Chapter 13

The Adolescent Surge in Depression and Emergence of Gender Differences

A Biocognitive Vulnerability-Stress Model in Developmental Context

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Alloy, L.B., & Abramson, L.Y. (2007). The adolescent surge in depression and emergence of gender differences: A biocognitive vulnerability-stress model in developmental context. In E. Walker & D. Romer (Eds.), Adolescent psychopathology and the developing brain (pp. 284-312). New York: Oxford University Press.

Depression is one of the most common forms of psychopathology (Kessler, 2002). Moreover, depression is highly recurrent (Judd, 1997) and associated with significant impairment (Greenberg et al., 1996; Gotlib & Hammen, 2002; Roy, Mitchell, & Wilhelm, 2001; Sullivan, LaCroix, Russo, & Walker, 2001). Indeed, due to depression's unique combination of high lifetime prevalence, early age of onset, high chronicity, and great role impairment (Kessler, 2000), the World Health Organization Global Burden of Disease Study ranked depression as the single most burdensome disease (Murray & Lopez, 1996).

Depression also is one of the most prevalent and serious problems faced by many adolescents. In contrast to the earlier belief that it could not occur in childhood or adolescence, it is now recognized that depression is a major mental health problem in adolescence (Compas, Connor, & Hinden, 1998; Kessler, Avenevoli, & Merikangas, 2001; Weissman et al., 1999). Not only is depression itself a major health problem for adolescents, it also is comorbid with and contributes to a wide range of other adolescent maladaptive outcomes and risk behaviors, including suicide, anxiety disorders, eating disorders, substance abuse, teen pregnancy and sexual risk-taking, and impairment in academic performance and family and social relationships (see Alloy, Zhu, & Abramson, 2003, for a review).

Surge in Depression and Emergence of Gender Differences in Adolescence

Adolescence, a transitional developmental period between childhood and adulthood, is characterized by more biological, psychological, and social role changes than any other stage of life except infancy (Holmbeck & Kendall, 2002). Developmental epidemiology has revealed two dramatic clinical phenomena of depression associated with adolescence that provide the point of departure for this chapter. First, whereas the rate of depression is low among children, there is a surge of depression during adolescence, with first episodes frequently occurring during this developmental period (Burke, Burke, Regier, & Rae, 1990). Prospective, longitudinal studies over the past decade (Hankin et al., 1998; Weissman et al., 1997) have shown that the rates of depression begin to increase dramatically in midadolescence, around age 13–14, and reach strikingly high levels by late adolescence, or age 18. Moreover, recent longitudinal studies have confirmed the continuity of depression from adolescence into adulthood (Lewinsohn, Rohde, Klein, & Seeley, 1999; Weissman et al. 1999), with formerly depressed adolescents showing very extensive impairment in young adulthood (Lewinsohn et al., 2003).

The second fact motivating this chapter is the robust finding of a gender difference in depression among adults. Twice as many adult women are depressed as adult men (Nolen-Hoeksema, 1990; Weissman & Klerman, 1977). Corroborating cross-sectional results (Nolen-Hoeksema & Girgus, 1994) with our prospective study, we (Hankin et al., 1998) found that the female preponderance in depression had clearly emerged by age 13-14. This gender gap in rates of depression widened dramatically between ages 14 and 18. By age 18, the sample exhibited the 2:1 ratio of greater female depression seen among adults. However, age may mask important developmental transitions (Rutter, 1989) that could more accurately pinpoint when the gender difference in depression emerges. In fact, pubertal development predicted the emergence of the gender difference better than age alone, as girls showed increased rates of depressive disorders after Tanner stage III (Angold, Costello, & Worthman, 1998).

Recent findings from two cross-sectional studies (Hayward, Gotlib, Schradley, & Litt, 1999; Siegel et al., 1998, 1999) suggest that ethnicity also must be considered in understanding the relation between puberty and the emergence of the female preponderance in depression. Specifically, gender differences in depression following puberty either do not occur or are weaker in African American and Latino adolescents. Two limitations of these studies, however, are that they were cross-sectional and assessed only depressive symptoms but not depressive episodes. Thus, it is not known whether ethnicity similarly would moderate the relationship between pubertal status and the emergence of gender differences in depression in longitudinal studies of onsets of depressive disorders.

Mechanisms Underlying the Surge of Depression and Emergence of Gender Differences in Adolescence

Why does depression surge so dramatically in adolescence, especially for females? Despite the public health and scientific significance of this question, the mechanisms underlying the adolescent surge and emergence of gender differences in depression are not understood well (Cyranowski, Frank, Young, & Shear, 2000; Hankin & Abramson, 2001; Nolen-Hoeksema & Girgus, 1994). It is puzzling that well corroborated theories of depression, when informed by adolescent development, have not as yet been applied to such core developmental depressive phenomena. In this regard, the cognitive vulnerability-stress model has been highly successful in elucidating the processes giving rise to depression, as well as generating powerful empirically supported therapies for it (Abramson et al., 2002). Thus, the overarching goal of this chapter is to examine the mechanisms underlying these developmental phenomena from the perspective of a biocognitive vulnerability-transactional stress model, embedded within a normative adolescent brain and cognitive development context.

A biocognitive vulnerability-transactional stress model of depression is especially plausible in explaining why many individuals become depressed for the first time during adolescence because some of the key etiological factors featured in the theory (e.g., Cognitive Vulnerability × Stress interaction, rumination, hopelessness) have just become developmentally operative during this period due to normative brain maturation and cognitive development (e.g., growth in attentional competence, working memory, hypothetical thinking, future orientation). Ironically, growth in cognitive competence during adolescence likely provides cognitive developmental "prerequisites" for the development of cognitive vulnerability to depression. Figure 13-1 provides an overview of the biocognitive vulnerability-transactional stress model that is developed throughout the remainder of this chapter.

Cognitive Vulnerability-Transactional Stress Model of Depression

The cognitive vulnerability-transactional stress model (Hankin & Abramson, 2001) is a developmentally sensitive elaboration of the two highly successful cognitive theories of depression, hopelessness theory (Abramson, Metalsky, & Alloy, 1989) and Beck's (1987) theory (see central portion of figure 13-1). The essence of this model is that individuals with cognitive vulnerability are more likely to become depressed than nonvulnerable individuals when they confront negative events and make negative inferences about the cause, consequences, or self-implications of the events (negative cognitive style). An individual exhibiting cognitive vulnerability who gets fired might attribute this negative event to stable, global causes (e.g., incompetence) and infer that she never will get another job and is worth-

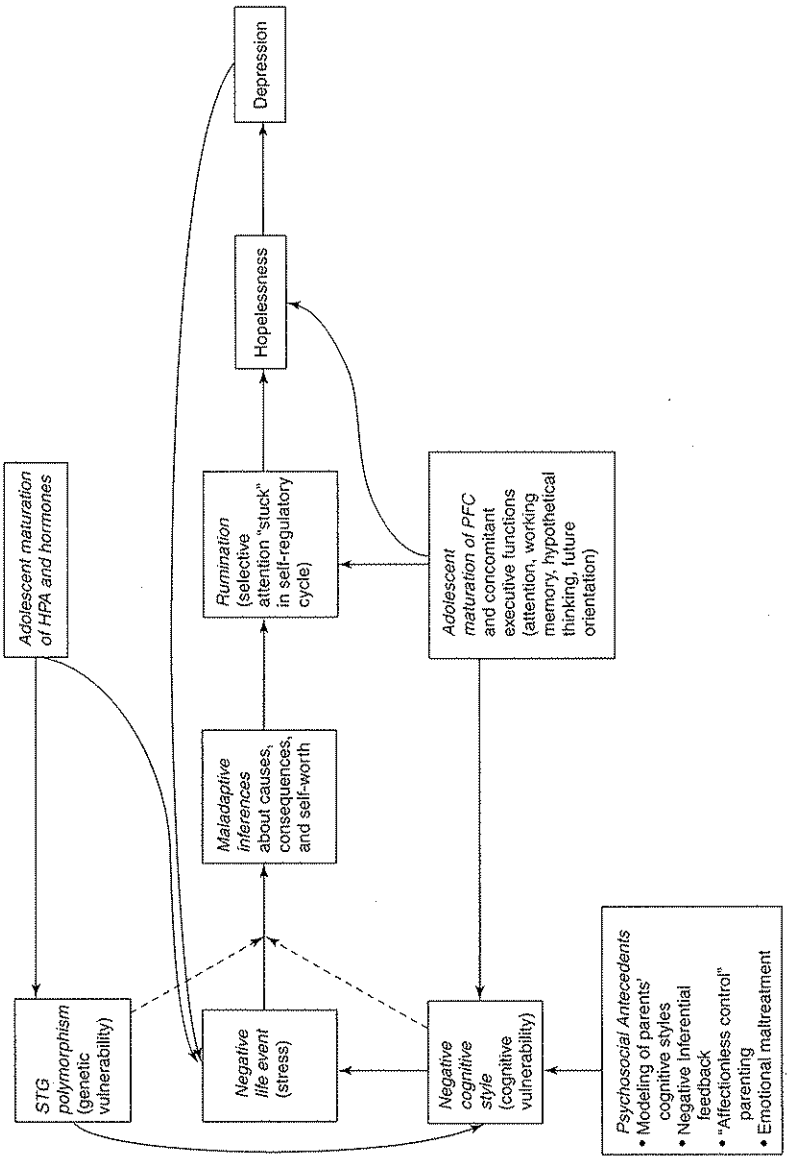

Figure 13-1 An integrated biological and cognitive vulnerability-stress model of depression in the context of normative adolescent brain and cognitive development. The causal relations proposed in the model are described throughout the chapter. Dashed arrows indicate interactive effects; thus, the model includes both Cognitive vulnerability × Stress and Genetic vulnerability × Stress interactions. Solid arrows indicate direct effects.

less. This model is a classic vulnerability-stress model because negative cognitive styles (the vulnerability) contribute only to the occurrence of depression in the presence, but not the absence, of a negative event (the stress). An additional feature of the model is that negative cognitions such as hopelessness mediate the link between the Cognitive Vulnerability × Stress component and the onset of depression. The model also features a transactional process in which increases in depression or cognitive vulnerability itself can contribute to the creation of further dependent, negative events.

We (Abramson et al., 2002; MacCoon, Abramson, Mezulis, Hankin, & Alloy, 2006) elaborated the model to emphasize selective attention in the causal chain and connect the concepts of cognitive vulnerability and rumination, another cognitive factor shown to be important in depression onset (Just & Alloy, 1997; Nolen-Hoeksema, 2000; Spasojevic & Alloy, 2001), duration (Nolen-Hoeksema, 1991), and severity (Just & Alloy, 1997), as well as in gender differences in depression among adults (Nolen-Hoeksema & Jackson, 2001). Self-regulation theorists (Carver & Scheier, 1998) emphasize that when faced with a negative event, it is adaptive to switch attention to this event, find a resolution, and then continue goal-directed behavior (i.e., the self-regulatory cycle). Selective attention often remains focused on the negative event until it is resolved or reduced. We highlighted three ways to exit this self-regulatory cycle: generate a solution to the problem, decrease the importance of the event, or distract attention away from the problem. Cognitively vulnerable individuals should have difficulty with all three exits due to their negative inferences.

For example, if a cognitively vulnerable adolescent attributes not getting a date to "ugliness," no solution is readily available. Instead, cognitively vulnerable individuals become "stuck" in the self-regulatory cycle with their attention focused on negative cognitive content because the inferences they generate in response to negative events lead only to yet further perceived problems (e.g., "no one will marry me because I am so ugly") rather than to resolutions. Such self-regulatory perseveration (Pyszczynski & Greenberg, 1987) constitutes rumination because selective attention remains focused on negative content, which, in turn, should result in the spiral into clinically significant depression. This self-regulatory perspective, then, highlights rumination as mediating the effects of cognitive vulnerability on depression.

Evidence for the Cognitive Vulnerability-Transactional Stress Model

The cognitive vulnerability-transactional stress model has garnered very considerable empirical support. Much of the most important evidence for the theory comes from the Temple–Wisconsin Cognitive Vulnerability to Depression (CVD) Project (Alloy & Abramson, 1999) and related studies. The CVD Project is a landmark, collaborative, two-site study that uses a behavioral high-risk design

(Alloy, Lipman, & Abramson, 1992) to test the cognitive vulnerability and other etiological hypotheses of the major cognitive theories of depression. More generally, the CVD Project examines the cognitive, psychosocial, and developmental risk factors and mechanisms in unipolar depression. We highlight the major findings here.

In the CVD Project, late adolescents (n = 349) at a major "age of risk" for depression and making the transition from late adolescence to early adulthood were followed for a total of 5.5 years. Ethnically diverse male and female university freshmen who were nondepressed and had no other Axis I psychiatric disorders at the outset of the study were selected to be at high risk (HR; n = 173) or low risk (LR; n = 176) for depression based on the presence versus absence of negative cognitive styles (negative inferential styles and dysfunctional attitudes).

Although nondepressed at the outset of the CVD Project, participants with past depression were retained in the final sample as long as they had remitted fully from any past depressive episode, to avoid having an unrepresentative group of HR participants. A powerful test of the cognitive vulnerability hypothesis is provided by our prospective findings. More than half of CVD Project participants entered college with no prior history of depression. These individuals potentially could experience their very first episode of depression during the follow-up. Consistent with the cognitive vulnerability hypothesis, Alloy et al. (1999, 2006) found that HR participants showed a greater likelihood than LR participants of a first onset of major depression (MD), minor depression (MiD), and hopelessness depression (HD; odds ratios = 5.6–11.7). These findings are especially important because they are based on a truly prospective test, uncontaminated by prior history of depression. Among participants with past depression, Alloy et al. (1999, 2006) found that HR participants were more likely than LR participants to develop recurrences of MD, MiD, and HD (odds ratios = 3.1–4.1). There were no risk group differences in prospective onsets of anxiety or other Axis I disorders in either the first onset or recurrence subsamples. However, in the full sample, the HR individuals did have a greater likelihood of an onset of anxiety disorder comorbid with depression, but not of anxiety disorders alone. In addition, Abramson et al. (1998) found that the HR group was more likely to exhibit suicidal ideation and attempts during the follow-up, mediated by hopelessness, than the LR group. These results are important because they provide the first demonstration that negative cognitive styles indeed appear to confer vulnerability to clinically significant depression and suicidality.

In elaborating our cognitive vulnerability-transactional stress model to place it in a self-regulatory context, we highlighted rumination as a form of self-regulatory perseveration that mediates the effects of cognitive vulnerability on depression. Consistent with this elaboration, Spasojevic and Alloy (2001) found that a ruminative response style mediated between cognitive risk and the development of prospective onsets of MD. Also, Robinson and Alloy (2003) extended the

rumination hypothesis and suggested that cognitively HR individuals who tend to ruminate on their negative cognitions when stressful life events occur (stress-reactive rumination, SRR), and thereby recursively activate their negative cognitions, would be more likely to become depressed. Consistent with this proposed extension, HR participants who were also high in SRR were more likely to have a past history and prospective onsets of MD and HD than were HR participants low in SRR or LR participants regardless of their SRR (Alloy et al., 2000; Robinson & Alloy, 2003). These findings indicate that rumination may act as both a mediator and moderator of cognitive vulnerability.

In the cognitive vulnerability-transactional stress model, negative cognitive styles are hypothesized to confer vulnerability to depression when individuals confront negative life events and this Vulnerability × Stress interaction should be mediated by hopelessness. In studies of depressive symptoms, we found that negative cognitive styles interacted with negative life events to predict prospective increases in depressive symptoms, mediated by hopelessness (Alloy & Clements, 1998; Alloy, Just, & Panzarella, 1997; Metalsky, Joiner, Hardin, & Abramson, 1993). In addition, we found that HR participants who experienced high stress were about 2.5 times more likely to have an onset of MD/MiD and HD, mediated by hopelessness, than HR participants who experienced low stress or LR participants regardless of stress. We also tested the transactional part (stress-generation hypothesis) of the cognitive vulnerability-transactional stress model, which suggests that cognitively vulnerable participants generate stressful events in their lives. Safford, Alloy, Abramson, and Crossfield (2006) found that, controlling for current and past depression, HR participants both generated more controllable events dependent on their behavior and experienced more fateful events independent of their behavior, thereby increasing the likelihood that their vulnerability will be translated into depression. Thus, cognitively vulnerable individuals experience more negative events and then interpret them more negatively as well (i.e., a "two-hit" model).

In addition, according to the cognitive vulnerability-stress model, people with negative cognitive styles are vulnerable to depression in part because they tend to engage in negatively toned information processing about themselves when they encounter stressful events. Accordingly, Alloy et al. (1997) found that relative to LR participants, HR participants showed preferential processing of self-referent negative depression-relevant information (e.g., faster processing and better recall of content involving themes of incompetence, worthlessness, and low motivation). These findings provide converging evidence for information processing effects of cognitive styles on laboratory tasks adapted from cognitive science paradigms and, thus, support the construct validity of the cognitive style questionnaire measures.

Much evidence indicates that social support buffers against depression when people experience stress (Cohen & Wills, 1985). Panzarella, Alloy, and Whitehouse (in press) hypothesized that social support buffers against depression by prevent-

ing the development of hopelessness through two mechanisms and, in particular, by providing positive or "adaptive" inferential feedback (IF) that promotes benign inferences about the causes, consequences, and meaning of negative events, rather than depressogenic ones. In contrast, negative or maladaptive IF from others should promote depressogenic inferences. For example, a person may express the following depressogenic inference to a close friend: "Now, I'll never get promoted; I am so stupid, I always foul things up." The friend could offer an adaptive inferential alternative, e.g., "She will forget about it in no time, and you will still be promoted because most of your work is really good. You are not stupid; your boss is just very difficult to get along with, and you are doing better at it all the time," that would lead the affected individual to reevaluate her thinking, thereby modifying the original maladaptive inference or at least decreasing its certainty and thereby the likelihood of hopelessness.

Consistent with prediction, Panzarella et al. found that high levels of adaptive IF prospectively predicted less negative inferences for actual stressful events experienced and less negative inferential styles during the follow-up. Moreover, participants who were HR experienced a high number of stressful events and had more negative IF from others were more likely to become hopeless and depressed than participants with 0, 1, or 2 of these vulnerability factors (Risk × Stress × IF interaction). In addition, in an experimental design, we found that participants whose partners were taught to deliver positive IF showed reduced depressive symptoms and inferences following a laboratory failure compared to participants whose partners provided general or no social support (Dobkin, Panzarella, Nesbitt, & Alloy, 2004).

What are the developmental origins of cognitive vulnerability to depression? We highlight the most relevant developmental findings from the CVD Project here (but see Alloy et al., 2004, for a detailed review; see also the bottom left portion of figure 13-1).

In an examination of the familial origins of negative cognitive styles, Abramson et al. (2006) found that HR participants' mothers had greater lifetime histories of depression than LR participants' mothers (35% vs. 18%) based both on direct interviews of mothers and offspring reports. HR participants' fathers were marginally more likely to have a history of depression than LR participants' fathers (18% vs. 12%) based on offspring report only.

Individuals may develop negative cognitive styles through a variety of familial socialization practices. If modeling of parents' styles is a contributor to the development of cognitive vulnerability to depression, then offspring's cognitive styles should correlate with those of their parents. In addition to modeling, the inferential feedback (IF) parents provide to their children about causes and consequences of negative events in the child's life may contribute to the child's cognitive risk for depression, such that offspring's cognitive styles will be associated with their parents' IF styles. Finally, negative parenting practices, such as "affectionless

control" (Parker, 1983), may also contribute to development of cognitive vulnerability to depression in offspring.

Alloy et al. (2001) examined the modeling, feedback, and parenting hypotheses. Consistent with modeling, mothers of HR participants had more negative cognitive styles, particularly dysfunctional attitudes, than mothers of LR participants. Supporting the feedback hypothesis, according to both child and parent reports on the Parental Attributions for Children's Events (PACE) Scale (Alloy et al., 2001), both mothers and fathers of HR participants provided more negative IF about causes and consequences of negative life events that happened to their child than did mothers and fathers of LR participants (see also Crossfield, Alloy, Abramson, & Gibb, 2002). Supporting the parenting hypothesis, according both to child and parent reports on the Children's Report of Parental Behavior Inventory (Schaefer, 1965), fathers of HR participants showed less warmth in raising their child than did fathers of LR participants (Alloy et al., 2001). Moreover, negative parental IF and fathers' low warmth predicted prospective onsets of depressive episodes in their offspring during the followup period, mediated by the offspring's cognitive risk status (Alloy et al., 2001).

Rose and Abramson (1992) hypothesized that a developmental history of maltreatment may contribute to the origins of cognitive vulnerability to depression. They hypothesized that emotional abuse should be especially likely to lead to development of negative cognitive styles because the depressive cognitions (e.g., "You're so stupid; you'll never amount to anything") are directly supplied to the child by the abuser. Consistent with the maltreatment hypothesis, we found that HR participants reported more emotional, but not physical or sexual, maltreatment than LR participants (Gibb et al., 2001a, 2001b). In addition, controlling for participants' initial depressive symptoms, a reported history of emotional maltreatment predicted onsets of MD, HD, and levels of suicidal ideation across the prospective follow-up, mediated totally or in part by their cognitive vulnerability and hopelessness (Gibb et al., 2001a, 2001b). Moreover, controlling for parental depression, parental cognitive styles, and parental abuse, peer victimization was also associated with cognitive HR status (Gibb, Abramson, & Alloy, 2004), suggesting that the association of emotional maltreatment with cognitive vulnerability is not entirely due to genetic effects or a negative family environment in general.

Application of the Cognitive Vulnerability-Stress Model to the Surge in Depression and Emergence of Gender Differences in Adolescence

The five constructs in the cognitive vulnerability-transactional stress model are negative life events (stress), cognitive vulnerability, rumination, Cognitive Vulnerability × Stress interaction, and hopelessness. If the model explains the adolescent surge in depression and the emergence of gender differences, then increases in levels or operation or "consolidation" of one or more of these variables should

precede the occurrence of these developmental phenomena. Adolescence typically generates more turmoil than childhood or adulthood. A developmental rise in the number of negative life events occurs after age 13 for both boys and girls (Garber, Keiley, & Martin, 2002; but see Compas, Davis, & Forsyth, 1985), but especially for adolescent girls (Ge et al., 1994) and especially in the interpersonal domain (Hankin & Abramson, 2001). Finally, depressed adolescent females generate interpersonal, negative events at a high rate (transactional-stress component; Hankin & Abramson, 2001). To understand the role of stress in contributing to the surge in depression and emergence of gender differences in adolescence, it is crucial that future studies remedy problems associated with prior life events research (e.g., poor sensitivity in dating event and symptom onset, lack of uniformity in event definitions across participants) and examine the same children longitudinally, thereby permitting construction of individual trajectories of growth in stress across adolescence and comparison of such trajectories with those for hopelessness and depression. Because many depression theories feature negative life events, it is also important to see whether the cognitive factors unique to the cognitive vulnerability-stress model show relevant developmental changes that proximally precede the surge in depression and emergence of gender differences in adolescence.

Preliminary indications suggest that cognitive vulnerability may "consolidate" (relative stability over time and consistency across situations) by adolescence and, thus, be accessible for the Cognitive Vulnerability × Stress interaction (Abramson et al., 2002; Gibb et al., 2006). A longitudinal study of third–eighth graders showed that attributional style became more stable in the later grades and interacted with stress to predict depression in older but not younger children (Nolen-Hoeksema, Girgus, & Seligman, 1992; Turner & Cole, 1994). Also, due to achievement of formal operations and the ability to contemplate the future, beginning in early adolescence, children can experience hopelessness, the mediating link in the chain culminating in depression (Hankin, Abramson, & Siler, 2001; Kazdin et al., 1983). Moreover, work demonstrating decreases in attributional positivity and optimism as children transition into adolescence (Mezulis, Abramson, Hyde, & Hankin, 2004; Stipek & MacIver, 1989) suggests that cognitive vulnerability may increase during this transition and, in turn, contribute to the surge in depression (see also Garber et al., 2002; Gibb et al., in press).

Developmental changes in cognitive vulnerability may contribute to the emergence of gender differences in depression in adolescence. Girls show greater rumination than boys postpuberty, but not before (Broderick, 1998; Smith, Floyd, Alloy, Hughes, & Neeren, 2006). Whereas prior research with psychometrically inadequate instruments typically has failed to reveal more negative cognitive styles among adolescent females than males, we recently developed a psychometrically superior measure of all three components of cognitive style for adolescents that did reveal more negative styles among adolescent females (Floyd, Alloy, Smith, Neeren & Thorell, 2006; Hankin & Abramson, 2002). Moreover,

in three cross-sectional studies, adolescent females' more negative cognitive styles and rumination mediated the gender difference in depressive symptoms (Floyd et al., 2006; Hankin & Abramson, 2002; Smith et al., 2006). Thus, preliminary support exists for the hypothesis that adolescent females may exhibit greater cognitive vulnerability than adolescent males, which contributes to the gender difference in adolescent depression.

Whereas prior work focused on cognitive vulnerability in achievement and interpersonal domains, physical attractiveness and body satisfaction may be very motivationally significant for adolescent girls (Floyd et al., 2006; Hankin & Abramson, 2001), who may exhibit high levels of cognitive vulnerability in this domain, which may contribute to their greater depression. Accordingly, adolescent girls showed greater rumination (Mezulis, Abramson, & Hyde, 2002) and more negative cognitive styles in the physical attractiveness domain than did their counterpart males (Hankin & Abramson, 2002). Converging evidence suggests that body dissatisfaction contributes to the gender difference in depression among adolescents (Floyd et al., 2006; Hankin & Abramson, 2001; Smith et al., 2006). For many girls puberty is negative because the associated physical changes (weight gain and increased adiposity) move them away from the thin female ideal currently popularized in modern Western society (Floyd et al., 2006; Nolen-Hoeksema & Girgus, 1994; Rierdan, Koff, & Stubbs, 1989). In contrast, boys tend to view puberty as desired given its association with valued physical characteristics such as increased muscularity and lean body mass (O'Dea & Abraham, 1999; Petersen & Taylor, 1980).

Thus, in our model, bodily changes that accompany pubertal development are important to the surge in depression and emergence of gender differences, not just hormonal changes or other components of puberty per se. These considerations also have implications for understanding ethnicity effects on the puberty-gender differences in depression association. The increased body fat accumulated during puberty is more disappointing for Caucasian than for African American girls (Casper & Offer, 1990; Halpern, Udry, Campbell, & Suchindran, 1999; Parker et al., 1995). Thus, puberty may not initiate the causal chain to depression among African American girls (Floyd et al., 2006).

The cognitive vulnerability-stress model suggests that a history of maladaptive inferential feedback about the causes and consequences of stressful events from significant others should contribute to development of cognitive vulnerability. Accordingly, Alloy et al. (2001) found that a reported childhood history of negative inferential feedback from parents was associated with negative cognitive styles and vulnerability to depression in the adolescent offspring. Negative inferential feedback from others may represent the milder end of a continuum of negative emotional feedback with emotional abuse at the extreme end (Alloy et al., 2001). In a developmental extension of the hopelessness theory, Rose and Abramson (1992) hypothesized that recurrent childhood abuse, particularly emotional abuse,

would lead to the development of cognitive vulnerability. We found preliminary support for this hypothesis with both retrospective (Gibb et al., 2004; Gibb et al., 2001a, 2001b; Spasojevic & Alloy, 2002) and prospective (Gibb et al., 2006) designs.

Negative inferential and emotional feedback from others may be especially likely to be internalized in early adolescence and contribute to the formation of cognitive vulnerability. Peers become increasingly important beginning in early adolescence (Harris, 1995; Steinberg, 2002), and rates of negative emotional feedback from peers, including teasing, harassment, rejection, and derogation (i.e., "relational aggression"; Crick & Grotpeter, 1996), rise at this time, especially among adolescent girls (e.g., Crick & Grotpeter, 1996). Thus, negative emotional feedback from peers, in particular, may contribute to both the adolescent surge in depression and emergence of gender differences (see Liu & Kaplan, 1999).

Placing the Cognitive Vulnerability-Stress Model in a Normative Adolescent Brain and Cognitive Development Context

Developmental psychopathologists (Cicchetti & Rogosch, 2002; Steinberg, 2002; Steinberg et al., 2004) and developmental neuroscientists (Casey, Tottenham, Liston, & Durston, 2005b; Walker, Sabuwalla, & Huot, 2004) emphasize that it is critical to apply knowledge of normative adolescent cognitive and brain development to the study of psychopathology in adolescence. On this view, an understanding of depression in adolescence from a cognitive vulnerability-transactional stress perspective must proceed with an explicit recognition of the brain maturation and concomitant cognitive capacities and attainments of the adolescent. It is well established that brain development and cognitive maturation occur concurrently in childhood and adolescence (Casey, Giedd, & Thomas, 2000; Sowell, Delis, Stiles, & Jernigan, 2001; Spear, 2000). Whereas prior work has tracked clinical phenomena as a function of age, little, if any, work has tracked changes in clinical phenomena as a function of cognitive development during adolescence (Steinberg, 2002). An important goal of this chapter is to embed the cognitive vulnerability-stress model in a normative adolescent brain and cognitive development context. Placed in such a context, some of the key etiological factors in the model (e.g., cognitive vulnerability, rumination, hopelessness) have just become developmentally operative during adolescence due to normative brain maturation and concomitant cognitive development (see bottom right portion of figure 13-1).

Contemporary noninvasive neuroimaging methods, such as magnetic resonance imaging (MRI) and diffusion tensor imaging (DTI), have provided evidence of changes in structural architecture and functional organization of the developing brain in vivo, as well as linkages between brain maturation and increases in cognitive

competencies (Casey, Galvan, & Hare, 2005a; Casey et al., 2005b; Liston et al., 2003; Nagy, Westerberg, & Klingberg, 2004). Longitudinal MRI studies show that cognitive milestones in development parallel the sequence in which the cortex matures (Giedd, 2004; Gogtay et al., 2004; Sowell et al., 2003, 2004). Regions subserving primary functions such as motor and sensory systems mature earliest and higher-order association areas, such as the prefrontal cortex (PFC), that integrate sensorimotor processes and control "executive functions" such as self-regulation, attention, working memory, and decision making mature more slowly, and not completely, until early adulthood (Casey et al., 2005a, 2005b; Gogtay et al., 2004; Sowell et al., 2004).

During adolescence, frontal lobe gray matter volume, representing dense concentrations of neuronal cell bodies, begins to decline following a rise throughout childhood, peaking at about age 12 (around puberty; Casey et al., 2005a; Giedd, 2004; Gogtay et al., 2004; Sowell et al., 2003; 2004). Gray matter loss during adolescence is thought to involve synaptic pruning and the elimination of connections (Casey et al., 2005b; Giedd et al., 1996; Pfefferbaum et al., 1994). Concomitant with the loss of gray matter, PFC white matter volume increases throughout adolescence until young adulthood and may reflect ongoing myelination of axons, promoting neuronal conduction and signal transmission (Casey et al., 2005b; Giedd et al., 1999; Gogtay et al., 2004; Paus et al., 1999; Thompson et al., 2000). The gray matter volume loss and white matter volume increases occur in parallel, suggesting that connections are being fine-tuned with the elimination of extra synapses and the strengthening of the relevant connections (Casey et al., 2005b).

Moreover, there are sex differences in PFC maturation and function that begin in adolescence and continue into early adulthood. Males have been found to show greater loss of PFC gray matter volume and greater increase of PFC white matter volume compared with females as a function of age and pubertal status (De Bellis et al., 2001; Giedd et al., 1999). In addition, there are sex-specific patterns of development of PFC activation to emotionally salient stimuli (i.e., emotional faces; Killgore, & Yurgelun-Todd, 2004; McClure et al., 2004). Normal pubertal development is associated with large increases in sex hormones and glucocorticoids (Walker et al., 2004), which influence brain maturation (De Bellis et al., 2001; Walker et al., 2004), in part through their effects on gene expression (Walker et al., 2004). Thus, the maturational and functional changes in the adolescent brain may be linked to the surge in depression that occurs in adolescence, and the sex differences in adolescent brain development and function may be associated with the emerging gender differences in depression during this developmental period.

The developmental changes in cortical development have been found to correlate with cognitive performance measures (Casey, Trainor, Orendi et al., 1997b, 2005b; Sowell et al., 2001). The fine-tuning of PFC structural architecture during adolescence observed in MRI studies is associated functionally (fMRI) with a shift

from diffuse recruitment of cortical regions by children performing cognitive tasks involving executive functions to more focal recruitment of PFC regions specifically implicated in cognitive control by adolescents (Brown et al., 2005; Casey et al., 2005a; Durston et al., 2005). Specifically, we hypothesize that four cognitive competencies (attentional executive functions, working memory, hypothetical thinking, and future orientation) attained during adolescence and linked to maturation of the PFC are cognitive developmental "prerequisites" for the Cognitive Vulnerability × Stress interaction to "pack its depressogenic punch." Ironically, adolescents' increased brain maturation and cognitive competence may come with a cost. It puts them at greater risk for depression than they were in childhood.

Earlier, we showed how attention is a self-regulatory mechanism in the causal chain of the cognitive vulnerability-stress model. Cognitively vulnerable individuals become stuck in the self-regulatory cycle with their attention focused on negative cognitive content (i.e., rumination; Abramson et al., 2002; MacCoon et al., 2006). An implication is that a cognitively vulnerable individual must have achieved substantial attentional executive functions in order for the Cognitive Vulnerability × Stress interaction to lead to full-blown depression. A cognitively vulnerable child who has not developed sufficient competence in selective and sustained attention may generate negative inferences when faced with a negative event but will not remain focused on such inferences and, thus, will not be as likely to suffer their depressogenic effects. We suggest that normative cognitive development of self-regulatory executive functions (i.e., sustained and selective attention and executive control over attentional switching) is a prerequisite for adolescents to fully engage in attempts to self-regulate negative affect and, thus, for full-blown rumination to occur (Abramson et al., 2002; Steinberg et al., 2004). Attentional processes are known to become more efficient with age and continue to develop through adolescence (Casey, Trainor, Giedd et al.et al., 1997a). Adolescent developmental maturation of the medial PFC and anterior cingulate cortex (ACC) is centrally involved in improved selective attention performance (Botvinik et al., 2001; Casey, Trainor, Giedd et al., 1997a). Thus, as attentional executive control develops during adolescence, rumination and the Cognitive Vulnerability × Stress interaction can fully "pack their punch" in contributing to the onset of and emergence of gender differences in depression.

Similarly, normative development of working memory is essential for maintaining information and the present context in mind (Cohen & Servan-Schreiber, 1992; Kimberg & Farah, 1993) and, thus, is also an important cognitive capacity underlying self-regulation. Thus, increases in working memory skills should also be a prerequisite for adolescents to fully engage in self-regulation and full-blown rumination. Working memory is most reliably associated with activation of the dorsolateral PFC (Owen, 2000).

With the advent of formal operations in adolescence comes the ability to think about possibilities rather than only concrete realities—in other words, abstract,

hypothetical thinking. Adolescents develop greater competence in generating options, viewing situations from many perspectives, and anticipating potential consequences of decisions (Keating, 1990, 2004). Such increased competency in hypothetical thinking is also subserved by maturation of the PFC and should be a prerequisite for generating negative implications of stressful events and for experiencing hopelessness (Steinberg, 2002). In addition, in order to experience hopelessness, the proximal cause of depressive symptoms in the cognitive vulnerability-stress model (Abramson et al., 1989), children must develop the normative capacity to think about the future, also a likely outgrowth of PFC maturation in adolescence.

Our hypotheses regarding the dependence of the adolescent surge in depression and emergence of gender differences on the normative cognitive development of executive functions subserved by PFC maturation suggest important directions for future research. Specifically, prospective longitudinal studies are needed that track the trajectories of development of executive functions (attentional competence, working memory, abstract, hypothetical thinking, future orientation) during the transition to adolescence and relate these trajectories to the trajectories of development of negative cognitive styles, rumination, and hopelessness, and, in turn, depression.

Placing the Cognitive Vulnerability-Stress Model in a Genetic Context

Genetic Vulnerability-Stress Model of Depression

It has been well established that genetic factors contribute to depression (e.g., Kendler et al., 1992). However, much less is known about the processes by which genetic vulnerability culminates in depression. Shedding light on this issue, Kendler et al. (1995) demonstrated in an adult twin sample that genes, assessed in the aggregate, affected sensitivity to the depressogenic effects of negative life events. Genetically high risk individuals were more likely to become depressed following negative life events than were their low risk counterparts, whereas neither risk group was likely to become depressed in the absence of negative events (Genetic Vulnerability × Stress interaction). Similar results were obtained with adolescent twins (Silberg et al., 2001). But what specific genes are involved in this effect?

Exciting recent work by Caspi et al. (2003) has identified one such gene. Caspi et al. (2003) reasoned that the serotonin (5-HT) system provides a highly plausible source of candidate genes to participate in a Genetic Vulnerability × Stress interaction culminating in depression. In particular, the selective serotonin reuptake inhibitor (SSRI) drugs, highly effective in treating depression, target the serotonin transporter (5-HTT), which is involved in the reuptake of serotonin at brain synapses. The serotonin transporter gene has a well-studied functional polymorphism in the promotor region, commonly designated as 5-HTTLPR. There are two common functionally

different alleles at the 5-HTTLPR site—the short (s) allele and the long (l) allele. The s allele is associated with reduced transcription and functional capacity of the serotonin transporter compared to the l allele (Lesch et al., 1996).

Although evidence for a direct association between the s allele at the 5-HTTLPR site and depression is inconclusive (Lesch, 2003), work with nonhuman primates (e.g., Bennett et al., 2002) and neuroimaging studies with humans (Hairi et al., 2002) suggest that the 5-HTTLPR genotype moderates the serotonergic response to stress and thus may interact with stressors to predict depression in humans. Accordingly, Caspi et al. (2003) reported a Gene × Environment interaction in which individuals with the s allele at the 5-HTTLPR site ("ss" and "sl") exhibited more depressive symptoms, diagnosable depression, and suicidality following stressful life events (including childhood maltreatment) than individuals homozygous for the long allele ("ll"); see top left portion of figure 13-1). The Caspi study has generated great excitement. To date, there are four published replications (Eley et al., 2004; Grabe et al., 2004; Kaufman et al., 2004; Kendler et al., 2005) and one failure to replicate (Gillespie et al., 2005).

The replications of Caspi et al. (2003) suggest important future directions for research on the 5-HTTLPR genotype in depression. First, whereas Caspi et al. (2003) found that major stressors interacted with the 5-HTTLPR genotype, Kendler et al. (2005) found this effect for *mild* stressors. Thus, future studies need to employ highly accurate measures of the full range of negative events, from mild to severe, especially because events triggering depression in adolescents often are mild to moderate (e.g., romantic breakup; Joyner & Udry, 2000). Second, it is important to determine whether the 5-HTTLPR Genotype × Stress interaction *specifically* predicts depression. Similar to our findings with cognitive vulnerability (Alloy et al., 2006; Hankin et al., 2004), Kendler et al. (2005) found that the 5-HTTLPR Genotype × Stress interaction showed specificity and predicted depression, but not anxiety. Third, Kaufman et al.'s (2004) finding that social support further moderates the 5-HTTLPR Genotype × Stress interaction (5-HTTLPR Genotype × Stress × Social Support interaction) reinforces the importance of examining whether maladaptive inferential and emotional feedback from parents and/or peers synergistically amplifies the effects of negative events on genetically (or cognitively) vulnerable adolescents (5-HTTLPR Genotype/Cognitive Vulnerability × Stress × Feedback interaction). Finally, in two of the replications (Eley et al., 2004; Grabe et al., 2004), the 5-HTTLPR Genotype × Stress interaction was obtained for females only. The moderation of this interaction by gender underscores the importance of examining whether the emergence of an operative 5-HTTLPR Genotype × Stress interaction mediates females' surge of depression in adolescence.

Of great importance, three studies have found ethnicity differences in 5-HTTLPR alleles and/or genotypes (Gelernter et al., 1997, 1998; Kaufman et al., 2004). African Americans are more likely to exhibit the "l" allele (or l/l genotype) and less likely to exhibit the "s" allele than Caucasians. These consistent findings

suggest that, holding all other factors constant, African Americans should be less prone than Caucasians to develop depression when confronted with negative events. Consistent with this prediction, in the recently completed National Comorbidity Survey Replication, with 9,282 English-speaking respondents aged 18 and older, non-Hispanic blacks showed a lower risk of mood disorders than non-Hispanic whites (Kessler et al., 2005). An important question for future research is whether African American girls' lower likelihood of showing a surge in depression during adolescence is partly due to a "protective" 5-HTTLPR genotype (and/or lesser cognitive vulnerability).

Placing the Genetic Vulnerability-Stress Model in a Neurodevelopment Context

To both understand and prevent depression, it is critical to know when the 5-HTTLPR Genotype × Stress interaction (and Cognitive Vulnerability × Stress interaction) becomes operative developmentally and contributes to clinically significant depression. Although some of the replications of Caspi et al. (2003) study have included adolescents (e.g., Eley et al., 2004; Kaufman et al., 2004), none has examined whether the 5-HTTLPR Genotype × Stress interaction shows developmental changes. An important next step will be to employ a prospective design to construct a developmental timeline of the potency of the 5-HTTLPR Genotype × Stress interaction in predicting depression.

Work on developmental genetics and neurodevelopment suggests that this interaction may come "online" as children transition from late childhood to adolescence, age 10–15 years (see top right portion of figure 13-1). Investigators (e.g., Walker et al., 2004) have drawn on recent advances showing that hormones affect gene expression (e.g., Kawata, 1995) to suggest that pubertal hormonal changes may trigger the expression of genetic vulnerabilities for various disorders, including depression. This is consistent with the finding that the heritability estimate for depression increases significantly in adolescence, rising dramatically after puberty (Silberg et al., 1999). The expression of such genetic vulnerabilities (along with increased levels of cognitive vulnerability) may contribute to adolescents' increased "stress sensitivity" (Spear, 2000; Walker et al., 2004). Thus, the 5-HTTLPR Genotype × Stress interaction may exert an increasingly stronger effect on depression over the transition to adolescence and thus may contribute to the surge in depression, especially among females.

Further contributing to adolescents' stress sensitivity may be a pubertal increase in activity of the hypothalamic-pituitary-adrenal (HPA) axis (Walker et al., 2004), an important biological stress-response system. Thus, the combination of increased levels and operation of cognitive vulnerability, increased expression of genetic vulnerabilities, and increased activity of the HPA axis may make adolescents especially vulnerable to the effects of stress just at the time when they are experiencing a developmental rise in the number of stressors. This scenario suggests a

"two-hit" model of the rise in depression during adolescence because both vulnerability to stress and stress itself are increasing.

Integrating the Cognitive Vulnerability-Stress and Genetic Vulnerability-Stress Models

To date, the cognitive and genetic approaches to depression have proceeded in parallel with little "cross-talk." Given the major success of each, it is time to integrate them. Providing a foundation for such an integration, two independent studies have shown that cognitive vulnerability has a genetic component (Lau, Rijsdijk, & Eley, in press; Schulman, Keith, & Seligman, 1993). In particular, higher concordances for cognitive vulnerability among monozygotic than dizygotic twins have been reported (Schulman et al., 1993). Similarly, in a study of over 1,300 adolescent twin and sibling pairs, model-fitting techniques revealed a genetic influence on cognitive vulnerability (attributional style; Lau et al., in press). For a number of reasons, it is highly plausible that the 5-HTTLPR genotype, in particular, is related to cognitive vulnerability. At a general level, cognitive vulnerability and 5-HTTLPR genotype vulnerability share some critical similarities. Both appear related to ethnicity, with African Americans less vulnerable than Caucasians. Moreover, both participate in vulnerability-stress interactions that moderate the effects of stress on the development of depression. At a more specific level, exciting recent work (Meyer et al., 2003, 2004) suggests that serotonin modulates dysfunctional attitudes, one type of cognitive vulnerability for depression. Finally, a link has been found between the 5-HTTLPR genotype and negative emotionality/neuroticism (e.g., Lesch et al., 1996, but for an exception, see Gelertner et al., 1998), which, in turn, interacts with negative life events to predict the development of cognitive vulnerability among children making the transition to adolescence (Mezulis, Hyde, & Abramson, in press).

Thus, a growing body of work suggests a link between cognitive vulnerability and the 5-HTTLPR genotype. Building on these findings, it will be important to determine whether cognitive vulnerability is related to the 5-HTTLPR genotype and whether cognitive vulnerability mediates, at least in part, the effects of this genotype on depression in response to the rise in adolescent stressors. The time is ripe to test such an integrated cognitive/genetic vulnerability-stress model as an explanation for the adolescent surge and emergence of gender differences in depression in a prospective study of children making the transition to adolescence.

Implications for Prevention

Insofar as depression has been ranked as the "single most burdensome disease" (Murray & Lopez, 1996) due to its unique combination of high lifetime prevalence, early age of onset, high chronicity, and great role impairment (Kessler, 2000), it

is critical to develop effective programs to prevent this disorder. Moreover, as this chapter has shown, the period from late childhood to early adolescence may represent a "window of opportunity" to implement programs to prevent the adolescent surge in depression, especially among females. Knowledge of mechanisms underlying the adolescent surge in depression would suggest interventions for short-circuiting it and the recurrences and great impairment it portends for young adulthood (e.g., Lewinsohn et al., 1999, 2003; Weissman et al., 1999).

A recent meta-analysis (Horowitz & Garber, 2005) of prevention programs for depression in children and adolescents indicates small to moderate effect sizes, suggesting that such prevention programs may be premature and insufficiently informed by knowledge of risk factors and mechanisms underlying the development of these disorders in adolescence. In addition, this meta-analysis showed that selective prevention programs targeting high-risk individuals are more effective than universal programs.

Our application of the cognitive vulnerability-stress model to the adolescent surge in depression suggests that identifying youth with negative cognitive styles and teaching them more adaptive ways to interpret negative events may be an especially potent way to short-circuit the rise in depression during adolescence. Given adolescents' increased brain maturation and cognitive competence (e.g., selective attention), negative cognitive styles may become especially depressogenic during adolescence because they are likely to lead to ever-escalating rumination in the face of negative events. Thus, it also may be helpful to teach cognitively vulnerable youth how to exit from a ruminative cycle (e.g., better problem solving, distraction from the problem, decrease in the importance of the problem). Further, recall that individuals with negative cognitive styles not only interpret negative events more pessimistically than do their nonvulnerable counterparts, but they also experience more negative events (Safford et al., 2006). Accordingly, cognitively vulnerable youth also should benefit from interventions designed to help them solve problems and increase their interpersonal skills in addition to decreasing the negativity of their event interpretations (see Jaycox, Reivich, Gillham, & Seligman, 1994, for a prevention program with a social problem-solving component). Indeed, given the developmental rise in negative life events after age 13 for both boys and girls (e.g., Garber et al., 2002), but especially for adolescent girls (Ge et al., 1994) in the interpersonal domain (Hankin & Abramson, 2001), teaching social problem solving skills should be an important component of preventive interventions with cognitively vulnerable youth.

An important issue in designing future programs to prevent the surge in depression in adolescence is the optimal age to intervene (e.g., high school vs. pre- or middle adolescence). In addition to cognitive vulnerability, 5-HTTLPR genotype may be an important identifier of youth most in need of interventions to prevent depression. An interesting possibility is that 5-HTTLPR genotype identifies individuals with cognitive vulnerability. Further, ethnicity and gender should

be considered in designing optimal prevention programs. Finally, how broad should prevention programs for depression be? Will such programs also prevent the surge in adolescence of disorders comorbid with depression such as bulimia?

In contemplating interventions to prevent depression, we are reminded of a quote by Jonas Salk. On the 30th anniversary of the first Salk vaccine trials, Martin Seligman asked Jonas Salk what he would be doing today if he were a young scientist. Without hesitation, Salk replied, "I'd still do immunization, but I'd do it psychologically rather than biologically" (quoted in Buchanan & Seligman, 1995, p. 250). This chapter suggests that one way to immunize against depression in youth is to modify negative cognitive styles.

References

Abramson, L. Y., Alloy, L. B., Hankin, B. L., Haeffel, G. J., MacCoon, D. G., & Gibb, B. E. (2002). Cognitive vulnerability-stress models of depression in a self-regulatory and psychobiological context. In I. H. Gotlib & C. L. Hammen (Eds.), *Handbook of depression* (3rd ed., pp. 268–294). New York: Guilford.

Abramson, L. Y., Alloy, L. B., Hogan, M. E., Whitehouse, W. G., Cornette, M., Akhavan, S., & Chiara, A. (1998). Suicidality and cognitive vulnerability to depression among college students: A prospective study. *Journal of Adolescence, 21*, 473–487.

Abramson, L. Y., Alloy, L. B., Lever, N. T., Chiara, A., Whitehouse, W. G., & Hogan, M. E. (2006). *The Temple–Wisconsin Cognitive Vulnerability to Depression (CVD) Project: Psychopathology in the parents of individuals at high and low cognitive risk for depression.* Manuscript in preparation, University of Wisconsin–Madison.

Abramson, L. Y., Metalsky, G. I., & Alloy, L. B. (1989). Hopelessness depression: A theory-based subtype of depression. *Psychological Review, 96*, 358–372.

Alloy, L. B., & Abramson, L. Y. (1999). The Temple-Wisconsin Cognitive Vulnerability to Depression (CVD) Project: Conceptual background, design and methods. *Journal of Cognitive Psychotherapy: An International Quarterly, 13*, 227–262.

Alloy, L. B., Abramson, L. Y., Gibb, B. E., Crossfield, A. G., Pieracci, A. M., Spasojevic, J., & Steinberg, J. (2004). Developmental antecedents of cognitive vulnerability to depression: Review of findings from the Cognitive Vulnerability to Depression (CVD) Project. *Journal of Cognitive Psychotherapy: An International Quarterly, 18*, 115–133.

Alloy, L. B., Abramson, L. Y., Hogan, M. E., Whitehouse, W. G., Rose, D. T., Robinson, M. S., Kim, R. S., & Lapkin, J. B. (2000). The Temple-Wisconsin Cognitive Vulnerability to Depression (CVD) Project: Lifetime history of Axis I psychopathology in individuals at high and low cognitive risk for depression. *Journal of Abnormal Psychology, 109*, 403–418.

Alloy, L. B., Abramson, L. Y., Murray, L. A., Whitehouse, W. G., & Hogan, M. E. (1997). Self-referent information processing in individuals at high and low cognitive risk for depression. *Cognition and Emotion, 11*, 539–568.

Alloy, L. B., Abramson, L. Y., Tashman, N. A., Berrebbi, D. L., Hogan, M. E., Whitehouse, W. G., Crossfield, A. G., & Morocco, A. (2001). Developmental origins of cognitive vulnerability to depression: Parenting, cognitive, and inferential feedback styles of the parents of individuals at high and low cognitive risk for depression. *Cognitive Therapy and Research, 25*, 397–423.

Alloy, L. B., Abramson, L. Y., Whitehouse, W. G., Hogan, M. E., Panzarella, C., & Rose, D. T. (2006). Prospective incidence of first onsets and recurrences of depression in individuals at high and low cognitive risk for depression. *Journal of Abnormal Psychology, 115,* 145–156.

Alloy, L. B., Abramson, L. Y., Whitehouse, W. G., Hogan, M. E., Tashman, N. A., Steinberg, D. L., Rose, D. T., & Donovan, P. (1999). Depressogenic cognitive styles: Predictive validity, information processing and personality characteristics, and developmental origins. *Behaviour Research and Therapy, 37,* 503–531.

Alloy, L. B., & Clements, C. M. (1998). Hopelessness theory of depression: Tests of the symptom component. *Cognitive Therapy and Research, 22,* 303–335.

Alloy, L. B., Just, N., & Panzarella, C. (1997). Attributional style, daily life events, and hopelessness depression: Subtype validation by prospective variability and specificity of symptoms. *Cognitive Therapy and Research, 21,* 321–344.

Alloy, L. B., Lipman, A., & Abramson, L. Y. (1992). Attributional style as a vulnerability factor for depression: Validation by past history of mood disorders. *Cognitive Therapy and Research, 16,* 391–407.

Alloy, L. B., Zhu, L., & Abramson, L. Y. (2003). Cognitive vulnerability to depression: Implications for adolescent risk behavior in general. In D. Romer (Ed.), *Reducing adolescent risk: Toward and integrated approach* (pp. 171–182). Thousand Oaks, CA: Sage.

Angold, A., Costello, E. J., & Worthman, C. M. (1998). Puberty and depression: The roles of age, pubertal status and pubertal timing. *Psychological Medicine, 28,* 51–61.

Beck, A. T. (1987). Cognitive models of depression. *Journal of Cognitive Psychotherapy: An International Quarterly, 1,* 5–37.

Bennett, A. J., Lesch, K. P., Heils, A., Long, J. C., Lorenz, J. G., Shoaf, S. E., et al. (2002). Early experience and serotonin transporter gene variation interact to influence primate CNS function. *Molecular Psychiatry, 7,* 118–122.

Botvinik, M., Braver, T. S., Barch, S. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review, 108,* 624–652.

Braver, T. S., Barch, D. M., Gray, J. R., Molfese, D. L., & Snyder, A. (2001). Anterior cingulated cortex and response conflict: Effects of frequency, inhibition and errors. *Cerebral Cortex, 11,* 825–836.

Broderick, P. C. (1998). Early adolescent gender differences in the use of ruminative and distracting coping strategies. *Journal of Early Adolescence, 18,* 173–191.

Brown, T. T., Lugar, H. M., Coalson, R. S., Miezin, F. M., Persen, S. E., & Schlaggar, B. L. (2005). Developmental changes in human cerebral functional organization for word generation. *Cerebral Cortex, 15,* 275–290.

Buchanan, G. M., & Seligman, M. E. P. (1995). Afterword: The future of the field. In G. M. Buchanan & M. E. P. Seligman (Eds.), *Explanatory style* (pp. 247–252). Hillsdale, NJ: Erlbaum.

Burke, K. C., Burke, J. D., Regier, D. A., & Rae, D. S. (1990). Age at onset of selected mental disorders in five community populations. *Archives of General Psychiatry, 47,* 511–518.

Carver, C. S., & Scheier, M. F. (1998). *On the self-regulation of behavior.* Cambridge: Cambridge University Press.

Casey, B. J., Galvan, A., & Hare, T. A. (2005a). Changes in cerebral functional organization during cognitive development. *Current Opinion in Neurobiology, 15,* 239–244.

Casey, B. J., Giedd, J. N., & Thomas, K. M. (2000). Structural and functional brain development and its relation to cognitive development. *Biological Psychology, 54,* 241–257.

Casey, B. J., Tottenham, N., Liston, C., & Durston, S. (2005b). Imaging the developing brain: What have we learned about cognitive development? *Trends in Cognitive Sciences, 9*, 104–110.

Casey, B. J., Trainor, R., Giedd, J., Vauss, Y., Vaituzis, C. K., Hamburger, S., Kozuch, P., & Rapoport, J. L. (1997). The role of the anterior cingulated in automatic and controlled processes: A developmental neuroanatomical study. *Developmental Psychobiology, 30*, 61–69.

Casey, B. J., Trainor, R. J., Orendi, J. L., Schubert, A. B., Nystrom, L. E., Cohen, J. D., Noll, D. C., Giedd, J., Castellanos, X., Haxby, J., et al. (1997). A pediatric functional MRI study of prefrontal activation during performance of a Go-No-Go task. *Journal of Cognitive Neuroscience, 9*, 835–847.

Casper, R. C., & Offer, D. (1990). Weight and dieting concerns in adolescents, fashion or symptom? *Pediatrics, 86*, 384–390.

Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., McClay, J., Mill, J., Martin, J.,Braithwaite, A., & Poulton, R. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5–HTT gene. *Science, 301*, 386–389.

Cicchetti, D., & Rogosch, F. A. (2002). A developmental psychopathology perspective on adolescence. *Journal of Consulting and Clinical Psychology, 70*, 6–20.

Cohen, J. D., & Servan-Schreiber, D. (1992). Context, cortex, and dopamine: A connectionist approach to behavior and biology in schizophrenia. *Psychological Review, 99*, 45–77.

Cohen, S., & Wills, T. A. (1985). Stress, social support, and the buffering hypothesis. *Psychological Bulletin, 98*, 310–357.

Compas, B. E., Connor, J. K., & Hinden, B. R. (1998). New perspectives on depression during adolescence. In R. Jessor (Ed.), *New perspectives on adolescent risk behavior* (pp. 319–364). Cambridge, UK: Cambridge University Press.

Compas, B. E., Davis, G. E., & Forsyth, C. J. (1985). Characteristics of life events during adolescence. *American Journal of Community Psychology, 13*, 677–691.

Crick, N. R., & Grotpeter, J. K. (1996). Children's treatment by peers: Victims of relational and overt aggression. *Development and Psychopathology, 8*, 367–380.

Crossfield, A. G., Alloy, L. B., Abramson, L. Y., & Gibb, B .E. (2002). The development of depressogenic cognitive styles: The role of negative childhood life events and parental inferential feedback. *Journal of Cognitive Psychotherapy: An International Quarterly, 16*, 487–502.

Cyranowski, J. M., Frank, E., Young, E., & Shear, K. (2000). Adolescent onset of the gender difference in lifetime rates of major depression: A theoretical model. *Archives of General Psychiatry, 57*, 21–27.

De Bellis, M. D., Keshavan, M. S., Beers, S. R., Hall, J., Frustaci, K., Masalehdan, A., Noll, J., & Boring, A. M. (2001). Sex differences in brain maturation during childhood and adolescence. *Cerebral Cortex,11*, 552–557.

Dobkin, R., Panzarella, C., Nesbitt, J., & Alloy, L. B. (2004). Adaptive inferential feedback depressogenic inferences, and depressed mood: A laboratory study of the expanded hopelessness theory of depression. *Cognitive Therapy and Research, 28*, 487–509.

Durston, S., Davison, M. C., Tottenham, N., Galvan, A., Spicer, J. A., Fossella, F., & Casey, B. J. (2005). A shift from diffuse to focal cortical activity with development. *Journal of Cognitive Neuroscience, 17*, S57–S58.

Eley, T. C., Sugden, K., Corsico, A., Gregory, A. M., Sham, P., Mcguffin, P., Plomin, R., & Craig, I. W. (2004). Gene-environment interaction analysis of serotonin system markers with adolescent depression. *Molecular Psychiatry, 9*, 908–915.

Floyd, T. D., Alloy, L. B., Smith, J., Neeren, A., & Thorell, G. (2006). *Puberty, ethnicity, and gender differences in depression: Role of cognitive vulnerability and the thin female ideal.* Manuscript in preparation, Temple University.

Garber, J., Keiley, M. K., & Martin, N. C. (2002). Developmental trajectories of adolescents' depressive symptoms: Predictors of change. *Journal of Consulting and Clinical Psychology, 70,* 79–95.

Ge, X., Lorenz, F. O., Conger, R. D., Elder, G. H., & Simons, R. L. (1994). Trajectories of stressful life events and depressive symptoms during adolescence. *Developmental Psychology, 30,* 467–483.

Gelernter, J., Kranzler, H., Coccaro, E. F., Siever, L. J., & New, A. S. (1998). Serotonin transporter protein gene polymorphism and personality measures in African American and European American subjects. *American Journal of Psychiatry, 155,* 1332–1338.

Gelernter, J., Kranzler, H., & Cubells, J. F. (1997). Serotonin transporter protein (SLC6A4) allele and haplotype frequencies and linkage disequilibria in African- and European-American and Japanese populations and in alcohol-dependent subjects. *Human Genetics, 101,* 243–246.

Gibb, B. E., Abramson, L. Y., & Alloy, L. B. (2004). Emotional maltreatment from parents, peer victimization, and cognitive vulnerability to depression. *Cognitive Therapy and Research, 28,* 1–21.

Gibb, B. E., Alloy, L. B., Abramson, L. Y., Rose, D. T., Whitehouse, W. G., Donovan, P., Hogan, M. E., Cronholm, J., & Tierney, S. (2001a). History of childhood maltreatment, negative cognitive styles, and episodes of depression in adulthood. *Cognitive Therapy and Research, 25,* 425–446.

Gibb, B. E., Alloy, L. B., Abramson, L. Y., Rose, D. T., Whitehouse, W. G., & Hogan, M. E. (2001b). Childhood maltreatment and college students' current suicidal ideation: A test of the hopelessness theory. *Suicide and Life-Threatening Behavior, 31,* 405–415.

Gibb, B. E., Alloy, L. B., Walshaw, P. D., Comer, J. S., Chang, G. H., & Villari, A. G. (2006). Predictors of negative attributional style change in children. *Journal of Abnormal Child Psychology 34,* 425–439.

Giedd, J. N. (2004). Structural magnetic resonance imaging of the adolescent brain. *Annals of the NY Academy of Science, 1021,* 77–85.

Giedd, J. N., Blumenthal, J., Jeffries, N. O., Castellanos, F. X., Lui, H., Zijdenbos, A., Paus, T., Evans, A. C., & Rapoport, J. L. (1999). Brain development during childhood and adolescence: A longitudinal MRI study. *Nature Neuroscience, 2,* 861–863.

Giedd, J. N., Snell, J. W., Lange, N., Rajapakse, J. C., Casey, B. J., Kozuch, P. L., Viatuzis, A. C., Vauss, Y. C., Hamburger, S. D., Kaysen, D., & Rapoport, J. L. (1996). Quantitative magnetic resonance imaging of human brain development: Ages 4–18. *Cerebral Cortex, 6,* 551–560.

Gillespie, N. A., Whitfield, J. B., Williams, D., Heath, A. C., & Martin, N. G. (2005). The relationship between stressful life events, the serotonin transporter (5-HTTLPR) genotype, and major depression. *Psychological Medicine, 35,* 101–111.

Gogtay, N., Giedd, J. N., Lusk, L., Hayashi, K. M., Greenstein, D., Vaituzis, A. C., Nugent, T. F., Herman, D. H., Clasen, L. S., Toga, A. W., Rapoport, J. L., & Thompson, P. M. (2004). Dynamic mapping of human cortical development during childhood through early adulthood. *Proceedings of the National Academy of Science, 101,* 8174–8179.

Gotlib, I. H., & Hammen, C. L. (2002). Introduction. In I. H. Gotlib & C. L. Hammen (Eds.), *Handbook of depression.* (3rd ed., pp. 1–20). New York: Guilford.

Grabe, H. J., Lange, M., Wolff, B., Volzke, H., Lucht, M., Freyberger, H. J., John, U., & Cascorbi, I. (2004). Mental and physical distress is modulated by a polymorphism in

the 5-HT transporter gene interacting with social stressors and chronic disease burden. *Molecular Psychiatry, 10*, 220–224.

Greenberg, P., Kessler, R., Nells, T., Finkelstein, S., & Berndt, E. R. (1996). Depression in the workplace: An economic perspective. In J. P. Feighner & W. F. Boyer (Eds.), *Selective serotonin reuptake inhibitors: Advances in basic research and clinical practice* (pp. 327–363). New York: Wiley.

Halpern, C., Udry, J. R., Campbell, B., & Suchindran, C. (1999). Effects of body fat on weight concerns, dating, and sexual activity: A longitudinal analysis of black and white adolescent girls. *Developmental Psychology, 35*, 721–736.

Hankin, B. L., & Abramson, L. Y. (2001). Development of gender differences in depression: An elaborated cognitive vulnerability-transactional stress theory. *Psychological Bulletin, 127*, 773–796.

Hankin, B. L., & Abramson, L. Y. (2002). Measuring cognitive vulnerability to depression in adolescence: Reliability, validity, and gender differences. *Journal of Child and Adolescent Clinical Psychology, 31*, 491–504.

Hankin, B. L., Abramson, L. Y., Miller, N., & Haeffel, G. J. (2004). Cognitive vulnerability-stress theories of depression: Examining affective specificity in the prediction of depression versus anxiety in three prospective studies. *Cognitive Therapy and Research, 28*, 309–345.

Hankin, B. L., Abramson, L. Y., Moffitt, T. E., Silva, P. A., McGee, R., & Angell, K. E. (1998). Development of depression from preadolescence to young adulthood: Emerging gender differences in a 10-year longitudinal study. *Journal of Abnormal Psychology, 107*, 128–140.

Hankin, B. L., Abramson, L. Y., & Siler, M. (2001). A prospective test of the hopelessness theory of depression in adolescence. *Cognitive Therapy and Research*, 25, 607–632.

Hariri, A. R., Mattay, V. S., Tessitore, A., Kolachana, B., Fera, F., Goldman, D., et al. (2002). Serotonin transporter genetic variation and the response of the human amygdala. *Science, 297*, 400–403.

Harris, J. R. (1995). Where is the child's environment? A group socialization theory of development. *Psychological Review, 102*, 458–489.

Hayward, C., Gotlib, I. H., Schradley, P. K., & Litt, I. F. (1999). Ethnic differences in the association between pubertal status and symptoms of depression in adolescent girls. *Journal of Adolescent Health, 25*, 143–149.

Holmbeck, G. N., & Kendall, P. C. (2002). Introduction to the special section on clinical adolescent psychology: Developmental psychopathology and treatment. *Journal of Consulting and Clinical Psychology, 70*, 3–5.

Horowitz, J. S., & Garber, J. (2005). *The prevention of depression in children and adolescents: A meta-analytic review*. Manuscript under editorial review.

Jaycox, L. H., Reivich, K. J., Gillham, J., & Seligman, M. E. P. (1994). Prevention of depressive symptoms in school children. *Behaviour Research and Therapy, 32*, 801–816.

Joyner, K., & Udry, J. R. (2000). You don't bring me anything but down: Adolescent romance and depression. *Journal of Health and Social Behavior, 41*, 369–391.

Judd, L. L. (1997). The clinical course of unipolar major depressive disorders. *Archives of General Psychiatry, 54*, 989–991.

Just, N., & Alloy, L. B. (1997). The response styles theory of depression: Tests and an extension of the theory. *Journal of Abnormal Psychology, 106*, 221–229.

Kaufman, J., Yang, B. Z., Douglas-Palumberi, H., Houshyar, S., Lipschitz, D., Krystal, J. H., & Gelernter, J. (2004). Social supports and serotonin transporter gene moderate

depression in maltreated children. *Proceedings of the National Academy of Science, 101,* 17316–17321.

Kawata, M. (1995). Roles of steroid hormones and their receptors in structural organization in the nervous system. *Neuroscience Research, 24,* 1–46.

Kazdin, A. E., French, N. H., Unis, A. S., Esveldt-Dawson, K., & Sherick, R. B. (1983). Hopelessness, depression, and suicidal intent among psychiatrically disturbed inpatient children. *Journal of Consulting and Clinical Psychology, 51,* 504–510.

Keating, D. P. (1990). Adolescent thinking. In S. Feldman & G. Elliott (Eds.), *At the threshold: The developing adolescent* (pp. 54–89). Cambridge, MA: Harvard University Press.

Keating, D. P. (2004). Cognitive and brain development. In R. J. Lerner & L. D. Steinberg (Eds.), *Handbook of adolescent psychology* (2nd ed.). New York: Wiley.

Kendler, K. S., Kessler, R. C., Walters, E. E., MacLean, C. J., Sham, P. C., Neale, M. C., Health, A. C., & Eaves, L. J. (1995). Stressful life events, genetic liability, and onset of an episode of major depression in women. *American Journal of Psychiatry, 152,* 833–842.

Kendler, K. S., Kuhn, J. W., Vittum, J., Prescott, C. A., & Riley, B. (2005). The interaction of stressful life events and a serotonin transporter polymorphism in the prediction of episodes of major depression: A replication. *Archives of General Psychiatry, 62,* 529–535.

Kendler, K. S., Neale, M. C., Kessler, R. C., Heath, A. C., & Eaves, L. J. (1992). A population-based study of major depression in women: The impact of varying definitions of illness. *Archives of General Psychiatry, 49,* 257–266.

Kessler, R. C. (2000). Burden of depression. In S. Kasper & A. Carlsson (Eds.), *Selective serotonin reuptake inhibitors 1990–2000: A decade of developments.* Copenhagen, Denmark: H. Lundbeck A/S.

Kessler, R. C. (2002). Epidemiology of depression. In I. H. Gotlib & C. L. Hammen (Eds.), *Handbook of depression.* (3rd ed., pp. 23–42). New York: Guilford.

Kessler, R. C., Avenevoli, S., & Merikangas, K. R. (2001). Mood disorders in children and adolescents: An epidemiologic perspective. *Biological Psychiatry, 49,* 1002–1014.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey replication. *Archives of General Psychiatry, 62,* 593–602.

Killgore, W. D. S., & Yurgelun-Todd, D. A. (2004). Sex-related developmental differences in the lateralized activation of the prefrontal cortex and amygdala during perception of facial affect. *Perceptual and Motor Skills, 99,* 371–391.

Kimberg, D. Y., & Farah, M. J. (1993). A unified account of cognitive impairments following frontal lobe damage: The role of working memory in complex, organized behavior. *112,* 411–428.

Lau, J. Y. F., Rijsdijk, F., & Eley, T. C. (in press). I think, therefore I am: A twin study of attributional style in adolescents. *Journal of Clinical Psychology and Psychiatry.*

Lesch, K. P. (2003). Neuroticism and serotonin: A developmental genetic perspective. In R. Plomin, J. C. DeFries, I. W. Craig, & P. McGuffin (Eds.), *Behavioral genetics in the postgenomics era* (pp. 389–424). Washington, DC: American Psychological Association.

Lesch, K. P., Bengel, D., Heils, A., Sabol, S. Z., Greenberg, B. D., Petri, S., Benjamin, J., Muller, C. R., Hamer, D. H., & Murphy, D. L. (1996). Association of anxiety-related traits with a polymorphism in the serotonin transporter gene regulatory region. *Science, 274,* 1527–1531.

Lewinsohn, P. M., Rohde, P., Klein, D. N., & Seeley, J. R. (1999). Natural course of ado-

lescent major depressive disorder: I. Continuity into young adulthood. *Journal of the American Academy of Child and Adolescent Psychiatry, 38*, 56–63.

Lewinsohn, P. M., Rohde, P., Seeley, J. R., Klein, D. N., & Gotlib, I. H. (2003). Psychosocial functioning of young adults who have experienced and recovered from major depressive disorder during adolescence. *Journal of Abnormal Psychology, 112*, 353–362.

Liston, C., Watts, R., Tottenham, N., Davidson, M. C., Niogi, S., Ulug, A., & Casey, B. J. (2003). Developmental differences in diffusion measures of cortical fiber tracts. *Journal of Cognitive Neuroscience, 15*, S57–S58.

Liu, X., & Kaplan, H. B. (1999). Explaining gender differences in symptoms of subjective distress in young adolescents. *Stress Medicine, 15*, 41–51.

MacCoon, D. G., Abramson, L. Y., Mezulis, A. H., Hankin, B. L., & Alloy, L. B. (2006). *The attention mediated hopelessness (AMH) theory: The role of attention in connecting cognitive vulnerability to rumination in depression.* Manuscript under editorial review.

McClure, E. B., Monk, C. S., Nelson, E. E., Zarahn, E., Leibenluft, E., Bilder, R. M., Charney, D. S., Ernst, M., & Pine, D. S. (2004). A developmental examination of gender differences in brain engagement during evaluation of threat. *Biological Psychiatry, 55*, 1047–1055.

Metalsky, G. I., Joiner, T. E., Hardin, T. S., & Abramson, L. Y. (1993). Depressive reactions to failure in a naturalistic setting: A test of the hopelessness and self-esteem theories of depression. *Journal of Abnormal Psychology, 102*, 101–109.

Meyer, J. H., Houle, S., Sagrati, S., Carella, A., Hussey, D. F., Ginovart, N., Goulding, V., Kennedy, J., & Wilson, A. A. (2004). Brain serotonin transporter binding potential measured with carbon 11–labeled DASB positron emission tomography: Effects of major depressive episodes and severity of dysfunctional attitudes. *Archives of General Psychiatry, 61*, 1271–1279.

Meyer, J. H., McMain, S., Kennedy, S., Korman, L., Brown, G., DaSilva, J., Wilson, A., Blak, T., Eynan-Harvey, R., Goulding, V., Houle, S., & Links, P. (2003). Dysfunctional attitudes and serotonin$_2$ receptors during depression and self-harm. *American Journal of Psychiatry, 160*, 90–99.

Mezulis, A. H., Abramson, L. Y., & Hyde, J. S. (2002). Domain specificity of gender differences in rumination. *Journal of Cognitive Psychotherapy: An International Quarterly, 16*, 421–434.

Mezulis, A. H., Abramson, L. Y., Hyde, J. S., & Hankin, B. L. (2004). Is there a universal positivity bias in attributions: A meta-analytic review of individual, developmental, and cultural differences in the self-serving attributional bias. *Psychological Bulletin, 130*, 711–747.

Mezulis, A. H., Hyde, J. S., & Abramson, L. Y. (in press). The developmental origins of cognitive vulnerability to depression: Temperament, parenting, and negative life events as contributors to negative cognitive style. *Developmental Psychology.*

Murray, C. J. L., & Lopez, A. D. (Eds.). (1996). *The global burden of disease: A comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020.* Cambridge, MA: Harvard University Press.

Nagy, Z., Westerberg, H., & Klingberg, T. (2004). Maturation of white matter is associated with the development of cognitive functions during childhood. *Journal of Cognitive Neuroscience, 16*, 1227–1233.

Nolen-Hoeksema, A. (1990). *Sex differences in depression.* Stanford, CA: Stanford University Press.

Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of the depressive episode. *Journal of Abnormal Psychology, 100*, 569–582.

Nolen-Hoeksema, S. (2000). The role of rumination in depressive disorders and mixed anxiety/depressive symptoms. *Journal of Abnormal Psychology, 109*, 504–511.

Nolen-Hoeksema, S., & Girgus, J. S. (1994). The emergence of gender differences in depression during adolescence. *Psychological Bulletin, 115*, 424–443.

Nolen-Hoeksema, S., Girgus, J. S., & Seligman, M. E. P. (1992). Predictors and consequences of childhood depressive symptoms: A 5-year longitudinal study. Journal of Abnormal Psychology, 101, 405–422.

Nolen-Hoeksema, S., & Jackson, B. (2001). Mediators of the gender difference in rumination. *Psychology of Women Quarterly, 25*, 37–47.

O'Dea, J. A., & Abraham, S. (1999). Onset of disordered eating attitudes and behaviors in early adolescence: Interplay of pubertal status, gender, weight, and age. *Adolescence, 34*, 671–679.

Owen, A. M. (2000). The role of the lateral frontal cortex in mnemonic processing: The contribution of functional neuroimaging. *Experimental Brain Research, 133*, 33–43.

Panzarella, C., Alloy, L. B., & Whitehouse, W. G. (in press). Expanded hopelessness theory of depression: On the mechanisms by which social support protects against depression. *Cognitive Therapy and Research.*

Parker, G. (1983). Parental "affectionless control" as an antecedent to adult depression. *Archives of General Psychiatry, 34*, 138–147.

Parker, S., Nichter, M., Nukovic, N., Sims, C., &Ritenbaugh, C. (1995). Body image and weight concerns among African American and White adolescent females: Differences that make a difference. *Human Organization, 54*, 103–114.

Paus, T., Zijdenbos, A., Worsley, K., Collins, D. L., Blumenthal, J., Giedd, J. N., Rapoport, J. L., & Evans, A. C. (1999). Structural maturation of neural pathways in children and adolescents: In vivo study. *Science, 283*, 1908–1911.

Petersen, A. C., & Taylor, B. (1980). The biological approach to adolescence: Biological change and psychological adaptation. In J. Adelson (Ed.), *Handbook of adolescent psychology*. New York: Wiley.

Pfefferbaum, A., Mathalon, D. H., Sullivan, E. V., Rawles, J. M., Zipursky, R. B., & Lim, K. O. (1994). A quantitative magnetic resonance imaging study of changes in brain morphology from infancy to late adulthood. *Archives of Neurology, 51*, 874–887.

Pyszczynski, T., & Greenberg, J. (1987). Self-regulatory perseveration and the depressive self-focusing style: A self-awareness theory of reactive depression. *Psychological Bulletin, 102*, 122–138.

Rierdan, J., Koff, E., & Stubbs, M. L. (1989). A longitudinal analysis of body image as a predictor of the onset and persistence of adolescent girls' depression. *Journal of Early Adolescence, 9*, 454–466.

Robinson, M. S., & Alloy, L. B. (2003). Negative cognitive styles and stress-reactive rumination interact to predict depression: A prospective study. *Cognitive Therapy and Research, 27*, 275–291.

Robinson, N. S., Garber, J., & Hilsman, R. (1995). Cognitions and stress: Direct and moderating effects on depressive versus externalizing symptoms during the junior high transition. *Journal of Abnormal Psychology, 104*, 453–463.

Rose, D. T., & Abramson, L. Y. (1992). Developmental predictors of depressive cognitive style: Research and theory. In D. Cicchetti & S. Toth (Eds.), *Rochester symposium on developmental psychopathology* (Vol. IV). Rochester, NY: University of Rochester Press.

Roy, K., Mitchell, P., & Wilhelm, K. (2001). Depression and smoking: Examining corre-

lates in a subset of depressed patients. *Australian and New Zealand Journal of Psychiatry, 35*, 329–335.

Rutter, M. (1989). Age as an ambiguous variable in developmental research: Some epidemiological considerations from developmental psychopathology. *International Journal of Behavioral Development, 12*, 1–34.

Safford, S. M., Alloy, L. B., Abramson, L. Y., & Crossfield, A. G. (2006). *Negative cognitive style as a predictor of negative life events in depression-prone individuals: A test of the stress generation hypothesis.* Manuscript under editorial review.

Schaefer, E. S. (1965). Children's reports of parental behavior: An inventory. *Child Development, 36*, 413–424.

Schulman, P., Keith, D., & Seligman, M. E. P. (1993). Is optimism heritable: A study of twins. *Behaviour Research and Therapy, 31*, 569–574.

Siegel, J. M., Aneshensel, C. S., Taub, et al. (1998). Adolescent depressed mood in a multiethnic sample. *Journal of Youth and Adolescence, 27*, 413–427.

Siegel, J. M., Yancey, A. K., Aneshensel, C. S., & Schuler, R. (1999). Body image, perceived pubertal timing, and adolescent mental health. *Journal of Adolescent Health, 25*, 155–165.

Silberg, J. L., Pickles, A., Rutter, M., Hewitt, J., Simonoff, E., Maes, H., Carbonneau, R., Murrelle, L., Foley, D., & Eaves, L. (1999). The influence of genetic factors and life stress on depression among adolescent girls. *Archives of General Psychiatry, 56*, 225–232.

Silberg, J., Rutter, M., Neale, M., & Eaves, L. (2001). Genetic moderation of environmental risk for depression and anxiety in adolescent girls. *British Journal of Psychiatry, 179*, 114–121.

Smith, J. M., Floyd, T. D., Alloy, L. B., Hughes, M., & Neeren, A. M. (2006). *An integrated model of the gender difference in depression: Pubertal development, response style, and body dissatisfaction.* Manuscript under editorial review.

Sowell, E. R., Delis, D., Stiles, J., & Jernigan, T. L. (2001). Improved memory functioning and frontal lobe maturation between childhood and adolescence: A structural MRI study. *Journal of the International Neuropsychology Society, 7*, 312–322.

Sowell, E. R., Peterson, B. S., Thompson, P. M., Welcome, S. E., Henkenius, A. L., & Toga, A. W. (2003). Mapping cortical change across the human life span. *Nature Neuroscience, 6*, 309–315.

Sowell, E. R., Thompson, P. M., Leonard, C. M., Welcome, S. E., Kan, E., & Toga, A. W. (2004). Longitudinal mapping of cortical thickness and brain growth in normal children. *Journal of Neuroscience, 24*, 8223–8231.

Spasojevic, J., & Alloy, L. B. (2001). Rumination as a common mechanism relating depressive risk factors to depression. *Emotion, 1*, 25–37.

Spasojevic, J., & Alloy, L. B. (2002). Who becomes a depressive ruminator?: Developmental antecedents of ruminative response style. *Journal of Cognitive Psychotherapy: An International Quarterly, 16*, 405–419.

Spear, L. P. (2000). The adolescent brain and age-related behavioral manifestations. *Neuroscience Biobehavioral Review, 24*, 417–463.

Steinberg, L. (2002). Clinical adolescent psychology: What it is, and what it needs to be. *Journal of Consulting and Clinical Psychology, 70*, 124–128.

Steinberg, L., Dahl, R., Keating, D., Kupfer, D. J., Masten, A. S., & Pine, D. (2004). The study of developmental psychopathology in adolescence: Integrating affective neuroscience with the study of context. In R. J. Lerner & L. D. Steinberg (Eds.), *Handbook of adolescent psychology* (2nd ed.). New York: Wiley.

Stipek, D., & MacIver, D. (1989). Developmental change in children's assessment of intellectual competence. *Child Development, 60*, 521–538.

Sullivan, M. D., LaCroix, A. Z., Russo, J. E., & Walker, E. A. (2001). Depression and self-reported physical health in patients with coronary disease: Mediating and moderating factors. *Psychosomatic Medicine, 63*, 248–256.

Thompson, P. M., Giedd, J. N., Woods, R. P., MacDonald, D., Evans, A. C., & Toga, A. W. (2000). Growth patterns in the developing brain detected by using continuum mechanical tensor maps. *Nature, 404*, 190–193.

Turner, J. E., Jr., & Cole, D. A. (1994). Developmental differences in cognitive diatheses for child depression. *Journal of Abnormal Child Psychology, 22*, 15–32.

Walker, E. F., Sabuwalla, Z., & Huot, R. (2004). Pubertal neuromaturation, stress sensitivity, and psychopathology. *Development and Psychopathology, 16*, 807–824.

Weissman, M. M., & Klerman, G. L. (1977). Sex differences in the epidemiology of depression. *Archives of General psychiatry, 34*, 98–111.

Weissman, M. M., Warner, V., Wickramaratne, P., Moreau, D., & Olfson, M. (1997). Offspring of depressed parents: 10 years later. *Archives of General Psychiatry, 54*, 932–942.

Weissman, M. M., Wolk, S., Goldstein, R. B., et al. (1999). Depressed adolescents grown up. *Journal of the American Medical Association, 281*, 1707–1713.