcomes, and tell us what is unique to the understanding and prediction of depression.
2. A greater emphasis on empirical studies with longitudinal designs, which will more precisely illuminate changes in vulnerabilities and clarify their mechanisms.
3. Further integrative studies that include multiple domains of vulnerabilities, to address the complex, transactional associations among biological, emotional, cognitive, and interpersonal processes. Such models are not difficult to construct, but are very challenging to test and validate through empirical investigations.

Figure 5.1 depicts one multidimensional developmental model of depression. The model is intended to highlight the complex and reciprocal interplay among diverse etiological influences on depression. Drawing from our empirical review, we have articulated a version of a model designed to encourage future directions. Temporally, the model commences with the genetic and environmental vulnerabilities the child faces from birth (or from prenatal life). Early life adversities may include traumatic experiences and severe abuse, but often include the more common problems of family instability; exposure to parental mental disorders and dysfunctional behavioral patterns; economic and social disadvantage; and harsh, insensitive parenting. Such adversities often occur early in life, but tend to be fairly chronic. These adversities shape individual vulnerabilities in biological, emotional, cognitive, and interpersonal functioning. Future research should increasingly and more precisely characterize the ways in which adversities exert maladaptive effects—and, ideally, define how particular adverse conditions operate at particular developmental stages to promote depression specifically.

Genetic factors have excited considerable attention throughout many aspects of human health and behav-

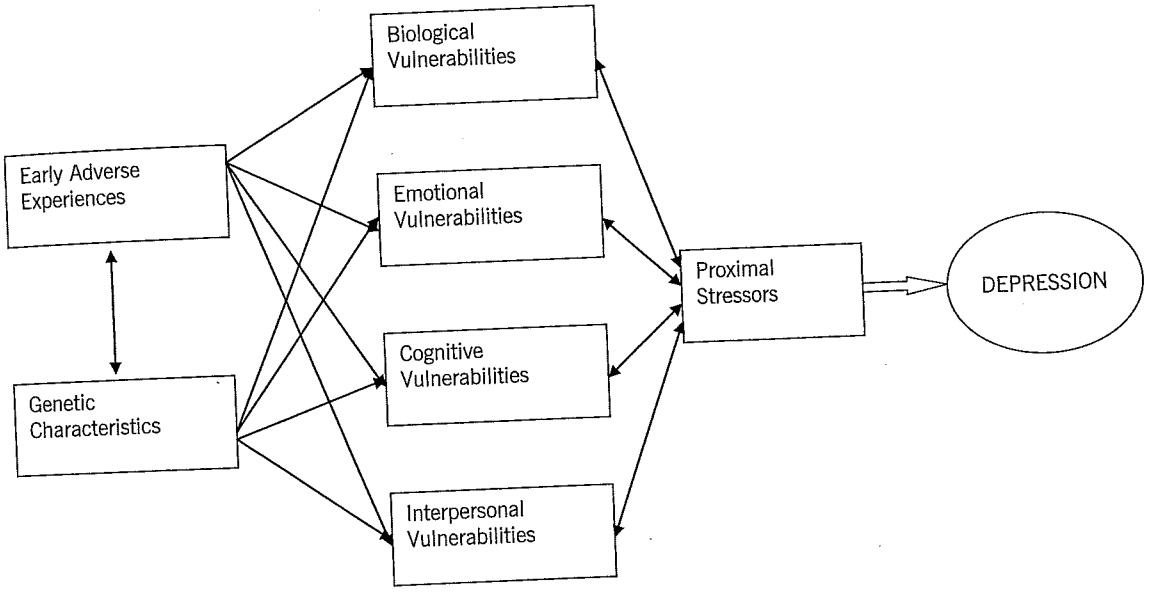

FIGURE 5.1. Multifactorial, transactional model of child and adolescent depression.

ior, and clearly represent a topic of obvious significance to our understanding of depression. There is substantial emerging evidence for heritable aspects of biological, emotional, cognitive, and interpersonal vulnerability. It is safe to predict that energetic efforts will be devoted to understanding the nature and mechanisms accounting for such genetic effects. In view of extensive evidence that the small effects of each of multiple genes are involved in human behavioral tendencies, the identification of candidate genes in youth depression along with the exploration of the various gene–environment interactions (and correlations) are likely to be of considerable interest to developmentally focused research.

The vulnerabilities depicted in Figure 5.1 are not meant to be exhaustive of possibilities for predicting risk for depression, but represent topics for which the literature so far has produced suggestive findings. Not shown in the figure because of their complexities are the multiple and bidirectional associations among the vulnerability factors themselves; nor is it easy to illustrate all the possible moderating and mediating pathways between the early adversity and genetic factors and the vulnerabilities to predict depression—although studies evaluating such complex pathways are clearly necessary.

In the model, the proximal predictor of depression consists of recent or ongoing stressful experiences, including both acute and chronic negative circumstances that typically trigger depression. Each of the vulnerability factors plays a role in how stress is construed or how it is processed at emotional and biological levels. Moreover, the vulnerability factors also play some role in the occurrence of stressors, not only in determining that an event is perceived as a stressor, but also in many cases contributing to maladaptive behaviors that cause stressors to occur. Also, individuals must deal with challenging circumstances but may lack the social, personal, biological, and cognitive resources needed to cope with or prevent stress effectively.

Depression is the outcome in the model, but it is also assumed that depression itself, as a commonly chronic or recurring condition, has deleterious effects on biological, emotional, cognitive, and interpersonal processes, thereby increasing or maintaining their likelihood for further negative impact on youth development. Imagine arrows issuing from depression back toward all the vulnerabilities, just as stress feeds both forward and backward. Indeed, a central question across levels of risk—including the biological substrate of depression—involves the effect of stress and prior

episodes of depression on the pathophysiology of depression. Depression is clearly a recurring problem for many sufferers, and perhaps depression experienced in adolescence represents a "degenerative" disorder that not only impedes normal developmental accomplishments but also heightens reactivity to future stressors.

The problem of depression in youth is a substantial one—not only because of its frequency and severity, but also because of the risk of a debilitating recurring disorder. Compounded with the impediments depression poses to achieving developmentally appropriate capabilities, depressed youth are likely to face their adulthood with impaired functioning in close relationships, parenting, and occupation, as well as emotional distress. We hope that further clarifications of etiological processes not only will serve the scientific goals of furthering the field's understanding of fundamental biological and psychosocial processes, but also will inform the interventions that are needed to interrupt the negative cycle.

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