

# The ABCs of Depression: Integrating Affective, Biological, and Cognitive Models to Explain the Emergence of the Gender Difference in Depression

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In adulthood, twice as many women as men are depressed, a pattern that holds in most nations. In childhood, girls are no more depressed than boys, but more girls than boys are depressed by ages 13 to 15. Although many influences on this emergent gender difference in depression have been proposed, a truly integrated, developmental model is lacking. The authors propose a model that integrates affective (emotional reactivity), biological (genetic vulnerability, pubertal hormones, pubertal timing and development) and cognitive (cognitive style, objectified body consciousness, rumination) factors as vulnerabilities to depression that, in interaction with negative life events, heighten girls' rates of depression beginning in adolescence and account for the gender difference in depression.

*Keywords:* gender, depression, temperament, body image, genetics

The gender difference in depression is among the most robust of findings in psychopathology research. Estimates are that, in adulthood, twice as many women as men are depressed (Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993; Lucht et al., 2003; Piccinelli & Wilkinson, 2000; Weissman et al., 1996; Weissman & Klerman, 1977). Although the exact gender ratio varies slightly from culture to culture, most nations have reported a gender ratio close to 2.0<sup>1</sup> (Angst et al., 2002; Kuehner, 2003). The World Health Organization has estimated that major depression is the leading cause of disease-related disability among women worldwide (Kessler, 2003). Research indicates that, although girls are no more depressed than boys in childhood (Anderson, Williams, McGee, & Silva, 1987; Cohen et al., 1993; Rutter, 1986), more girls than boys are depressed by ages 13 to 15 (Hankin et al., 1998; Kessler et al., 1993; Twenge & Nolen-Hoeksema, 2002; Wade, Cairney, & Pevalin, 2002). In one well-sampled study of 15- to 24-year-olds, males had a lifetime incidence of major depressive disorder of 11%, compared with 21% for females (Kessler et al., 1993). If we are to understand the gender difference in depression in adulthood, we must understand its development in adolescence.

The emergence of the gender difference in depression in adolescence has attracted the attention of medical and psychological researchers. The gender difference in depression has been attributed to a wide variety of factors, including girls' and women's

greater ruminative coping (Nolen-Hoeksema & Girgus, 1994), dependence on relationships or affiliative needs (Cyranowski, Frank, Young, & Shear, 2000), ovarian and adrenal hormonal changes at puberty (Goodyer, Herbert, Tamplin, & Altham, 2000; Halbreich & Kahn, 2001; Steiner, Dunn, & Born, 2003), genetic factors (Jacobson & Rowe, 1999; Kendler, Gardner, Neale, & Prescott, 2001; Kendler, Kessler, Neale, Heath, & Eaves, 1993; Silberg et al., 1999; Zubenko, Hughes, Maher, et al., 2002), body dissatisfaction (Nolen-Hoeksema & Girgus, 1994), greater cognitive vulnerability (Hankin & Abramson, 2001), exposure to negative life events (Kendler et al., 1993; Silberg et al., 1999), experiences of rape and child sexual abuse (Kendler, Gardner, & Prescott, 2002), gender intensification and adherence to traditional gender roles (Aube, Fichman, Saltaris, & Koestner, 2000), and interactions among these factors (Hankin & Abramson, 2001; A. Petersen, Sarigiani, & Kennedy, 1991).

The numerous theories and models of depression that have been proposed previously can be broadly categorized as proposing explanations for the emergence of the gender difference in depression emphasizing affective factors such as temperament or emotion regulation (e.g., Cyranowski et al., 2000; Kendler et al., 1993), biological factors such as genetics or pubertal hormones (e.g.,

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<sup>1</sup> Most the nations for which epidemiological data are available are European or European-derived nations such as the United States and New Zealand. The World Health Organization data also documented the 2:1 ratio in adulthood in Korea and Hong Kong (Piccinelli & Homen, 1997). There is a paucity of research on the causes of gender differences in nations other than European or European-derived ones. Our theory is intended to address these cultures and might not apply to substantially different cultures such as those found in sub-Saharan Africa or Arab nations. A few exceptions have also been documented, including Old Order Amish and Orthodox Jews (Angst et al., 2002; Piccinelli & Wilkinson, 2000). From the point of view of ABC theory, these exceptions are interesting because they are religious groups with norms that sharply reduce the sexual objectification of girls in adolescence in matters such as clothing. Clearly, more research on gender and depression in diverse ethnic groups and in understudied nations is an important next step.

Cyranowski et al., 2000; Eley et al., 2004), or cognitive factors such as cognitive style or rumination (e.g., Hankin & Abramson, 2001; Nolen-Hoeksema & Girgus, 1994). Although the list of possible causal factors has been reviewed previously, what is lacking is a well-defined, integrated, testable, developmental model of gender differences in depression that (a) integrates affective, biological, and cognitive factors; (b) is consistent with the developmental timing of the emergence of the gender difference; and (c) takes into account the most recent research and theorizing in relevant areas. The purpose of this article is to articulate such a model.

Our proposed ABC (affective, biological, cognitive) model integrates affective, biological, and cognitive factors into a vulnerability–stress model in which these factors are presumed to confer vulnerability that, in the presence of stress, leads to depression and, specifically, to the gender difference in depression. A conceptual diagram of the model is shown in Figure 1. Each of the main elements of the model—*affective vulnerability, biological vulnerability, cognitive vulnerability, and negative life events*—is detailed and evaluated below. We emphasize the ways in which these vulnerabilities emerge or intensify in early adolescence. First, however, we consider conceptual issues in the measurement of depression, and we summarize three prior reviews on which the current model builds.

The Conceptualization and Measurement of Depression

One debate in depression research concerns the distinction between depressed mood, measured on a continuous scale by symptoms, and depressive disorders, measured categorically by diagnostic interviews (Flett, Vredenburg, & Krames, 1997). Consistent with the conclusions of many experts, we view depressed mood

and depressive disorders as variations along a continuum (Flett et al., 1997). For example, Hankin, Fraley, Lahey, and Waldman (2005) examined whether child and adolescent depression was continuous or categorical in a population-based sample of over eight hundred 9- to 17-year-olds. Youth and parents reported on *DSM-IV* depression symptoms in clinical interviews, and the data were analyzed using Meehl’s (1995) taxometric procedures. The researchers found that depression symptoms in this age group were best described as dimensional and not categorical; this was true for both the emotional distress and vegetative symptoms of depression.

Moreover, the evidence indicates that even subclinical or moderate levels of depressive symptoms are associated with diminished psychosocial functioning (Gotlib, Lewinsohn, & Seeley, 1995; Lewinsohn, Solomon, Seeley, & Zeiss, 2000), and the gender difference in depression is found whether assessed by symptom measures (e.g., Allgood-Merten, Lewinsohn, & Hops, 1990) or diagnoses (e.g., Kessler et al., 1993). In what follows, we use *depression* as a global term encompassing both diagnoses of depression and elevated scores on symptom scales.

Prior Reviews

Three prior reviews have been particularly influential in the conceptualization of the causes of gender differences in depression: those by Nolen-Hoeksema and Girgus (1994), Hankin and Abramson (2001), and Cyranowski and colleagues (2000). Here, we briefly review the major emphases of each.

Nolen-Hoeksema and Girgus (1994) considered the following etiological factors: personality (e.g., dependence on relationships, causal attributions, and ruminative coping), biological factors (hormonal changes at puberty), body dissatisfaction, and social chal-

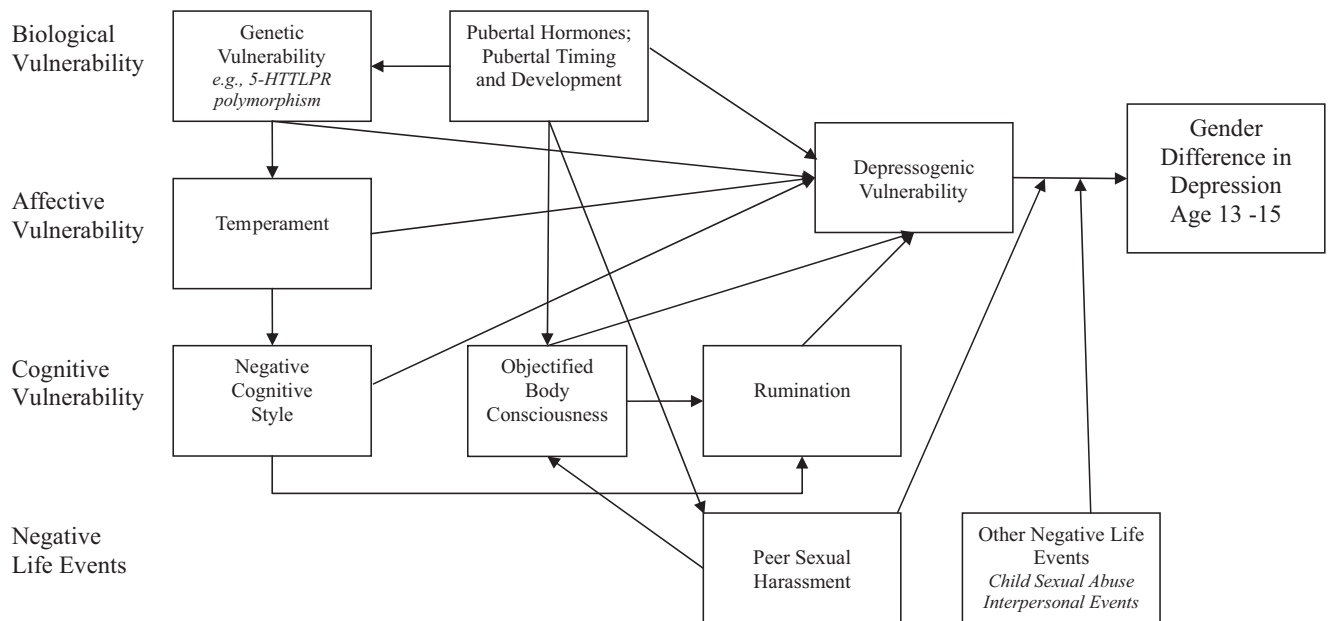


Figure 1. A conceptual diagram of the ABC model of the emergence of gender differences in depression in adolescence. (Depressogenic Vulnerability represents the collection of all the vulnerabilities, all of which are hypothesized to interact with negative events to yield gender differences in depression.)

lenges (rape and sexual abuse, parental and peer enforcement of gender roles). They found some evidence for each of these factors in the etiology of the gender difference, except that the data on hormonal influences were sparse and conflicting.

Nolen-Hoeksema and Girgus (1994) further proposed three basic developmental models of how the gender difference might appear. According to Model 1, the same factors cause depression in girls and in boys, but one or more of these factors become more prevalent for girls than boys in early adolescence. Model 2 posits that different factors cause depression in girls compared with boys and that the factors for girls become more common in early adolescence, whereas the factors for boys do not become more common or increase only slightly. Model 3 rests on an interactive model that hypothesizes that, even before puberty, girls have more vulnerability factors for depression than boys do but that it is not until early adolescence, when major new challenges arise, that the vulnerability factors interact with the challenges to produce more depression. Nolen-Hoeksema and Girgus concluded that the evidence best supported Model 3 and the interactions specified by it.

Hankin and Abramson (2001), beginning with a generic cognitive vulnerability–stress model, articulated a host of elaborations that incorporated aspects of research on rumination, interpersonal theories of depression, genetic vulnerability for depression, and developmental factors (pubertal onset). These added factors were hypothesized to occur both proximally and distally. Among the distal factors they considered were genetic vulnerability and early environmental adversity. The conceptualization of cognitive vulnerability, a proximal factor, was elaborated to include ruminative response styles and negative cognitions about body image. Initial negative affect was introduced into the cognitive vulnerability–stress causal chain. In addition, Hankin and Abramson elaborated the types and frequencies of stressful negative events that may interact with vulnerabilities to produce depression. Stimulated by interpersonal theories of depression (Hammen, 1991, 1999), Hankin and Abramson considered stress-generation processes and specifically interpersonal stress generation. Distinguishing between independent negative events (those outside the individual's control) and dependent events (those to which the individual contributes), they noted the evidence that depression itself can lead to increases in interpersonal negative events because the person may seek reassurance excessively or withdraw from others, leading to rejection. Hankin and Abramson further discussed how gender differences along points in this causal chain may explain the gender difference in depression.

In contrast to the cognitive vulnerability–stress model proposed by Hankin and Abramson (2001), Cyranowski and colleagues proposed a model emphasizing the role of pubertal hormones in interaction with gender role intensification in adolescence. They suggested that pubertal hormones “sensitize females to the depressogenic effects of negative events” (Cyranowski et al., 2000, p. 22). According to their model, this happens through the mechanism of increased affiliative needs driven by intensified gender-role socialization as well as pubertal increases in oxytocin. This pubertal intensification in affiliative needs combines with other depressogenic vulnerability factors, such as a highly anxious temperament, insecure attachment, and poor coping skills, to create an overall depressogenic diathesis that interacts with stressors in the adolescent transition to produce depression.

### Rationale for the ABC Model

Although the reviews by Nolen-Hoeksema and Girgus (1994), Hankin and Abramson (2001), and Cyranowski and colleagues (2000) all provide excellent articulations of how specific vulnerability factors may interact with a general increase in stressors in adolescence to produce the gender difference in depression, a new review and theoretical model are needed for several reasons. First, relevant research has mushroomed since the time of these reviews. For example, exciting new studies go far beyond the evidence of heritability of depression and point to specific loci that confer vulnerability to depression, some of which may be sex specific. New evidence links pubertal processes, particularly pubertal timing, with the emergence of the gender difference in depression. Recent theory and research go far beyond the construct of body image or body dissatisfaction to specify particular cognitive processes that link body dissatisfaction to depression, particularly in girls.

Second, although all three prior reviews considered multiple factors, each had a particular focus and, as such, did not fully integrate what we view to be the key causal factors. In contrast, we propose that there are important developmental relationships among affective, biological, and cognitive vulnerabilities so that these vulnerability factors do not operate simply in an additive manner, but, rather, the presence of one type of vulnerability may increase the likelihood of the development of another type of vulnerability, thus increasing an individual's overall depression vulnerability. For example, our model holds that affective vulnerability contributes developmentally to the emergence of greater cognitive vulnerability in early adolescence. As a second example, we propose that biological factors (genetic vulnerability) contribute to affective vulnerability. As a third example, we propose that biological factors (pubertal timing in girls) contribute to negative life events (peer sexual harassment victimization [PSHV]). In sum, we propose that important affective and biological features of development can be integrated with cognitive factors into a developmental vulnerability–stress model of gender and depression in substantive ways that neither ignore the unique contributions of these processes nor simply provide more pathways to depression that combine additively.

Table 1 summarizes the crucial elements of each of the three prior models as well as the ABC model. Note that the ABC model incorporates many elements of the previous models, but is distinctive in the following ways: (a) It is an integrative model, that is, it integrates key categories of factors. For example, genetic vulnerability (a biological factor) is presumed to influence temperament (an affective vulnerability), and temperament is presumed to contribute to negative cognitive style (a cognitive vulnerability; see Figure 1). (b) The ABC model incorporates cutting-edge research not available at the time the other models were proposed. Examples are new research on genetic polymorphisms that confer vulnerability to depression, research on objectified body consciousness (OBC), and research on peer sexual harassment. The combination of these two primary differences allows the ABC model to provide greater elaboration on the processes by which depression vulnerability develops than was evident in prior reviews.

The ABC model is a vulnerability–stress model of the emergence of the gender difference in depression insofar as we posit

Table 1  
*Comparison of the ABC Model With Prior Models*

Model component	ABC model	Nolen-Hoeksema & Girgus (1994)	Cyranowski, Frank, Young, & Shear (2000)	Hankin & Abramson (2001)
Biological vulnerability				
Familial risk	M			M
Specific genetic mechanisms	M			M
Pubertal hormones	M (DHEA, estradiol, progesterone, testosterone)	M	M (oxytocin)	
Pubertal timing	M			M
Affective vulnerability				
Personality style: dependency/affiliation needs		M	M	
Temperament: negative emotionality	M			M
Cognitive vulnerability				
Negative cognitive style	M			M
Rumination	M	M		M
Body image	M (objectified body consciousness)	M		M
Negative life events				
Broadly construed	M		M	M
Sexual abuse	M (abuse & harassment)	M (abuse only)		M (abuse only)
Interpersonal events	M		M	M (stress generation)
Pressure to conform to gender roles	M	M		
Summary: overall model	Integrates vulnerabilities developmentally across the affective, biological, and cognitive domains	Girls have more risk factors (rumination) before adolescence and face more stresses in early adolescence	Emphasizes one pubertal hormone (oxytocin) in interaction with gender role socialization	Emphasizes cognitive vulnerability and increased stress in the transition to adolescence

*Note.* M means that the factor is incorporated in the given model. ABC model = affective, biological, cognitive model.

that affective, biological, and cognitive vulnerabilities converge in early adolescence to form a depressogenic vulnerability that, in the presence of stress, produces depression. An important component of this model is the cognitive vulnerability–stress pathway, as articulated by Hankin and Abramson (2001). However, affective vulnerability (temperament) is also integrated into this pathway; we provide conceptual and empirical evidence that temperament contributes to the development of cognitive vulnerability and, in addition, is a vulnerability factor itself to depressive outcomes. Biological vulnerability (genetic vulnerability, puberty) is also integrated into this pathway. Again, we provide conceptual and empirical evidence that genetic vulnerability is a contributing factor and that it can be integrated into this pathway and that pubertal processes contribute to the development of cognitive vulnerability (particularly in the body image domain), exacerbate affective vulnerability, and evoke unique stressors that interact with other vulnerability factors. A conceptual diagram of the model is shown in Figure 1. Each of the main elements of the model— affective vulnerability, biological factors, cognitive vulnerability, and stress—is detailed and evaluated below.

The ABC model is a developmental model of the emergence of the gender difference in depression insofar as we examine how depressogenic vulnerability across three domains (affective, biological, and cognitive) emerges and/or increases in early adolescence to increase overall depressogenic vulnerability, particularly for female adolescents. We further elaborate on the increase in

stressors in early adolescence, again, particularly for girls, that interact with these increasing vulnerabilities to produce the gender difference in depression.

We retain Nolen-Hoeksema and Girgus's (1994) three generic developmental models to explain the emergence of the gender difference, but we modify them and identify additional possible models. Our first three models are similar to Nolen-Hoeksema and Girgus's, and the last three are additional models. Our goal in identifying these models is not to propose them as competing models because more than one may accurately describe relevant processes. Rather, our goal is to think of them as heuristics, which then allow us to spot patterns in complex data reviewed later in this article. For us, Developmental Model 1 states that the causes of depression are the same for boys and girls but that an important causal factor (or factors) becomes more prevalent for girls than boys in early adolescence. Empirically, this would imply that, if Factor A is an important causal factor, then the correlation between Factor A and depression is similar for boys and girls and mean levels of Factor A are similar for boys and girls in childhood but that mean levels of Factor A become higher for girls than boys beginning in early adolescence. Examples of factors in our model that may fit this pattern are negative cognitive style and negative life events (e.g., PSHV). Our Developmental Model 2 states that the causes of depression are somewhat different in girls compared with boys and that levels of the causes for girls rise in early adolescence. In this model, Factor A would correlate much more

highly with depression for girls than for boys, and levels of Factor A would rise in early adolescence. Examples of such a factor might be OBC or gonadal hormones. Our Developmental Model 3 states that girls are higher in vulnerabilities for depression even before adolescence and that the increases in stressful or negative life events in adolescence combine with the vulnerabilities to produce depression. That is, mean levels of Factor A are higher for girls than for boys even in childhood, but the gender difference in depression does not appear until the incidence of negative life events increases for girls at the beginning of adolescence. Our Developmental Model 4 holds that there is greater female variability in one or more of the vulnerability factors. That is, mean levels of Factor A are the same in boys and girls, but if girls display greater variance in Factor A, there will be more high-scoring, high-vulnerability girls. An example in our model might be temperament and, specifically, negative emotionality. Developmental Model 5 focuses on vulnerability–stress interactions and presents the possibility that a vulnerability–stress interaction may be more potent for girls than for boys. Finally, our Developmental Model 6 is a mixed model that specifies that there are multiple pathways to depression (e.g., Kendler et al., 2002), some of which are more common in girls than in boys, and that different pathways may conform to Developmental Model 1, 2, 3, 4, or 5. Each pathway explains only a small percentage of the variance in depression, but taken together, the multiple pathways can explain most cases and can explain the gender difference.

We maintain the distinction between vulnerability and risk originated by G. W. Brown and Harris (1978). Vulnerability factors are interactive; that is, the vulnerability factor, by itself, is not hypothesized to increase depression. Rather, vulnerability factors, combined with negative life events, increase the chance of depression. Risk factors, in contrast, show simple main effects that increase the chances of depression. Our model emphasizes vulnerability factors that interact with negative life events.

### Cognitive Vulnerability to Depression

#### *Cognitive Vulnerability–Stress Models*

Several recent reviews have articulated the general cognitive vulnerability–stress model of depression (Abramson & Alloy, 2006; Abramson et al., 2001; Hankin & Abramson, 2001). Cognitive vulnerability–stress models of depression posit that individuals with certain negative cognitive styles have greater chances of developing depression when they encounter negative or stressful life events. Two of the most well-examined cognitive vulnerability models of depression are the hopelessness theory and the ruminative response styles theory (Abramson, Metalsky, & Alloy, 1989; Nolen-Hoeksema, 2000). In this section, we review these cognitive vulnerability–stress models and the evidence supporting their utility in explaining the emergence of the gender difference in depression.

The hopelessness theory of depression proposes that individuals who make negative inferences about causality, self, and consequences in response to negative events will be most likely to develop depression in the wake of negative events (Abramson et al., 1989). Retrospective and prospective tests of the hopelessness theory in adults and adolescents have supported the hypothesis that these cognitive styles do in fact confer vulnerability to depression (Abramson et al., 1999; Alloy et al., 2000; Alloy & Clements,

1998; Alloy, Lipman, & Abramson, 1992; Hankin, Abramson, & Siler, 2001; Metalsky & Joiner, 1992; Metalsky, Joiner, Hardin, & Abramson, 1993; Schwartz, Kaslow, Seeley, & Lewinsohn, 2000).

Perhaps the most rigorous examination of the cognitive vulnerability–stress model in young adults occurred in the Temple-Wisconsin Cognitive Vulnerability to Depression (CVD) Project (Alloy et al., 2000). In this two-site study, college freshmen were selected on the basis of being high or low on standardized measures of cognitive vulnerability. Stressful life events and depression were assessed every 6 weeks over a 2.5-year period and then every 4 months for 3 additional years. Participants high in cognitive vulnerability who experienced more stressful life events were significantly more likely to develop depression over the course of the study. The cognitive vulnerability–stress model predicted both first episodes of depression (for participants high in cognitive vulnerability with no prior history of depression) and recurrences of depression (Alloy et al., 2000). To date, this is the most rigorous prospective study testing the core vulnerability–stress interaction predicted by the cognitive vulnerability–stress model of depression.

The cognitive vulnerability–stress interaction has been demonstrated in adolescents as well. For example, Hankin and colleagues (Hankin & Abramson, 2002; Hankin et al., 2001) found, in two studies, that cognitive style interacted significantly with negative events to predict depressive symptoms in high school students.

Overall, attempts to apply the cognitive vulnerability–stress model to predicting depression in children and younger adolescents have yielded inconsistent results that, we believe, reflect a particular pattern. According to two meta-analyses of studies of children and adolescents, attributional style correlates with depressive symptoms ( $r = .41$ , Gladstone & Kaslow, 1995; and  $r = .50$ , Joiner & Wagner, 1995). These correlations, however, test simple main effects and do not test the interaction of cognitive style and stress that is implied by the theory. Tests of this interaction have yielded more mixed results (Joiner & Wagner, 1995). Some prospective studies have found that negative attributional style, in interaction with negative events, predicts increases in depressive symptoms in children and adolescents (Abela, 2001; Abela & Sarin, 2002; Dixon & Ahrens, 1992; Hilsman & Garber, 1995; Nolen-Hoeksema, Girgus, & Seligman, 1992; Panak & Garber, 1992; Prinstein & Aikins, 2004; Robinson, Garber, & Hilsman, 1995; Schwartz et al., 2000; Turner & Cole, 1994). However, others have not found a significant cognitive vulnerability–stress interaction (Bennett & Bates, 1995; Cole & Turner, 1993; Hammen, Adrian, & Hiroto, 1988). Analysis by age indicates that negative attributional style, in interaction with negative events, typically predicts depression in children only after age 11 (Abela, 2001; Garber, Keiley, & Martin, 2002; Nolen-Hoeksema et al., 1992; Schwartz et al., 2000; Turner & Cole, 1994; but see Lewinsohn, Joiner, & Rohde, 2001), and these same studies failed to find this predicted interaction in children prior to age 11.

We hypothesize that one reason for the inconsistency in results is that children younger than 11 may not yet have formed a stable cognitive style that functions as a reliable vulnerability factor. Developmental researchers have maintained that late childhood, roughly ages 8–12 years, is a critical period for the development of the cognitive capacities necessary for forming attributions that distinguish between dispositional and situational causes (Dweck & Leggett, 1988; Fincham & Cain, 1986). Only in late elementary school do children conceive of ability as a stable trait that is

different from effort (Nicholls, 1978; Rholes, Blackwell, Jordan, & Walters, 1980; Stipek & MacIver, 1989). Consistent with the notion that cognitive style consolidates increasingly over time, 6- to 12-month test-retest reliabilities for cognitive style are only .37 for third graders (Nolen-Hoeksema et al., 1992) but are .50–.66 by early adolescence (Garber & Flynn, 2001; Nolen-Hoeksema et al., 1992) and are .80 for college students (Alloy et al., 2000).

We suspect that a second reason for the apparent inconsistency in the literature is a result of studying samples with large age ranges that encompass childhood and adolescence, when the interaction emerges only in early adolescence (e.g., the Cole & Turner, 1993, study included fourth, sixth, and eighth graders).

According to Nolen-Hoeksema's (1987, 1990, 2000) response styles theory, rumination represents another cognitive vulnerability factor in depression. People with a ruminative response style think repetitively and passively about the negative emotions elicited by negative events. Numerous studies confirm the prediction that ruminative response styles predict depression (reviewed by Nolen-Hoeksema, 2000). For example, a study of college students at the time of the 1989 San Francisco earthquake showed that those who were characterized by a ruminative style before the earthquake experienced elevated depressive symptoms shortly after the earthquake and 7 weeks after it, even controlling for depressive symptoms before the quake (Nolen-Hoeksema & Morrow, 1991). Rumination also predicts depressive disorders (Nolen-Hoeksema, 2000). The rumination-depression link has been found with third and seventh graders as well (Abela, Brozina, & Haigh, 2002).

Although most of the theorizing on rumination has focused on main effects of rumination on depression, Nolen-Hoeksema and Girgus (1994) briefly speculated that preexisting tendencies to ruminate may interact with an escalation in stressors at the beginning of adolescence to increase rates of depression (see also Nolen-Hoeksema, 1994). This approach is consistent with a cognitive vulnerability-stress interaction model. Nolen-Hoeksema, Larson, and Grayson (1999) tested the interaction of rumination and chronic strain with a sample of adults and found only weak evidence for it. Certainly, more empirical tests would be desirable.

### *Gender and Cognitive Vulnerability*

According to cognitive vulnerability-stress models, the gender difference in depression could be explained by one or more of several factors. First, females could demonstrate greater cognitive vulnerability either from childhood or beginning in early adolescence (Developmental Model 1 or 3). If gender differences in cognitive vulnerability do indeed contribute to gender differences in depression, then the gender difference in cognitive vulnerability should precede the gender difference in depression. Second, girls could experience more stressors beginning in early adolescence (Developmental Model 1). Third, the vulnerability-stress-depression relationship could be stronger for females than for males (Developmental Model 2). Here, we examine evidence for the first and third hypotheses, namely, that females demonstrate greater cognitive vulnerability that may contribute to the gender difference in depression and/or that the strength of the cognitive vulnerability-stress interaction is greater for females than for males. The second hypothesis is examined in the section on stressful life events.

Research with children under 11 indicates no gender difference in attributional style or that boys may have the more negative style

(Abela, 2001; Mezulis, 2004; Mezulis, Hyde, & Abramson, 2006; Nolen-Hoeksema, Girgus, & Seligman, 1991; Thompson, Kaslow, Weiss, & Nolen-Hoeksema, 1998). However, research with adolescents shows girls having more negative styles (Hankin & Abramson, 2002; Nolen-Hoeksema & Girgus, 1994).

There is some indication, in a study of high school students, that negative attributional style is more strongly linked to depression for girls than for boys (Gladstone, Kaslow, Seeley, & Lewinsohn, 1997). Another high school study found that negative cognitive style mediates the gender difference in depressive symptoms, reducing the gender-depression association by 53% (Hankin & Abramson, 2002). There is also evidence of a more potent cognitive vulnerability-stress interaction for adolescent girls compared with adolescent boys. Prinstein and Aikins (2004), beginning with 10th graders (16-year-olds), administered measures of attributional style, peer acceptance/rejection (a stressor), and depressive symptoms. They recontacted the students 17 months later and again assessed depressive symptoms. They found a three-way interaction between attributional style, peer rejection, and gender. For girls, the combination of a negative attributional style and peer rejection at Time 1 predicted depressive symptoms at Time 2, controlling for Time 1 depressive symptoms. The effect was not significant for boys.

Nolen-Hoeksema (2001; Nolen-Hoeksema & Girgus, 1994; Nolen-Hoeksema et al., 1999) proposed that the gender difference in depression can be accounted for, at least in part, by gender differences in the tendency to ruminate. In a 1-year longitudinal study of adults, for example, gender differences in rumination were significant at both Time 1 and Time 2 ( $d = 0.23$  and  $0.26$ , respectively; Nolen-Hoeksema, et al., 1999). Rumination had a direct effect on depressive symptoms, as well as a tendency to exacerbate the effects of chronic strain on depressive symptoms over time. Rumination has also been demonstrated to partially mediate the relationship between gender and depressive symptoms (Treyner, Gonzalez, & Nolen-Hoeksema, 2003; see also J. Roberts, Gilboa, & Gotlib, 1998).

Developmental research on gender differences in rumination has yielded mixed results, sometimes finding significant gender differences as early as fourth grade (age 9; Broderick, 1998; Broderick & Korteland, 2002) and sometimes not finding significant gender differences in childhood (Broderick & Korteland, 2004). Gender differences are significant in samples of early and middle adolescents (Compas, Malcarne, & Fondacaro, 1988) and college students (Nolen-Hoeksema, 1987).

Mezulis, Abramson, and Hyde (2002) expanded the conceptualization of rumination to explore domains about which individuals might ruminate. The traditional focus of rumination research, by both Nolen-Hoeksema and others, has been on rumination about depressed mood. It is possible, in addition, to ruminate about negative events. Mezulis and colleagues found that college women ruminated more than college men not only about depressed mood but also about negative events in three domains: achievement, interpersonal, and body image/attractiveness. However, gender differences in rumination about depressed mood and about negative events in the achievement domain were small ( $d = 0.24$  and  $0.28$ , respectively). Gender differences in rumination about interpersonal and body image events were considerably larger ( $d = 0.55$  and  $0.68$ , respectively). These findings highlight the importance of considering different domains of rumination and point to the crucial importance of the interpersonal and body image do-

mains in explaining gender differences in depression (Cyranowski et al., 2000; Nolen-Hoeksema & Girgus, 1994).

### *Cognitive Vulnerability in the Body Image Domain*

Cognitive vulnerability–stress models of depression to date have emphasized negative beliefs about one’s own worth and competence in the traditional domains of achievement and interpersonal relationships. However, theory and research in the psychology of women and gender suggest that the construct of cognitive vulnerability should be expanded to include negative beliefs about the body as well. Introducing negative cognitive processes related to the body (OBC) as a cognitive vulnerability factor provides one way to integrate the social and physical aspects of puberty into cognitive vulnerability to depression.

We propose that the tendency to have negative beliefs about one’s body is a form of cognitive vulnerability that is intensified in adolescence as a result of social and biological aspects of the adolescent transition. Prior reviews have noted that body dissatisfaction contributes to depression in adolescent girls (Hankin & Abramson, 2001; Nolen-Hoeksema & Girgus, 1994). OBC theory goes beyond these observations to specify that two processes are involved in the generation of negative beliefs about the body: (a) self-surveillance, a cognitive process in which individuals become observers and critics of their bodies and appearance; and (b) body shame, an affective component in which individuals feel shame when their bodies do not conform to cultural ideals (McKinley & Hyde, 1996; see also Fredrickson & Roberts, 1997). Importantly for the current argument, research supports the link between OBC and depressive symptoms. Body surveillance correlates significantly with depressive symptoms in samples of college women ( $r = .39$ , Muehlenkamp & Saris-Baglama, 2002;  $r = .26$  for women but only  $.09$  for men, Tiggemann & Kuring, 2004). In one longitudinal project, self-surveillance at age 11 significantly predicted depressive symptoms at age 13 for girls (controlling for age 11 symptoms), but the prediction from age 11 surveillance to age 13 depression was not significant for boys (Grabe, Hyde, & Lindberg, 2007). In addition, self-surveillance at age 11 predicted rumination at age 13 for girls but not for boys, suggesting that habitual self-surveillance, particularly among girls, may be a cognitive process that contributes to the development of habitual rumination. These observations suggest that OBC theory and rumination theory can be integrated, as shown in Figure 1.

*Gender and cognitive vulnerability in the body image domain.* Developmental changes unique to adolescence may increase both self-surveillance and body shame, particularly for girls. Early adolescence is a well-established time of intensified pressure to conform to gender role expectations. This gender-intensification hypothesis holds that pressures for gender-role conformity—for girls to be feminine (and not masculine) and for boys to be masculine—increase dramatically in early adolescence (Crouter, Manke, & McHale, 1995; Hill & Lynch, 1983; A. Petersen et al., 1991). Consistent with this hypothesis, boys increase in masculinity and girls increase in femininity over early adolescence (Galambos, Almeida, & Petersen, 1990; A. Petersen et al., 1991). The pressures come from multiple sources including family, peers, and the media. One important effect of gender intensification is to increase the salience of certain domains typically associated with gender-role expectations. For girls in adolescence, physical attrac-

tiveness is a key domain (Jackson, 1992). Pressure to conform to gendered expectations for appearance may contribute to gender differences in cognitive vulnerability in the appearance domain and may lead to the experience of more negative events in this domain. In addition, as we discuss in more detail below, puberty brings about changes in body fat and body shape that may be experienced by girls as negative and may evoke greater self-evaluation.

Numerous studies have found that girls and women have more negative body esteem than men and boys (e.g., McCauley, Mintz, & Glenn, 1988; Mendelson, Mendelson, & White, 2001; Mintz & Betz, 1986; Polce-Lynch, Myers, Kliewer, & Kilmartin, 2001). Research has linked these gender differences in body esteem to gender differences in depression. For example, Allgood-Merten et al. (1990) found that, in a high school sample, the gender difference in depression was eliminated when body image and self-esteem were controlled (see also Marcotte, Fortin, Potvin, & Papillon, 2002; Seiffge-Krenke & Stemmler, 2002, with a German sample). Supporting the hypothesis that the direction of influence goes from body esteem to depression, body image measures predict subsequent increases in depressive symptoms for adolescent girls, but depression does not predict body dissatisfaction (Stice & Bearman, 2001; Stice & Whitenton, 2002).

Research based in OBC theory has confirmed the existence of gender differences in self-surveillance, with girls engaging in it more (McKinley, 1998; Tiggemann & Kuring, 2004, with an Australian sample). The gender difference is moderate to large in magnitude in samples of college students; for example,  $d = 0.64$  in the Tiggemann and Kuring (2004) study, and  $d = 0.45$  in the McKinley (1998) study. Moreover, surveillance is negatively correlated with body esteem ( $r = -.39$ ) in samples of college students (McKinley & Hyde, 1996).

Our own data confirm developmental changes in the magnitude of the gender difference in self-surveillance. Using longitudinal data, we found that the gender difference in self-surveillance is already significant in fifth grade (age 11,  $d = 0.49$ ; Lindberg, Hyde, & McKinley, 2006). The gender difference in rumination is not significant at that age, nor is the gender difference in symptoms of depression (Grabe et al., 2007). By age 13, the gender difference in self-surveillance has grown larger ( $d = 0.64$ ), and the gender difference in rumination has become significant ( $d = 0.36$ ), as has the gender difference in depression ( $d = 0.31$ ; Grabe et al., 2007).

*Relationships among cognitive style, rumination, and objectified body consciousness.* There is a small body of evidence suggesting that cognitive style, rumination, and OBC may be related both over time and within individuals. The integration of these three vulnerabilities is recognized by the ABC model, and here we specifically outline two ways in which these vulnerability factors may be related. First, cognitive style may prospectively predict rumination. Cognitive style represents the negative content of cognitions following stress, whereas rumination primarily represents the perseveration of attention on negative emotional and cognitive content; conceptually, both are implicated in the development and maintenance of negative affect, and so one would expect at minimum a correlation between the two. However, Abramson and colleagues (2001) suggested that rumination may mediate the relationship between cognitive style and depression because it may be particularly difficult to disengage attention from highly negative thoughts regarding the causes and consequences of

negative events; evidence for this predicted mediation effect was found in the CVD study (Spasojevic & Alloy, 2001). It is an interesting and as yet untested question whether this mediation may operate developmentally specifically in the transition to adolescence, that is, if individuals who have more negative cognitive styles over time may be more likely to ruminate as well.

Second, the self-surveillance component of OBC may prospectively predict rumination as well; self-surveillance may represent a specific example of ruminative focus on the self that is both perseverative and evaluative. Evidence from our longitudinal data supports the prospective relationship between self-surveillance and rumination. For girls, self-surveillance at age 11 is significantly correlated with depression at age 11 and at age 13. Structural equation modeling confirmed that, for girls, rumination and body shame mediate the relationship between age 11 self-surveillance and age 13 depression, with age 11 depression controlled (Grabe et al., 2007). The model for boys is significantly different from that for girls; specifically, age 11 self-surveillance does not predict age 13 rumination. These data, taken together, support the notion that the gender difference in self-surveillance precedes and predicts gender differences in both rumination and depression and that self-surveillance may contribute, developmentally, to rumination.

In summary, our model posits three cognitive vulnerabilities to depression: cognitive style as originally proposed by Abramson and colleagues (1989), rumination as originally proposed by Nolen-Hoeksema (1991, 2000), and OBC. Both cognitive style and OBC are cognitive vulnerability factors that emerge in the early adolescent period as a result of cognitive development and puberty's biological, psychological, and social impact. One potential pathway between cognitive style on the one hand and depression on the other may be mediated through the additional cognitive pathway of rumination. The pathway from OBC to depression may also be mediated by rumination. All three show gender differences by early adolescence, with girls displaying the greater vulnerability. These vulnerabilities are then hypothesized to interact with negative life events in predicting depression.

### The Role of Temperament and Affect

Affective models of depression posit that temperament is associated with and predictive of depression as well as anxiety (Compas, Connor-Smith, & Jaser, 2004; Gjone & Stevenson, 1997; Goodyer, Ashby, Altham, Vize, & Cooper, 1993; Kendler et al., 1993; S. Roberts & Kendler, 1999; Rothbart & Bates, 1998). Rothbart and colleagues (Rothbart & Bates, 1998; Rothbart, Posner, & Hershey, 1995) delineated four possible pathways linking temperament and depression. Here, we highlight the importance of two of those pathways as critical to understanding the emergence of the gender difference in depression in adolescence: (a) mediation via cognitive vulnerability and (b) moderation via the interaction between temperament and environmental stressors. Thus, we hypothesize that temperament is a vulnerability factor for depression and that the effect may be partially mediated by negative cognitive style, which then predisposes the individual to depression (Mezulis, 2004; Mezulis et al., 2006).

Rothbart and Bates (1998) conceptualized temperament as constitutionally based individual differences in emotional, motor, and attentional reactivity and self-regulation. These individual differences are thought to be present from infancy and to be relatively

stable over time. A constellation composed of high negative affect, high reactivity, high intensity of emotional reactions, low adaptability, and low approach is typically labeled *negative emotionality* or *negative affectivity*. Children high in negative emotionality typically dislike and/or avoid novel situations; show distress to novelty; become upset, fearful, sad, or tearful easily; and appear highly sensitive to negative stimuli (Belsky, Hsieh, & Crnic, 1996; Buss & Plomin, 1986). By contrast, a constellation of temperamental characteristics composed of high positive affect, high sociability, high adaptability, and high approach is typically labeled *positive emotionality*. Children high in positive emotionality typically seek novel sensations and situations and are goal oriented, sociable, easygoing, and extraverted (Rothbart & Bates, 1998).

Several studies have found that both negative emotionality and positive emotionality are associated with depression in adults, adolescents, and children (Clark, Watson, & Mineka, 1994; Colder, Mott, & Berman, 2002; Goodyer et al., 1993; Kendler et al., 1993; S. Roberts & Kendler, 1999; Rothbart & Bates, 1998). The pattern of results indicates that low positive emotionality may be uniquely associated with depression, whereas high negative emotionality is a nonspecific factor predictive of both depression and anxiety (Anthony, Lonigan, & Hooe, 2002; Clark & Watson, 1991; Phillips, Lonigan, & Driscoll, 2002). In one of the few prospective studies examining the relationship between temperament and depression, Newman, Caspi, and Moffitt (1997) followed a large community sample of children from age 3 to age 21. They found that observer-rated behavioral inhibition in early childhood predicted greater diagnoses of depression in adulthood. Davies and Windle (2001) found evidence for a temperament–environment interaction, with temperamental characteristics typical of negative emotionality (low concentration, low rhythmicity, and low adaptability) significantly interacting with parents' marital discord to predict depression. Moreover, the relationship between temperament and depression holds even when overlap between temperament and symptom measures is removed (Lengua, West, & Sandler, 1998).

Our model proposes three relationships between negative emotionality and depression vulnerability in adolescence. First, we propose, as outlined above, a direct relationship between negative emotionality and depression that represents an underlying affective vulnerability present since childhood and continuing into the adolescent period; this vulnerability becomes salient when it encounters the particular negative life events that become prominent in early adolescence. Second, we suggest that negative emotionality contributes developmentally to greater cognitive vulnerability, which, as discussed above, emerges in early adolescence. Third, we propose that hormonal and social changes associated with puberty may exacerbate aspects of negative emotionality, specifically emotional reactivity to stress, for some already vulnerable individuals. The first relationship between affective vulnerability and depression is outlined above. Below, we examine the other two hypotheses.

Our model integrates cognitive and affective approaches by hypothesizing that negative emotionality contributes to the development of the negative cognitive style that confers vulnerability to depression. In support of this hypothesis, Rothbart and Bates (1998) argued that temperament directly affects children's perception of the aversiveness of events. Negative emotionality may be associated with a greater perception of novel events as stressful

(Costa, Somerfield, & McCrae, 1996). Children high in this temperamental characteristic may be more likely to experience intense negative affect in response to negative life events than children low on this dimension. Weiner (1985) noted that individuals' affective responses to negative events precede their cognitive responses of making attributions for the event. The quality of the affective response may affect the nature of the attributions made. For example, the stronger the aversive affective reaction, the more likely people are to interpret events as catastrophic (Teasdale, 1988). Negative emotionality is also associated with greater attention to negative events (Derbyberry & Reed, 1994), an increase in self-focus that is in turn associated with increased negative expectancies for the future (Pyszczynski, Holt, & Greenberg, 1987), and more focus on negative aspects of the self, other people, and the world (Watson & Clark, 1984).

Few studies have examined the relationship between negative emotionality and cognitive vulnerability to depression. Lengua, Sandler, West, Wolchik, and Curran (1999) assessed temperament, cognitive appraisals of negative events, and depression in a sample of children whose parents had recently divorced. Children identified three stressful events and rated several negative cognitions they had in response to these events, including negative self-evaluation, perceived rejection by others, and loss. Lengua and colleagues found that children's cognitive appraisals of negative events mediated the relationship between negative emotionality and depression, even after controlling for children's ratings of how emotionally upset each event made them. Mezulis and colleagues (2006), in a prospective study, found that greater mother-rated withdrawal negativity in infancy interacted with child-reported negative life events in the past year to predict negative cognitive style at age 11. Children high in negative emotionality who experienced greater numbers of negative life events had the most negative cognitive styles. Thus, the available evidence, although limited, supports the hypothesis that negative emotionality is associated with making more negative cognitive evaluations of negative events. Over time, this pattern of negative cognitive evaluations of events then contributes to the development of a stable negative cognitive style, which confers vulnerability to depression.

We also suggest that normal adolescent biological development across the pubertal period exacerbates emotional reactivity for all adolescents, a developmental trend that may be particularly depressogenic for adolescents who are already affectively vulnerable. Adolescence involves dramatic biological changes, including changes in mean levels and variability in hormones associated with stress and neurotransmitters associated with emotion regulation (pubertal hormones are reviewed in more detail below). Compared with children and adults, adolescents report more extreme negative emotions in response to stress (Larson, Csikszentmihalyi, & Graef, 1980; Larson & Richards, 1994) as well as greater physiological responses to stress (Allen & Matthews, 1997). Spear (2000) suggested that these changes in stress sensitivity may increase already vulnerable adolescents' vulnerability to a host of psychopathologies, including depression.

For the question of gender differences in depression, low positive emotionality seems to be an unlikely pathway because there is no gender difference in positive emotionality early in life (see below). High negative emotionality is the likely path insofar as the model posits that a contributing factor is stronger negative emotional responses to stressful events. The relationship between temperament and depression may be moderated by negative life events (an affective vulnerability–stress pathway) and may be partially

mediated by negative cognitive style. Moreover, a gender difference in anxiety, girls and women being more anxious, has been found consistently in research (Feingold, 1995), and depression and anxiety are frequently comorbid. These observations, too, lend support to the hypothesis that negative emotionality, rather than positive emotionality, is the dimension of temperament to consider when predicting adolescent gender differences in depression.

In summary, we propose that temperament—specifically, high negative emotionality—confers affective vulnerability to depression via a pathway that is partially mediated by cognitive vulnerability (see Figure 1). Emotional reactivity may involve both a stable vulnerability component that continues to influence depressive outcomes in adolescence and a vulnerability component that is exacerbated by the biological changes of adolescence.

### *Gender Differences in Temperament*

If temperament—specifically, negative emotionality and/or positive emotionality—is predictive of depression, then the emergence of gender differences in depression in adolescence would be explained by any of five possibilities: (a) a gender difference in negative emotionality, with girls scoring higher, combined with an increase in negative life events in early adolescence (Developmental Model 3 described earlier); (b) a gender difference in positive emotionality, with girls scoring lower (Developmental Model 3); (c) a stronger association between negative emotionality and cognitive vulnerability for girls than for boys; (d) a more potent interaction between temperament and negative life events for girls than for boys (a variation of Developmental Model 2); or (e) in the absence of an average gender difference in temperament, a greater variance in scores for girls so that more girls are above the cutoff for high negative emotionality or below the cutoff for low positive emotionality (Developmental Model 4).

A meta-analysis synthesized the diverse studies of gender differences in temperament (Else-Quest, Hyde, Goldsmith, & Van Hulle, 2006). For several measures associated with negative emotionality, findings of no gender difference were the rule: For emotionality,  $d = 0.01$ ; for negative affect,  $d = -0.06$ ; for sadness,  $d = -0.10$ , all nonsignificant (negative values indicate girls scoring higher on that dimension). Similarly, positive affect (pleasure) showed gender similarities ( $d = -0.09$ ). There was some support for the possibility of greater female variance in temperament. The statistic used to evaluate this hypothesis is the variance ratio (VR), which is the ratio of the male variance to the female variance. Thus, values  $> 1.0$  reflect greater male variance, and values  $< 1.0$  reflect greater female variance. For emotionality,  $VR = 0.94$ , indicating slightly greater female variance. For negative affectivity,  $VR = 0.88$ , again indicating somewhat greater variance among girls than among boys. This greater female variance could create a larger pool of girls at the upper tail of the distribution, that is, more girls than boys scoring high on this dimension, creating more vulnerable girls in the absence of a gender difference in mean scores.

In summary, we propose that early temperament—negative emotionality—contributes to the development of depression in two ways: It is a vulnerability itself that, in interaction with stress, contributes to depression, and it contributes to negative cognitive style. There is little evidence for a gender difference in negative emotionality, but girls may show somewhat greater variance on this dimension, creating a distribution in which there are more high-scoring girls.

Below, we consider biological factors that may contribute to depression, focusing first on genetic factors. We hypothesize that temperament may represent a pathway between genetic vulnerability and depression.

### Biological Factors

Biological factors that may contribute to depression and specifically to gender differences in depression include genetics, hormones and pubertal processes, and the brain and neural processes.

#### *Genetic Vulnerability*

A number of lines of evidence point to genetic influences on depression. In addition, some of the evidence indicates that some genetic vulnerability may be gender linked.

Sullivan, Neale, and Kendler (2000) meta-analyzed studies using genetic designs to evaluate family resemblance for major depression. On the basis of twin studies, depression was moderately heritable, with 37% of the variance due to additive genetic effects. Individual-specific environment effects, such as negative life events that occur to one twin but not the other, also contributed substantially to the variance (63%), but measurement error could not be separated from individual-specific environmental variance.

Agrawal, Jacobson, Gardner, Prescott, and Kendler (2004) concluded that depression is equally heritable in males and females (see also Glowinski, Madden, Bucholz, Lynskey, & Heath, 2003). Another twin study, however, found evidence that depression is more heritable in boys and that environmental factors account for more variance in girls' depression (Rice, Harold, & Thapar, 2002). This question is therefore unresolved at the moment.

Earlier in this article, we noted the stress-generation hypothesis—that depression itself may contribute to greater stress in one's environment (Hammen, 1991, 1999; Hankin & Abramson, 2001). Consistent with this view, twin heritability research shows that depression and some negative life events share common additive genetic variance (e.g., Thapar, Harold, & McGuffin, 1998). Therefore, the genetic factors that contribute to depression may also lead the vulnerable individual to experience more stress.

Beyond the estimation of heritabilities, research has advanced considerably with the Human Genome Project. With the goal of identifying specific loci linked to depression, several studies have been conducted using genomewide scans to detect linkage among relatives affected by depression. Significant linkage has been observed for chromosome 15q, and the effects were not gender specific (Holmans et al., 2004). Suggestive but inconclusive evidence was also found for linkages on chromosomes 6, 8, and 17. Another study, using a different sample showing a strong family history of depression, identified a region on chromosome 12 that confers a predisposition to depression in men, but not women; that is, the genetic effect is gender specific (Abkevich et al., 2003). (For other examples, see Zubenko, Hughes, Maher, et al., 2002; Zubenko, Hughes, Stiffler, Zubenko, & Kaplan, 2002.)

Using genotyping methods, several specific loci have been identified that confer vulnerability to depression. Research has documented an association between major depression and serotonin receptor genes HTR1A (Lemondé et al., 2003; Neumeister, Young, & Stastny, 2004) and HTR2A (Eley et al., 2004; for a review of these and hypotheses about other loci and neurotrans-

mitters, see Levinson, 2005). Another candidate gene is TPH2, the tryptophan hydroxylase gene that is active in the central nervous system; TPH is the rate-limiting enzyme in serotonin synthesis (Li & He, 2006; Walther et al., 2003). Imaging genetics have also demonstrated a link between this polymorphism and amygdala reactivity (S. M. Brown et al., 2005). Yet another candidate is the MAOA gene (Gutierrez et al., 2004; Yu et al., 2005). Monoamine oxidase A plays a key role in the metabolism of several neurotransmitters, including serotonin.

Perhaps the most promising avenue of research linking specific loci to depression has been on genes related to the serotonin transporter (5-HT) system. The 5-HT system is an excellent candidate given the success of the selective serotonin reuptake inhibitor antidepressants in treating depression (Tamminga et al., 2002). Genotyping studies have shown an association between major depression and the short variant (s allele) of 5-HTTLPR, the promoter region of the 5-HTT (serotonin transporter) gene located on chromosome 17, in both adults (Hoefgen et al., 2005) and samples of children and adolescents (Kaufman et al., 2004; Nobile, Cataldo, & Giorda, 2004). Many of these studies support a vulnerability–stress model in which the s allele of 5-HTTLPR confers a vulnerability to depression; when combined with stress, depression is a likely outcome (Caspi et al., 2003; Kaufman et al., 2004; Kendler, Kuhn, Vittum, Prescott, & Riley, 2005; Zalsman et al., 2006). Occasional failures to replicate the genetic effect (e.g., Young, Smolen, Stalling, Corley, & Hewitt, 2003) may be due to omission of stress from the model or the testing of children prior to adolescent increases in depressive symptomatology.

What are the processes by which the 5-HTTLPR polymorphism may affect depression? New imaging genetics research by Hariri and colleagues points to plausible structures and functions (S. M. Brown & Hariri, 2006; Hariri et al., 2005; Hariri, Drabant, & Weinberg, 2006; Heinz et al., et al., 2007; Pezawas et al., 2005). Using fMRI methods, this research has shown that persons who are carriers of the short allele display hyperreactivity of the amygdala in response to threat stimuli (Hariri et al., 2005; Heinz et al., 2007). Regions of the prefrontal cortex, also known to be involved in emotion information processing, have also been linked to 5-HTTLPR and to temperament (S. M. Brown & Hariri, 2006; Hariri et al., 2006).<sup>2</sup>

<sup>2</sup> A complete review of research on brain structures and functions related to depression and of adolescent brain development is beyond the scope of this article. Major reviews are available elsewhere (Cameron, 2004; Dahl, 2004; Dahl & Hariri, 2005; Romeo & McEwen, 2006; Spear, 2000). In brief, three changes may be relevant here. First, synaptic pruning occurs in the cortex, leading to more efficient and focused information processing. We might speculate that this more focused information processing may support rumination and self-surveillance related to OBC. Second, because the adolescent brain is developing so rapidly, it is characterized by great plasticity and therefore may be more vulnerable to stress (Romeo & McEwen, 2006). Third, the adolescent brain develops less sensitivity to rewards (Spear, 2000), which may contribute to depression insofar as reduced experience of positive reinforcement (Lewinsohn, Hoberman, & Hautzinger, 1985) or reduced positive affect (Clark & Watson, 1991) lead to depression. Moreover, insofar as pubertal development—and specifically brain development—occurs somewhat earlier in girls than in boys (Giedd et al., 1999), all three of these changes may occur earlier in girls, creating greater vulnerability in them at earlier ages.

*Gender and genetic vulnerability.* A number of mechanisms may explain how genetic vulnerability can contribute to gender differences in depression: more potent genotype–stress interactions for females than for males, X-linked genetic factors, gene–gene interactions, sex-specific trait loci, hormone-induced gene expression, and sexual differentiation of the brain. For example, for 5-HTTLPR, Eley and colleagues (2004) found a significant genotype–stress interaction involving 5-HTTLPR for female, but not male, adolescents (see also Sjöberg et al., 2006). This finding fits Developmental Model 5 discussed earlier. Two studies have found evidence that the MAOA gene, located on the X chromosome, conferred vulnerability to depression more in women than in men (Gutierrez et al., 2004; Yu et al., 2005).

Other possible mechanisms through which genetic polymorphisms may contribute to gender differences in depression include gene–gene interactions (e.g., Jabbi et al., 2007, for MAOA and COMT), sex-specific trait loci (e.g., Curtisinger, 2002; De Vries, 2004, 2005; Nuzhdin, Pasyukova, Dilda, Zeng, & Mackay, 1997; Zhao, Ma, Cheverud, & Wu, 2004), and hormone-induced gene expression (e.g., Marino, Galluzzo, & Ascenzi, 2006; Tung et al., 2006). For example, estrogen regulates gene expression in the developing midbrain and specifically increases serotonin receptor levels in both the midbrain and hypothalamus (Beyer et al., 2003; Zhou, Cunningham, & Thomas, 2002).

Whatever the final outcome of the genome studies, mediating processes will need to be identified. These are likely to involve synthesis of neurotransmitters such as serotonin and structures such as the amygdala (Eley et al., 2004; Hariri et al., 2006). We believe that the behavioral and developmental link between these genetic factors and the phenotype of depression is in part through temperament. Indeed, the 5-HTTLPR region has been linked to negative emotionality in 2-month-old infants (Auerbach et al., 1999).

In summary, depression shows moderate heritability. A number of specific loci have been identified that confer vulnerability to depression, presumably through their effects on the serotonin system; evidence for the 5-HTTLPR polymorphism is particularly strong. These genetic factors interact with environmental stressors in producing depression. A number of mechanisms, such as more potent genotype–stress interactions for females, X linkage, or hormone-induced gene expression, may explain how these genetic effects could create gender differences in depression. Genotype–stress interactions can account for adolescent emergence if stressors such as peer sexual harassment and body image concerns increase sharply for girls in early adolescence (Developmental Model 2). In short, known genetic mechanisms are proposed to contribute to the adolescent upsurge in depression and specifically to the emergence of the gender difference in depression between ages 13 and 15.

### *Puberty and Hormones*

The timing of the emergence of gender differences in depression between 13 and 15 years of age implicates pubertal processes. Here, we review evidence regarding both pubertal timing and development and sex hormones associated with puberty (for a review of gender differences at puberty, see Hayward, 2003).

Puberty is not a single event but rather a process that occurs over several years, resulting in adult appearance and physiology. Pu-

erty is actually preceded by adrenarche—the time of increasing secretion of adrenal androgens—which generally begins somewhat before age 8 (Grumbach & Styne, 1998; Remer, Boye, Hartmann, & Wudy, 2005). The adrenal gland secretes adrenal androgens, including dehydroepiandrosterone (DHEA) and its sulfate (DHEAS; see Sulcová, Hill, Hampl, & Stárka, 1997, for data on gender differences in DHEA and DHEAS levels over the life span). Adrenal androgens stimulate the growth of pubic and axillary hair, which generally appears around age 8 in both girls and boys. Adrenarche is then followed by gonadarche, which involves the maturation of the gonads—ovaries in females, testes in males—and sharp increases in their production of the sex hormones estrogen, progesterone, and testosterone. These changes then create the other changes of puberty.

Despite much publicity about dramatic cases of early puberty, the age of menarche—first menstruation—has held constant for the last several decades in the United States at 12.8 years for Whites and 12.2 years for African Americans (Fechner, 2003). The comparable event for boys is oigarche or first ejaculation (Kaltiala-Heino, Marttunen, Rantanen, & Rimpela, 2003). Pubertal processes occur somewhat earlier in girls than in boys (Fechner, 2003). Male gonadal development occurs 6 months to a year later than in females, and the growth spurt in height occurs roughly 2 years later, on average, in boys compared with girls. In a large Finnish study, 51% of girls had experienced menarche before age 13, compared with 38.4% of boys who had experience oigarche before age 13 (Kaltiala-Heino, Marttunen, et al., 2003).

*Gender and pubertal processes.* Pubertal timing (early, on time, or late) and development (stage of development at a particular time point) are crucial to understanding the transition to adolescence and the emergence of gender differences in many domains, including depression. Early puberty in general is disadvantageous for girls (Caspi, Lynam, Moffitt, & Silva, 1993; Caspi & Moffitt, 1991; Ge, Conger, & Elder, 1996). For example, Ge and colleagues (1996) found that early puberty was associated with psychological distress (depression and anxiety) in adolescent girls (see also Graber, Lewinsohn, Seeley, & Brooks-Gunn, 1997; Seiffge-Krenke & Stemmler, 2002; Stice, Presnell, & Bearman, 2001). In the largest study to date, Kaltiala-Heino, Kosunen, and Rimpela (2003) found that early puberty was associated with depression among girls; among boys, both very early and late puberty were associated with depression.

Numerous hypotheses have been proposed regarding the processes involved in the effects of pubertal status and timing (Caspi & Moffitt, 1991; Ge et al., 1996). Here, we focus on five that are particularly relevant: (a) The early timing hypothesis holds that numerous developmental tasks must be accomplished in childhood before the transition to adolescence if that transition is to be successful. Early-puberty girls and perhaps early-puberty boys therefore have had insufficient time to accomplish these developmental tasks, putting them at risk for psychological distress. (b) Various physical changes across the span of pubertal development (e.g., breast development and menarche) reorganize the girl's body image (Brooks-Gunn & Petersen, 1983; Koff, Rierdan, & Silverstone, 1978), which may itself be a stressor and may increase OBC. (c) Physical changes associated with puberty, for example, breast development, result in a changed social environment for a girl. Peers react to her differently. Boys are more likely to sexualize her, and she is more likely to be the object of peer sexual

harassment, as discussed below. These effects are particularly serious for early-puberty girls. (d) The overweight hypothesis argues that the weight gain associated with puberty makes girls—but not boys—feel overweight, which makes them vulnerable to depressed mood (Ge, Elder, Regnerus, & Cox, 2001). (e) The interaction hypothesis holds that pubertal timing is a vulnerability that interacts with stressful events to increase the risk for depression (Ge, Conger, & Elder, 2001). Ge, Conger, and Elder (2001), in one of the few studies to test several of these hypotheses simultaneously for the outcome of depression, found support for the early timing and interaction hypotheses for girls but not for boys.

Another line of evidence implicating puberty in the emergence of gender differences in depression comes from studies that have compared the relative importance of age versus pubertal stage as predictors of internalizing disorders. Angold, Costello, and Worthman (1998), in a large, well-sampled study, found that pubertal stage better predicted the gender difference in depression than age did; girls were more likely to be depressed only after mid-puberty, Tanner Stage III and above. In a large Australian sample, menarche was the best predictor of increases in depression and anxiety (Patton et al., 1996).

We believe that early puberty is a vulnerability factor for girls that, when combined with stressors, leads to depression. Following especially from Hypotheses b, c, and d above, early puberty is less of a vulnerability factor for depression in boys in large part because puberty moves boys more toward the idealized masculine, muscular body type. For girls, in a society that glamorizes thinness in women, puberty means added fat, which moves them away from the ideal body (Stice, 2003). Thus early-puberty girls should be most vulnerable, and puberty—especially early puberty—results in a changed social environment for girls that makes them more vulnerable to objectification and peer sexual harassment (Hypothesis c, changed social environment). All of these may have interactive effects (Hypothesis e) insofar as the effects of puberty are exacerbated by stressors but may be buffered by protective factors.

Our data from the Wisconsin Study of Families and Work provide support for these links in the model. We hypothesized that, especially for girls, pubertal development leads to increased PSHV as well as to increased body mass index (BMI), reflecting the addition of fat. PSHV in turn heightens body surveillance. Body surveillance and higher BMI both predict greater body shame. As hypothesized, at age 11, stage of pubertal development predicted the extent of PSHV for girls, but not boys (J. Petersen & Hyde, 2007). For girls at age 11, all paths in the model were significant (Lindberg, Grabe, & Hyde, 2007). The boys' model was significantly different, and the paths from pubertal development to PSHV and from pubertal development to BMI were not significant. To use the developmental models stated earlier, these results help to account for the gender difference in depression using a Developmental Model 2 explanation: Somewhat different factors predict depression symptoms for girls compared with boys (paths in the model are significant for girls but not for boys), and levels of these factors (pubertal development, self-surveillance, and BMI) rise for girls in early adolescence.

*Pubertal hormones.* Yet another possibility is that the increased rate of depression in adolescent girls may be associated with the pubertal rise in the adrenal androgens (DHEA and

DHEAS), sex steroids (estradiol, progesterone, and testosterone), and/or the gonadotropins (FSH and LH).

Several studies implicate the adrenal androgens in gender differences in depression (van Broekhoven & Verkes, 2003). Nottelmann, Susman, Inoff-Germain, et al. (1987), with a sample between the ages of 9 and 14, found that adjustment problems for girls are associated with low levels of DHEAS. In various samples, Goodyer and colleagues (2000) have found that DHEA hyposecretion or hypersecretion is associated with major depression in adolescence, suggesting the possibility of a U-shaped function relating DHEA levels and depression. Moreover, administration of DHEA to women with adrenal insufficiency reduces depression and anxiety (Arlt et al., 1999; see also Wolkowitz et al., 1999). The mechanism behind this action appears to be that DHEA protects against the negative effects of cortisol in the brain and also modulates various neurotransmitters, including raising serotonin levels (van Broekhoven & Verkes, 2003).

Numerous studies have linked estrogens to depression and the neurotransmitters known to be associated with depression, including MAO and serotonin (for reviews, see Chakravorty & Halbreich, 1997; Halbreich & Kahn, 2001; Östlund, Keller, & Hurd, 2003; Rubinow, Schmidt, & Roca, 1998; Steiner et al., 2003; Wissink, van der Burg, Katzenellenbogen, & van der Saag, 2001). In brief, estrogen—like other gonadal steroid hormones—acts on intracellular estrogen receptors (Rubinow & Schmidt, 2003). When the hormone binds to the receptor, a cascade of events ensues that modulates the transcription of genes that encode the manufacture of numerous proteins. Among these are proteins necessary for the synthesis of serotonin. Estrogen also increases serotonergic postsynaptic responsivity, increases the number of serotonergic receptors, increases the transport and uptake of serotonin (Halbreich & Kahn, 2001), and operates through both genomic and nongenomic mechanisms (Sanborn & Hayward, 2003). Postpartum depression and premenstrual dysphoric disorder both occur at the time of low estrogen levels, again suggesting a link.

If low estrogen levels are associated with depression, then why would estrogen be associated with depression in pubertal girls, whose estrogen levels are rising? One hypothesis is that estrogen homeostasis is disrupted during puberty and that this disrupted homeostasis may disturb serotonin processes and thereby trigger mood disorders (Halbreich & Kahn, 2001). Altemus (2006) argued that, more generally, women's experience of greater hormone fluxes across the life span play a role in depression by destabilizing homeostatic systems. This explanation is consistent with Brooks-Gunn and Warren's (1989) finding that among pubertal girls, negative affect occurs specifically at the time of rapid increases in estradiol levels.

Finally, testosterone—both level and diurnal variation—has been associated with several forms of psychopathology in both boys and girls around ages 11 to 13. Angold, Costello, Erkanli, and Worthman (1999) found that testosterone levels increase substantially over the five Tanner stages of puberty for girls and that higher levels of testosterone are associated with higher rates of diagnosable depression. Granger et al. (2003) also found positive correlations between testosterone levels and depressive symptoms for girls. For boys, lower levels of testosterone are associated with higher levels of anxiety–depression. Testosterone is also known to

have effects on multiple neurotransmitter systems, including serotonin, dopamine, and GABA (Rubinow & Schmidt, 1996).

Links between hormones and depression are likely to be complex, nonlinear, and interactional. For example, a study of men showed a U-shaped relationship between depressive symptoms and testosterone levels; men with low levels or high levels of testosterone reported the most depressive symptoms, compared with those with moderate levels (Booth, Johnson, & Granger, 1999). As noted earlier, Goodyer and colleagues (2000) found a similar curvilinear relationship between DHEA levels and depression among adolescents. A curvilinear effect was also found for the relationship between estradiol and negative affect for early adolescent girls; depressive affect is highest for girls with rapidly increasing but subadult levels of hormone (Brooks-Gunn & Warren, 1989).

Interactions have been found as well. Booth, Johnson, Granger, Crouter, and McHale (2003), with a sample of youth between the ages of 6 and 18 years, found that testosterone's positive relation to risk behavior and negative relation to depression depended on the quality of parent-child relations. Testosterone did not show simple correlations with depression for either boys or girls. However, for both boys and girls, testosterone levels interacted significantly with the quality of the parent-child relationship in predicting depressive symptoms. Boys with low testosterone levels and low-quality parental relationships showed the highest levels of depressive symptoms; the same was true for girls.

These complex processes can be organized into six possible models for the association between pubertal hormones and adolescent affect (see Brooks-Gunn, Graber, & Paikoff, 1994, for the first three; see also Susman, 1997). We introduce these not as competing models but rather as heuristic devices. The models are as follows: (a) According to the *direct hormonal effects model*, hormones have a direct, linear effect on negative affect. Brooks-Gunn and colleagues (1994) dismissed this model, which guided early research, as being far too simple and as having little explanatory power empirically. (b) The *indirect reactivity model* postulates that hormones do not have direct effects on affect but rather have indirect effects through their effects on some more general physiological system such as arousal, neurotransmitters, or reactivity to stressors. An example of evidence for this model is the research on the effects of estrogen on the serotonin system discussed earlier. (c) According to the *indirect secondary sexual characteristics model*, the effect of hormones on negative affect is mediated by the physical changes—such as breast development—that are created by pubertal hormones; these secondary sex characteristics can in turn create an adverse social environment, such as increased peer sexual harassment. Data reviewed earlier, showing that for girls, Tanner stage of pubertal development in early adolescence predicts the amount of peer sexual harassment they receive (J. Petersen & Hyde, 2007), are consistent with this model. (d) The *interaction model* states that social context or events, in addition to hormones, also contribute to negative affect, either directly or as moderators of the effects of hormones. In one study of adolescent girls, hormones accounted for 4% of the variance in negative affect, social events accounted for 8% to 18% of the variance, and the interaction of pubertal development with negative life events accounted for 9% to 15% of the variance (Brooks-Gunn & Warren, 1989). As noted earlier, in one study testosterone levels interacted with the quality of the parent-child relationship

to predict depressive symptoms (Booth et al., 2003). (e) Nonlinear effects of hormones on depression may occur. For example, a U-shaped curve might relate hormone levels to depressed affect, with the best psychological functioning associated with intermediate levels of hormones. An example is the study by Booth and colleagues (1999), reviewed earlier, in which the relationship between testosterone levels and depression among men was U-shaped. These nonlinear effects may be direct effects of hormones or may interact with negative life events or social context.

In summary, the evidence suggests that DHEA, estrogen, and/or testosterone may be involved in the increase in girls' depression at ages 13 to 15, although certainly they are not the sole cause. Current research does not definitively identify which hormone or hormones is most potent, but research does point to the likelihood of nonlinear and interaction effects. In addition, pubertal development and timing are important, especially insofar as early puberty is disadvantageous for girls, in part because they are then sexually objectified by peers. Hormone-gene interactions are another possibility. We cannot reach a definitive conclusion regarding the effects of hormones or pubertal development on gender differences in depression. Our goal, instead, has been to suggest possible mechanisms and review related research, leaving it to future researchers to test the models against each other.

### The Brain

A review of research on brain structures and functions that contribute to depression is beyond the scope of this article. (For reviews, see Charney & Nestler, 2004; Davidson, Pizzagalli, Nitschke, & Putnam, 2002; Thase, Jindal, & Howland, 2002.) We therefore restrict ourselves to noting two areas of brain differentiation that are relevant to gender differences.

It has been known for decades that the developing brain undergoes sexual differentiation once the SRY gene has caused differentiation of the testes and they are producing testosterone, which then bathes the developing male brain (e.g., Phoenix, Goy, Gerall, & Young, 1959; for newer research, see Dewing, Shi, Horvath, & Vilain, 2003). Research has not yet documented the connections to depression or gender differences in depression, but these very basic gender-differentiated mechanisms leading to brain differentiation must certainly be on the list of possible processes.

A second area worth noting is that gender differences have been found in serotonin neurotransmission in human brains (e.g., Nishizawa et al., 1997; Sakai et al., 2006). Again, these basic processes are worthy of future research attention.

### Negative Life Events

Negative or stressful life events are a key component of our model (see Figure 1). The affective, biological, and cognitive components of our model of depression are predicated on interactions between these vulnerabilities and negative or stressful life events. Indeed, these vulnerability factors should not necessarily lead to depression in the absence of negative events. It is well established that stressful life events are associated with the onset of episodes of depression (e.g., Kendler, Karkowski, & Prescott, 1999) and that negative life events predict depressive symptoms specifically in adolescence (Grant et al., 2003; Grant, Compas, Thurm, McMahon, & Gipson, 2004; Tram & Cole, 2000). First

episodes of depression—which are particularly relevant to the emergence of depression in adolescence—are especially likely to be triggered by negative life events (Monroe & Harkness, 2005).

The evidence suggests that gender differences exist in the frequency and type of negative events during childhood and adolescence. A number of studies have found that girls report more negative events than boys do (e.g., Davies & Windle, 1997; Graber, Brooks-Gunn, & Warren, 1995), particularly in early adolescence (Compas, Davis, & Forsythe, 1985). M. Davis, Matthews, and Twamley (1999) conducted a meta-analysis of studies of gender differences in major and minor life events. They found, overall, that females were exposed to more stress than males were, by only a small amount ( $d = 0.12$ ). The gender difference in exposure was not significant for children or young adults but was significant for adolescents ( $d = 0.12$ ), with girls exposed to more negative events. Gender differences were larger for appraisals of the events than simply for occurrence of the events. For appraisals,  $d = 0.11$  for children, 0.29 for adolescents, 0.19 for young adults, and 0.18 for adults. The gender difference in appraisals was also larger for major life events ( $d = 0.37$  for adolescents) than for daily stresses ( $d = 0.22$  for adolescents). These findings indicate that, consistent with the ABC model, adolescence is a time of enlarged gender differences in exposure to negative life events, with girls having the greater exposure; moreover, gender differences in appraisals of stress are substantial in adolescence.

Can these relatively modest gender differences in exposure to negative life events explain the 2:1 gender ratio for depression in adolescence and its persistence throughout adulthood? First, the gender differences in appraisals are larger than the gender differences in events. When predicting depression, appraisals of the event are more important than simply whether the event occurred (Lazarus, Averill, & Option, 1970; Lazarus & Launier, 1978). This then raises the question of why gender differences in appraisals are larger than gender differences in events. One possible explanation comes from our vulnerability–stress interaction model. That is, girls who score higher on some aspect of vulnerability—for example, negative emotionality in temperament—may find negative events more aversive than other girls and than most boys and appraise them more negatively. Second, a recent, large daily diary study found evidence of more substantial gender differences in stressors in adolescence; for example, for interpersonal stressors  $d = 0.50$  (Hankin, Mermelstein, & Roesch, 2007), suggesting that retrospective reports of stressors may underestimate the gender difference. Regarding persistence of the gender difference into adulthood, if adolescent depression goes untreated, it may not remit and may continue for years, or it may remit and then recur. Therefore, untreated or insufficiently treated adolescent depression could itself maintain a gender difference throughout adulthood.

Among the most serious of negative life events is child sexual abuse. Girls are more than twice as likely as boys to report sexual abuse in childhood and adolescence, and the greatest increase in sexual abuse for female victims is in the early adolescent period (Costello, Erkanli, Fairbank, & Angold, 2002; Finkelhor, 1984; Laumann, Gagnon, Michael, & Michaels, 1994; Russell, 1984; Tolin & Foa, 2006). Cutler and Nolen-Hoeksema (1991), on the basis of a review of relevant studies, concluded that the incidence of child sexual abuse was between 7% and 19% for girls and between 3% and 7% for boys. A major national survey found that 17% of women and 12% of men had had sexual contact, as a child,

with a late adolescent or an adult (Laumann et al. 1994). In another sample, 30% of women reported child sexual abuse defined broadly to include behaviors such as sexual touching, and 8% reported child sexual abuse that involved intercourse (Kendler et al., 2000).

Some research indicates that different factors predict juvenile-onset compared with adult-onset depression (Jaffee et al., 2002). Specifically, in one sample of women, more severe child sexual abuse, involving actual or attempted intercourse, was associated with depression onset before age 16, whereas less severe child sexual abuse was associated with adult-onset depression (Hill, Pickles, Rollinson, Davies, & Byatt, 2004).

Kendler, Kuhn, and Prescott (2004), with a sample of female adult twins from the Virginia Twin Registry, found that both child sexual abuse and other stressful life events predicted the onset of major depression. Women with child sexual abuse and especially those exposed to severe child sexual abuse had both an overall increased rate of major depression and a substantially increased sensitivity to the depressogenic effects of negative life events. That is, early exposure to child sexual abuse made them more vulnerable to later negative life events. Weiss, Longhurst, and Mazure (1999) hypothesized a biological mechanism for this effect, in which the early severe stressor produces long-term dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis. This dysregulation makes the individual more vulnerable to later stressors in adolescence or adulthood. Weiss and colleagues also found some evidence that child sexual abuse was more likely to lead to depression in females than in males.

Research indicates that girls experience more interpersonal negative events, particularly with family and peers, than boys do (Crick, Casas, & Nelson, 2002; Larson & Ham, 1993; Stark, Spirito, Williams, & Guevremont, 1989). In research on high school students' reports of their main problems, boys report more problems with school, whereas girls report more problems with interpersonal relationships (Phelps & Jarvis, 1994; Stark et al., 1989). Cliques and adolescent peer networks become powerful beginning in early adolescence. Girls report more intimacy in their friendships than boys do, and girls are more likely to be part of a clique (Urberg, Degirmencioglu, Tolson, & Halliday-Scher, 1995), which can create both pleasure and pain, for example, if one is excluded. Boys, in contrast, have more diversity in their friendships, and disruptions may therefore be less distressing (Urberg et al., 1995). Following the match hypothesis (see below), girls may suffer a double hit insofar as they are most likely to experience negative events in precisely the domain in which they are most cognitively vulnerable, namely, the interpersonal domain. One study found that higher rates of depression in adolescent girls were explained by greater exposure to stress, particularly interpersonal stress (Shih, Eberhart, Hammen, & Brennan, 2006).

One particularly serious kind of negative interpersonal event occurs with peer sexual harassment. In a survey of high school students, 38% reported having been sexually harassed by peers before sixth grade (American Association of University Women [AAUW], 2001). The harassing behaviors reported in the AAUW (2001) study include spreading sexual rumors, calling someone gay or lesbian, or pulling at/off someone's clothing. The rate of peer harassment increases through middle school (McMaster, Connolly, Pepler, & Craig, 2002). Although roughly as many boys as girls report being victims, girls are more upset by the incidents

(AAUW, 2001; Murnen & Smolak, 2000). For example, in the AAUW study, girls were considerably more likely than boys to feel self-conscious (44% to 19%), embarrassed (53% to 32%) and less confident (32% to 16%) because of the incident. Peer sexual harassment clearly is a stressor that, by itself or in interaction with cognitive vulnerability or emotional reactivity, may contribute to depression and specifically to gender differences in depression. Girls who experience early puberty receive heightened exposure to PSHV; Tanner stage correlates positively with reported PSHV in both fifth and seventh grades (Petersen & Hyde, 2007).

In regard to family factors, marital discord and parental depression are important stressors for children and adolescents (Davies & Windle, 1997). Research indicates that girls are more vulnerable than boys to marital discord and maternal depression (Davies & Windle, 1997; Fergusson, Horwood, & Lynskey, 1995). Moreover, maternal depression and marital discord influence each other bidirectionally (Gotlib & Hooley, 1988), and marital discord may mediate or moderate (exacerbate) the impact of maternal depression on children (Downey & Coyne, 1990; Goodman, Brogan, Lynch, & Fielding, 1993; Goodman & Gotlib, 1999). Thus, the greater vulnerability of girls to these events may be magnified.

An important component of the cognitive vulnerability–stress model is the match hypothesis, which holds that individuals may have different cognitive styles for different domains of negative events (Abramson et al., 1989; Beck, 1967, 1987) and that a match between cognitive vulnerability in a particular domain and a negative event in that domain increases the likelihood of the development of depressive symptoms. Several studies have demonstrated that individuals with cognitive vulnerability to interpersonal events are in fact particularly likely to become depressed following negative interpersonal events, in comparison to negative achievement events (reviewed by Coyne & Whiffen, 1995). Moreover, gender differences in cognitive vulnerability may vary by domain. Relatively little research has tested this hypothesis and its implications for the gender difference in depression. Our study with college students found that gender differences in rumination were small in the achievement domain ( $d = 0.20$ ) but moderate to large ( $d = 0.50$  to  $0.70$ ) in the interpersonal and the body image/attractiveness domains (Mezulis et al., 2002).

In summary, girls overall experience somewhat more negative life events than boys do (with a larger gender gap in appraisals) and are considerably more likely to experience child sexual abuse. Girls also experience more interpersonal negative events. Early-puberty girls are particularly likely to experience peer sexual harassment.

### Conclusion

We have proposed an affective, biological, cognitive model for the emergence of gender differences in depression in adolescence. The model, in brief, holds that cognitive vulnerability to depression involves negative cognitive style, rumination, and OBC. Cognitive vulnerability interacts with negative life events—particularly events in the domain of cognitive vulnerability (the match hypothesis)—to increase symptoms of depression or trigger a diagnosable episode of depression. Affect is an integrated component of the model, which posits that early temperament qualities of high negative emotionality are themselves vulnerabilities and, in addition, contribute, developmentally, to negative cognitive style.

Three biological components have been proposed: genetic influences, the hormones involved in puberty (DHEA, estrogen, and testosterone), and pubertal timing. Finally, social factors are integral to the model insofar as the negative life events that interact with the vulnerabilities often come in a social, interpersonal form—whether peer sexual harassment, parents' marital discord, or a troubled relationship with parents.

We have presented empirical evidence supporting many components of the model. Negative cognitive style, when paired with stressors, prospectively predicts depression. Gender differences in negative cognitive style are not present in childhood but emerge in adolescence. OBC—and particularly self-surveillance—is an additional potent cognitive vulnerability that contributes to the gender difference in depression and in all likelihood is affected by gender intensification occurring at the beginning of adolescence, as well as by peer sexual harassment. Girls and women are somewhat more likely to be exposed to negative life events and, especially in adolescence, have considerably more negative appraisals of the events. Child sexual abuse is a particularly destructive negative event that is more likely to be experienced by girls than boys during childhood and early adolescence. Peer sexual harassment, which intensifies in early adolescence, is an additional stressor. The hypothesis is relatively new that temperament—high negative affectivity—is a vulnerability to the formation of negative cognitive style. Therefore, evidence is less abundant, but the existing data support the hypothesis.

The evidence on the role of puberty and pubertal hormones in adolescent depression is complex. In regard to puberty, a number of possibilities exist; the evidence indicates that early timing of puberty for girls confers vulnerability to depression. DHEA, estradiol, and testosterone may all play a role in depression. It is unlikely that the effects are simple, linear ones. Rather, research suggests curvilinear effects and hormone levels interacting with stressors. Interactions between genetic vulnerability and hormones are likely.

### Multiple Pathways

A theory that proposes a single pathway for the development of depression, or for girls' accelerating rates of depression in adolescence, is doomed to be contradicted with data. It seems clear that there are multiple pathways to adolescent girls' depression (e.g., Harrington, Rutter, & Fombonne, 1996). An important concept from developmental psychopathology applies here, *equipotentiality*, which is the notion that subgroups of people who have the same disturbance arrive there from different origins (Cicchetti & Rogosch, 1996). In the current context, different individuals with the same outcome—adolescent depression—may arrive there through more than one pathway. The ABC model, we believe, provides the structure to accommodate these individual differences and multiple pathways.

As an example, one girl may be the victim of child sexual abuse in middle childhood or in the transition to adolescence. That negative life event or series of events, by itself, may be sufficient to cause depression in adolescence. The pathway may be that the child sexual abuse leads to a very negative cognitive style, which confers a substantial vulnerability to depression. Peer sexual harassment could then easily serve as the trigger to depression. Alternatively, the pathway might involve dysregulated HPA axis

functioning created by the child sexual abuse, which makes her highly emotionally reactive to even the moderate stressors of adolescence, such as a change in schools.

As a second example, a girl may, from infancy, be high on the temperament dimension of negative emotionality. Over years, exposure to normal stressors either evokes a more negative emotional response from her or she simply notices more negative events in her environment. These cumulate over time to create a negative cognitive style at the beginning of adolescence that, combined with negative events such as peer harassment, lead to depression.

As a third example, by age 11 or 12, a girl may have developed a high level of OBC—in particular, self-surveillance—because she has grown up in a family that places strong emphasis on appearance, because of overexposure to mass media emphasizing the thin ideal, or perhaps because of early pubertal development. Her cognitive vulnerability, combined with a stressor such as appearance-related teasing by peers, then leads to depression, perhaps comorbid with an eating disorder.

In a fourth example, a girl is in the middle of the pubertal transition, her estradiol levels are rising rapidly, and her endocrine homeostasis is temporarily disrupted. Neurotransmitter homeostasis is therefore also disrupted, and she is biologically vulnerable. If a negative life event, such as parental divorce, occurs at that time, depression is triggered.

One implication of this principle of multiple pathways is that, in empirical tests of this model, any single pathway should yield significant results but that the effect will not be large because only a subset of girls follow that pathway to depression.

Although the explicit focus of this review has been gender differences in depression, an implicit focus is within-gender individual differences. That is, why do some adolescent girls become depressed, whereas others do not? The vulnerability factors that we emphasized—for example, genetic vulnerability, pubertal timing, temperament, and negative cognitive style—all represent dimensions of individual differences and therefore should explain within-gender variability in the appearance of depression, as well as gender differences. They should account, as well, for the pathways to the absence of depression and to healthy functioning.

### Future Directions

We believe that, in addition to providing a comprehensive, integrated structure for the understanding of current research findings, our model identifies crucial lacunas in the literature and points to important new directions for research.

Several pathways in the model are well researched and may need little further investigation. For example, many aspects of the cognitive vulnerability–stress pathway have been well established. Perhaps the best example is the wealth of research examining the interaction of negative cognitive style with stress to predict depression in adolescents, as well as to partially mediate the gender difference. There is compelling evidence regarding the contribution of gender differences in rumination and OBC to the gender difference in depression. However, other aspects of the cognitive vulnerability–stress model are less well developed. These include interactive pathways that have not been examined much to date; for example, (a) does a ruminative response style interact with stress to predict depression, and is this interaction more potent for girls than boys, or (b) does OBC interact with stress to predict

depression, and is this interaction more potent for girls than boys? Several important developmental predictions based upon the model are also ready for testing. We know relatively little yet about the development of these cognitive vulnerabilities, such as at what ages they become stable, what factors contribute to them, and at what ages gender differences appear. These are important lines for future research.

In many ways, the most exciting future directions derive from the integrative nature of our model, which generates many testable hypotheses. One area is the interplay between affective vulnerability and cognitive vulnerability. What is the best model to integrate the two? Developmentally, does affective vulnerability, present from infancy, precede and predict cognitive vulnerability (e.g., Mezulis, 2004; Mezulis et al., 2006)? Do affective vulnerability and cognitive vulnerability affect each other reciprocally? When does OBC—both self-surveillance and body shame—appear in childhood, and does it precede and predict rumination? Our data indicate that the gender difference in self-surveillance is present by age 11 (Lindberg et al., 2006), but is it present even earlier? Does temperamental vulnerability contribute to self-surveillance or body shame?

The integration of biological and affective vulnerabilities is another exciting avenue for future research and generates additional testable hypotheses. The list of genes that are associated with depression has expanded rapidly; we hope for a comprehensive list in the near future. An important task will be to identify those genes that contribute to the gender difference in depression and those that do not, to determine whether gene–gene interactions occur, and then to trace the pathway from genes to girls' increasing depression in adolescence, both through biological mechanisms such as amygdala reactivity and through affective mechanisms such as temperament.

The data on pubertal hormones and adolescent depression are scattered and ambiguous. Research is needed to sort out these potential effects, testing all possible models, including nonlinear effects, interactions of hormone levels with stressors, and interactions of hormones and genes as one might expect if hormone-induced gene expression is involved.

We believe that only by applying an integrated model combined with multiple levels of analysis will the puzzle of the gender difference in depression in adolescence be solved.

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