

# Development of Gender Differences in Depression: An Elaborated Cognitive Vulnerability-Transactional Stress Theory

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Descriptive epidemiological studies are reviewed, showing that the female preponderance in depression begins to emerge around age 13. A developmentally sensitive, elaborated cognitive vulnerability-transactional stress model of depression is proposed to explain the “big fact” of the emergence of the gender difference in depression. The elaborated causal chain posits that negative events contribute to initial elevations of general negative affect. Generic cognitive vulnerability factors then moderate the likelihood that the initial negative affect will progress to full-blown depression. Increases in depression can lead transactionally to more self-generated dependent negative life events and thus begin the causal chain again. Evidence is reviewed providing preliminary support for the model as an explanation for the development of the gender difference in depression during adolescence.

Many decades of research on depression have produced several big, undisputed facts that any powerful theory of depression should be able to explain (Lewinsohn, Hoberman, Teri, & Hautzinger, 1985). We focus on two of these facts. First, the rates of depression rise dramatically during adolescence. Second, starting in early adolescence, more girls than boys begin to become depressed, and this gender difference in depression persists throughout adulthood (Nolen-Hoeksema, 1990) across many countries and cultures (Weissman et al., 1996). Although the timeline of the emergence of gender differences in depressive symptoms and disorders has been delineated fairly clearly, the mechanisms by which girls become more depressed than boys during adolescence are not understood well.

Many previous articles have examined various vulnerability factors to explain the emergence of the gender difference in depression (see Nolen-Hoeksema & Girgus, 1994; Hankin & Abramson, 1999, for reviews of this work). We do not comprehensively review the descriptive and etiological studies investigating why more adolescent girls than boys are depressed. Instead, we illustrate how an elaboration of an existing depression theory can account for such big facts as the general surge of depression during adolescence and the specific emergence of more girls than boys becoming depressed during this period. Given the important scientific goal of having general theories to explain disparate research findings, it is puzzling that previous theorists have not explored

whether current depression theories can explain the big fact of the emergence of gender differences in depression during adolescence. In contrast to this prior work, we elaborate extant, established depression theories to explain how more girls than boys start becoming depressed in adolescence. Using a bootstrap procedure, we integrate and expand on central findings from basic research in depression and gender socialization to articulate an elaborated cognitive vulnerability-transactional stress theory of depression that can explain the fact of the development of gender differences in depression.

Our goals in presenting this expanded theory are to (a) integrate findings from disparate areas of depression research (e.g., cognitive, interpersonal, genetic, etc.), (b) offer a model capable of explaining depression vulnerability over the lifecourse that is consistent with existing theories of depression, and (c) explicate particular mechanisms contributing to the emergence of the gender difference in depression. In so doing, we illustrate a general strategy for determining whether existing theories can (or cannot) elucidate known group differences in depression (e.g., boys vs. girls or Asian Americans vs. European Americans).

We begin by briefly reviewing the descriptive epidemiological studies to construct a general timeline for the overall development of depression and the emergence of the gender difference in depression, in particular. Then, we consider how our approach compares with prior work and how our general, integrative model may advance the study of depression, broadly, and elucidate the mechanisms contributing to the emergence of the gender difference in depression during adolescence, more specifically. Next, we present the model and specify the particular factors and processes that make up its causal chain. Following the model's presentation, we review pertinent studies for evidence that supports or contradicts the mechanisms articulated in our theory. This review is divided into two parts. First, we focus on studies that have examined factors relevant to the general depression model. Second, we review studies that have investigated gender differences to evalu-

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ate whether the proposed general model accounts for the specific case of the emergence of the gender difference in depression.

### Descriptive Timeline for the Development of Depression by Gender

Before reviewing how depression evolves over time by gender, it is important to define depression and consider particular conceptual issues that can influence the delineation of a general timeline of depression. These issues include (a) the definition of depressed mood and disorder, (b) when children can accurately report on depression, (c) possible gender differences in reporting depression, and (d) sample selection issues.

#### *Issues Concerning the Definition and Assessment of Depression*

Briefly, depressed mood refers to depression as an affective quality, such as feeling sad or unhappy, for an unspecified time period, but it does not signify whether other symptoms are present or absent. A depressive disorder is categorically defined and is composed of a list of different depressive symptoms (e.g., loss of sleep, changes in appetite, concentration problems) that must be present for a specified time period (e.g., at least 2 weeks) along with functional impairment. To date, there is little agreement in the field whether depressed mood is on a continuum of severity with depressive disorder or if a depressive disorder is fundamentally different from depressed mood (e.g., Coyne, 1994). We take the perspective that depressed mood and disorder are best viewed as varying along a continuum (Lewinsohn, Solomon, Seeley, & Zeiss, 2000; see Flett, Vredenburg, & Krames, 1997, for a review in adults) because depressed mood carries risk for development of depressive disorder (Harrington, Fudge, Rutter, Pickles, & Hill, 1990; Pine, Cohen, Cohen, & Brook, 1999; Weissman et al., 1999). Moreover, elevations of depressive mood and symptoms are not benign but rather are an area of concern. Moderate depressive symptoms are associated with academic and peer difficulties (Nolen-Hoeksema, Girgus, & Seligman, 1992; Petersen, Sarigiani, & Kennedy, 1991), overall poor psychosocial functioning (Gotlib, Lewinsohn, & Seeley, 1995), and may precede a depressive episode, although many with depressed mood will not develop a disorder (Pine, Cohen, Gurley, Brook, & Ma, 1998). We review studies examining both depressive mood and disorders and note what level of depression was investigated.

Children can report accurately on their own depressed mood and symptoms (Kazdin, 1994) and can recognize readily various different emotions (positive/negative valence and self/other perspectives) after age 9 (Harter, 1999). A related issue is whether children, or their parents or teachers, are the best reporters of their depressed mood and symptoms. Studies examining the concordance among children's self reports and parents' or teachers' reports of the child's depression have found moderate agreement (Kazdin, 1994). Children may be the best informants about their own affect after age 9 when they can recognize and identify different emotions. However, other informants, such as parents or teachers, may be better at describing other observable depressive symptoms, such as reduced energy level, changes in eating, and so forth.

Another important issue is the possibility that in reality, males and females do not differ in the prevalence rates of depression, but an apparent gender difference in depression is observed because females are more likely than males to report or discuss their depression. However, little evidence exists to support this reporting bias hypothesis (see Nolen-Hoeksema, 1990).

The sample of children being investigated can impact importantly the prevalence rates of depression and the accuracy of construction of a general timeline for depression development. In general, inpatient child clinical samples often show higher prevalence rates of depression, more severe symptoms, and increased comorbidity of depression with other psychiatric disorders (e.g., anxiety or behavior disorders) compared with unselected community samples (Caron & Rutter, 1991; Newman, Moffitt, Caspi, & Silva, 1998). For example, Angold and Rutter (1992) found that the gender difference in depressed mood began to emerge around age 10 in their large sample of child clinical inpatients, whereas other studies of unselected, community children and adolescents show a different developmental timeline of depression, as seen below. To delineate a clear picture of the development of depression, it is important to use large, unselected community samples to guard against potential biases from inpatient clinical samples.

#### *Timeline for the Emergence of the Gender Difference in Depression*

On the basis of these relevant conceptual and methodological considerations for descriptive epidemiological research, we selectively focus our review of descriptive depression studies to large, community samples to draw our general timeline for the development of depression overall and by gender. Overall, depressed mood is fairly common during adolescence, particularly among adolescent girls, with the likelihood of depressed mood ranging between 25–40% for girls and 20–35% for boys (Petersen et al., 1993). Prospective longitudinal studies (Cole, Martin, Peeke, Seroczynski, & Fier, 1999; Ge, Lorenz, Conger, Elder, & Simons, 1994; Petersen et al., 1991) investigating children from preadolescence to young adulthood found that girls' depressive symptoms and depressed mood increased after age 13, whereas boys' symptoms and mood remained constant. Increased depressed mood was also found among girls after age 13 in a large national cross-sectional study (Wichstrom, 1999). Another community cross-sectional study (Compas et al., 1997) found that more girls than boys displayed higher levels of a mixed anxiety-depressive syndrome, and this effect was especially detectable among referred adolescents.

Cross-sectional studies of children and adolescents provide evidence that more girls than boys are diagnosed with clinical depression after age 13 (Angold, Costello, & Worthman, 1998; Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993; Silberg et al., 1999). However, there are methodological and conceptual limitations with using only cross-sectional designs. Prospective studies can provide a more accurate descriptive picture of the emergence of gender differences in depression. Consistent with the cross-sectional studies of depressive disorders and the prospective studies of depressed mood, prospective community studies indicate that more girls than boys show clinical depression beginning after age 13 (Cohen, Cohen, Kasen, & Velez, 1993; Hankin et al., 1998; Reinherz, Giaconia, Lefkowitz, Pakiz, & Frost, 1993;

Weissman, Warner, Wickramaratne, Moreau, & Olfson, 1997). For example, one study (Hankin et al., 1998) showed that both boys and girls became increasingly more depressed between the ages of 15 and 18 (from 3% to 17%), and this increase was greater for girls (from 4% to 23%) than for boys (from 1% to 11%). Thus, these prospective community studies suggest that middle adolescence may represent a critical time for increased vulnerability to depressive mood and disorders, especially for girls.

However, chronological age may mask important developmental transitions (Rutter, 1989) that could more accurately pinpoint when the gender difference in depression emerges. Consistent with this hypothesis, pubertal development (measured by Tanner stages) predicted the emergence of the gender difference in depression better than age alone, as girls reported increased rates of depressive disorders after Tanner Stage III (Angold et al., 1998). Graber, Lewinsohn, Seeley, and Brooks-Gunn (1997) found higher prevalence rates of depression among female adolescents who passed through puberty early (30% rate) or late (34%), compared with girls passing through puberty on time (22%). A 4-year longitudinal study (Ge, Conger, & Elder, 1996) found that early-maturing girls experienced more symptoms of anxiety and depression and were more vulnerable to continuity in these emotional problems compared with on-time or later maturing girls.

Last, it is important to examine how ethnicity may play a role in the development of gender differences in depression. Two studies (Schraedley, Gotlib, & Hayward, 1999; Siegel, Aneshensel, Taub, Cantwell, & Driscoll, 1998) found that Hispanic adolescents reported the greatest level of depressed mood compared with Caucasians or African Americans. Neither study found significant interactions between ethnicity and gender in predicting depressed mood. Further, Hayward and colleagues (1999) found that post-menarcheal Caucasian girls reported greater depressed mood compared with same-aged premenarcheal girls. Moreover, this study found that menarche was associated with depressed mood only among Caucasian girls but not among Hispanic or African American girls. Siegel et al. (1998) found that early maturing girls of all ethnicities reported the highest depressed mood levels. Thus, these few studies highlight the need for further research on ethnicity and the development of the gender difference in depression.

#### *Disorders Comorbid With Depression*

Depressive disorders show substantial comorbidity with other psychiatric disorders, especially anxiety, externalizing, and eating disorders (Newman et al., 1996; see Angold, Costello, & Erkanli, 1999, for a review). Given such comorbidity patterns, it is important to consider whether gender differences emerge in these comorbid disorders according to the same developmental timeline as for depression or whether this timeline applies particularly to depression. Below, we review various studies examining the developmental patterns of depression comorbidity with other disorders that indicate that the descriptive timeline for the emergence of the gender difference in depression applies most specifically to depression and not to all psychopathological disorders.

The timeline for the emergence of the gender difference in anxiety shows that girls develop anxiety disorders earlier and at a faster rate than boys, such that by age 6, twice as many girls have experienced an anxiety disorder (Lewinsohn, Gotlib, Lewinsohn, Seeley, & Allen, 1998). Prospective community studies (Cohen et

al., 1993; Cole et al., 1999; Pine et al., 1998; Reinherz et al., 1993; see review by Kovacs & Devlin, 1998) show that more girls than boys have an anxiety disorder, and the anxiety disorder often precedes the onset of a depressive disorder. Thus, existing evidence indicates that more girls than boys develop anxiety symptoms and disorders, and this gender difference emerges earlier than for depression.

More boys than girls show overt aggressive, externalizing behaviors that increase or stay stable from kindergarten through 7th grade (Keiley, Bates, Dodge, & Pettit, 2000; Loeber & Keenan, 1994). However, there may not be a gender difference in aggression when relational and physical aggression are considered together (Crick et al., 1999). Starting in preschool, girls show more aggression than boys within interpersonal relationships (Crick & Grotpeter, 1995; Crick et al., 1999). Such relational aggression has been linked to greater peer rejection, loneliness, and depression (Crick & Grotpeter, 1995), perhaps especially for girls (Rys & Bear, 1997). Thus, the timeline for the emergence of gender differences in aggressive behaviors also differs from the one for depression as girls show more relational aggression than boys early in childhood, and boys exhibit more physical aggression than girls.

More adolescent girls than boys have been diagnosed with lifetime histories of eating disorders (Lewinsohn et al., 1993; Steiner & Lock, 1997). A 4-year longitudinal study of community adolescent girls found the peak risk for the onset of binge eating to occur at age 16 and the peak onset for purging to occur at age 18 (Stice, Killen, Hayward, & Taylor, 1998).

#### *Conclusion*

In summary, the descriptive picture painted by the prevalence and comorbidity studies suggests several findings requiring theoretical explanation. First, more girls than boys start becoming depressed around age 13. Second, the descriptive timeline for depression indicates that both boys and girls experience a rapid increase in depression throughout adolescence. Third, the comorbidity studies suggest that this pattern applies most specifically to depression and does not apply to all psychopathology. Fourth, girls are more anxious and have more anxiety and eating disorders than boys. Anxiety often precedes depression and may confer additional risk for later depression. Fifth, girls are more relationally aggressive than boys, and relational aggression is associated with depression. We now turn to previous articles that have offered explanations of the emergence of the gender difference in depression.

#### *Comparison to Prior Work*

Prior influential articles in this area include Nolen-Hoeksema and Girgus's (1994) review and Cyranowski, Frank, Young, and Shear's (2000) specific model intended to account for why more adolescent girls than boys are depressed. We discuss each article and then address how the current integrative model builds on and advances beyond these prior articles.

#### *Nolen-Hoeksema and Girgus's (1994) Review*

Nolen-Hoeksema and Girgus's (1994) review of the emergence of gender differences in adolescent depression made several im-

portant contributions. First, Nolen-Hoeksema and Girgus reviewed studies from diverse literatures, including biological, personality, and social theories, aimed at explaining why more girls are depressed than boys starting in early adolescence. Second, they advocated moving beyond single factor explanations of this gender difference in favor of multivariate, integrated models. However, a limitation of their article was that Nolen-Hoeksema and Girgus (1994) did not explicitly articulate a theory with specific factors and mechanisms to explain the emergence of the gender difference in depression. Instead, they organized the research findings into three possible heuristic frameworks. The first framework was a general mediational model in which the etiological factors contributing to depression are identical for boys and girls, but these factors increase among early adolescent girls more than among boys. The second, moderational, framework suggested that the causes of depression differ for boys versus girls, and the different causes for early adolescent girls explain the gender difference in depression. The third framework consisted of a general vulnerability-stress model in which girls have more risk factors than boys prior to the emergence of the gender difference in depression, and these elevated risk factors interact with social challenges that occur during early adolescence.

Although these three heuristic frameworks effectively organized the existing research findings, they are limited in that they do not form a cohesive theoretical model that clearly suggests specific, testable and novel hypotheses. Moreover, it is unclear how Nolen-Hoeksema and Girgus's (1994) preferred heuristic framework (Model 3) relates to existing theories of depression. To advance understanding of factors and mechanisms conferring vulnerability to developing depression generally, and the emergence of the gender difference in depression specifically, a cohesive general theoretical model of depression is needed. The emergence of gender differences in depression should be derivable from such a general model once each gender's values on the theory's causal variables are known. In other words, an explanation of gender differences should be a special case of the general theory. In their broad vulnerability-stress framework, Nolen-Hoeksema and Girgus (1994) suggested that a developmental rise in social challenges throughout adolescence interacts with preexisting vulnerability factors, but they did not specify what particular kinds of social challenges increase over time, with which vulnerabilities these challenges interact, or the mechanisms through which this interaction operates. Reflecting the fact that their broad vulnerability-stress framework does not constitute a theoretical model with testable hypotheses, we were unable to locate any empirical studies testing their vulnerability-stress framework. Thus, a more specifically articulated theoretical model, building on Nolen-Hoeksema and Girgus' (1994) general vulnerability-stress framework, that explicates the particular social challenges, vulnerability factors, and mechanisms by which these interact, is needed to advance further understanding of how depression develops and how more girls than boys become depressed.

#### *Cyranowski et al.'s (2000) Theoretical Model*

On the other hand, Cyranowski et al. (2000) presented a theoretical model that specifically addressed why more girls than boys become depressed in early adolescence. Their vulnerability-stress model focused on an interpersonal, affiliative need that places

adolescent girls at particular risk for interpersonal negative events. An important strength of their model is the identification of important motivational strivings—affiliative needs in their model—that can importantly affect depression vulnerability. Although this model may specifically account for the higher rates of depression among adolescent girls, it is limited in that it is specific to adolescent girls and cannot easily address processes contributing to depression vulnerability outside adolescence and/or among boys. Also, Cyranowski et al.'s theory focuses particular attention on the interpersonal aspects of depression vulnerability but does not explicitly consider or integrate other known risk factors for depression, such as cognitive vulnerability or genetic risk. A powerful theoretical model to explain the development of gender differences in depression should not ignore current, empirically supported general depression theories unless these existing theories have been shown to have limitations that preclude explanation of the emergence of gender differences in depression.

#### *Need for Integrated Theory*

Thus, despite the significant contributions of Nolen-Hoeksema and Girgus (1994) and Cyranowski et al. (2000), there is still a need for an integrated, general theory that explicitly proposes particular factors and mechanisms to account for the general development of depression and the specific emergence of the gender difference in depression. As we have argued, before scrapping current empirically supported theories, it makes sense to integrate and elaborate on existing general depression theories to explain the development of gender differences in depression. An important scientific goal is to determine whether an existing general theory, or an elaboration of it, can explain critical specific cases (e.g., why more adolescent girls than boys become depressed). Thus, it is important to take the next logical step and provide such a general theoretical depression model based on current theory and basic research findings that can specifically account for the emergence of gender differences in depression during adolescence. There are certain advantages that a general depression model has compared with either a broad, nonspecific explanatory framework (e.g., Nolen-Hoeksema & Girgus, 1994) or a developmentally and gender-specific model (e.g., Cyranowski et al., 2000). A general theory cuts across developmental levels, connects to other known facts and theories, integrates and subsumes disparate findings and hypotheses into a consolidated model, and explains individual and between group differences through mechanisms specified in the model. We now turn to our integrative cognitive vulnerability-transactional stress theory elaborated from existing general depression models.

#### *Generic Cognitive Vulnerability-Stress Model*

We start by first briefly describing a generic cognitive vulnerability-stress model based on current cognitive theories of depression (e.g., Beck, 1987; Abramson, Metalsky, & Alloy, 1989). Both Beck (1987) and Abramson and colleagues (1989) have articulated depression theories that are conceptually similar enough to be considered together as a generic cognitive vulnerability-stress model, which is illustrated in Figure 1.

The essence of the cognitive vulnerability-stress model is that an individual with cognitive vulnerability is more likely to become

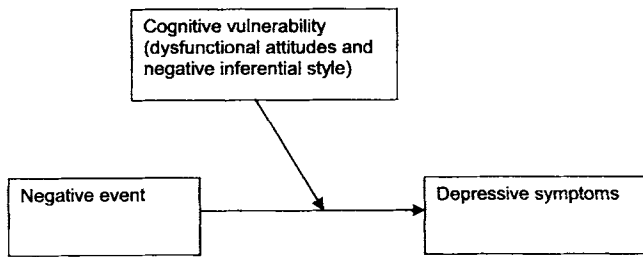


Figure 1. Schematic diagram of the generic cognitive vulnerability-stress model of depression.

depressed than a nonvulnerable individual when she or he confronts a negative event and interprets the event in a negatively biased manner (dysfunctional attitudes; Beck, 1987) and/or makes negative inferences about the cause, consequences, or self-implications of the event (negative inferential style; Abramson et al., 1989). An individual with dysfunctional attitudes may think his or her self worth hinges on being perfect or others' approval. For example, the dysfunctional attitude, "I'm no good unless people approve of me" may be activated if a coworker does not say hello. A person with a negative inferential style who fails an exam might attribute the poor test grade to stable and global causes (e.g., "I've always been stupid and I'm stupid in all academic areas") and infer that she will never get a job and is worthless. Typical methods of measuring cognitive vulnerability include self-report questionnaires and information-processing procedures (see Abramson et al., in press; Ingram, Miranda, & Segal, 1998).

This generic cognitive vulnerability-stress model has garnered considerable empirical support for predicting prospective changes in depression among children and adolescents (Hankin, Abramson, & Siler, 2001; Nolen-Hoeksema et al., 1992; Panak & Garber, 1992; Robinson, Garber, & Hilsman, 1995) and adults (see Abramson et al., in press; Abramson et al., 1999; Ingram et al., 1998; for reviews).

The current generic cognitive vulnerability-stress model without additions appears capable of partially accounting for the development of the gender difference in depression. For example, as is

reviewed later, prospective research has found a developmental rise in the number of negative life events occurring after age 13 for both boys and girls, but especially for adolescent girls (Ge et al., 1994). On the basis of this finding, the existing generic cognitive vulnerability-stress model would predict elevations in depression, particularly for girls, after age 13 as the number of negative events occurring to an individual increases.

### General Elaborated Cognitive Vulnerability-Transactional Stress Depression Model

However, a more powerful and complete explanation for how individuals in general, and adolescent girls in particular, become depressed can be achieved by elaborating on the existing, empirically supported generic cognitive vulnerability-stress theory in Figure 1. This generic cognitive vulnerability-stress model forms the backbone to which we add particular expansions to articulate an updated general depression model. The power of this elaborated, general cognitive vulnerability-transactional stress theory is that it can be used to predict and explain the development of depression, generally, and particular factors can be derived from the general model to account for the emergence of the gender difference in depression, more specifically. In Figure 2, we present our general elaborated cognitive vulnerability-transactional stress model.

#### Reasons for Elaborated Theory

We believe that revising and expanding on this basic cognitive vulnerability-stress framework makes sense at this time for four main reasons. First, basic research in psychopathology (e.g., Metalsky, Joiner, Hardin, & Abramson, 1993) and emotion (e.g., Watson, 2000) suggests the need to incorporate initial negative affect as a component in the elaborated causal chain leading to depression. Second, as cognitive approaches to depression have advanced theoretically and empirically over the past decade, we believe it is time to update and clarify how cognitive vulnerability is conceptualized and operates within a depressogenic causal chain. Third, given the further theoretical sophistication of and

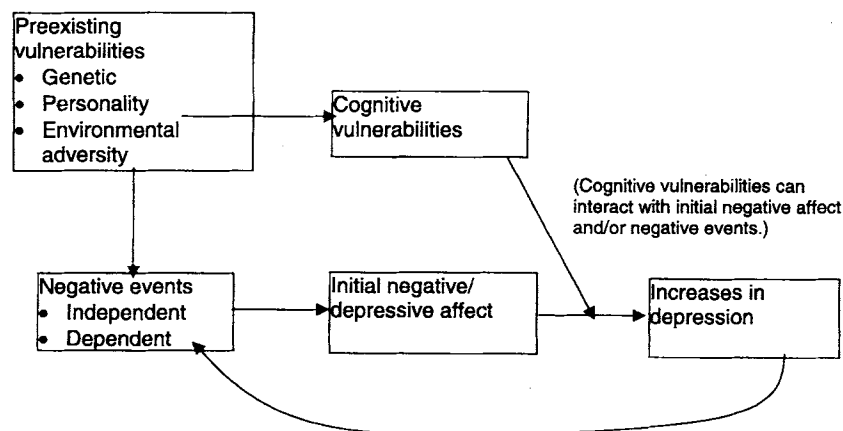


Figure 2. Schematic diagram of the proposed, general elaborated cognitive vulnerability-transactional stress model of depression.

empirical support for the interpersonal approach to depression (see Joiner & Coyne, 1999), it is important to adopt an interdisciplinary approach and integrate key aspects of the interpersonal theories into the cognitive vulnerability-stress model. Last, as our descriptive timeline of depression review illustrated, the dramatic surge in depression from childhood through adolescence suggests the need to have a developmentally sensitive model consistent with a developmental psychopathological perspective.

### *Initial Negative Affect*

As can be seen in Figure 2, the elaborated causal chain begins with the occurrence of a negative event (either independent or dependent; see below). A negative event contributes to elevations in initial levels of negative affect. Within the construct of negative affect, we include various negative emotions, including anxious, depressive, and angry affect. Measurement of negative affect can be accomplished through multiple methods, including self-report questionnaires, interview, observation, and psychophysiological assessment (see Ekman & Davidson, 1994; Lewis & Haviland-Jones, 2000; Watson, 2000). Supporting the first step in the causal chain, prospective studies have found that negative life events predict later increases in depression, even after controlling for negative/depressive affect at the start of the investigation (e.g., Compas, Howell, Phares, Williams, & Giunta, 1989; Hammen, 1988; Hammen, Burge, & Adrian, 1991; Hankin et al., 2001; Panak & Garber, 1992; Robinson et al., 1995; Stanger, McConaughy, & Achenbach, 1992; Windle, 1992).

As the initial negative affect increases, and if it remains, it can lead to increases in depressive symptoms, such as difficulty sleeping and poor concentration. Thus, whereas the existing generic cognitive vulnerability-stress model states that a negative event contributes to depressive symptoms (especially when moderated by cognitive vulnerability), we posit that initial negative affect comes between the occurrence of a negative event and later increases in depressive symptoms. We include this intermediary step of initial negative affect in the causal chain for three related reasons.

First, research examining the temporal relationship between negative events and the rise in depressive symptoms has found that initial elevations of nonspecific negative affect follow the occurrence of a negative event. Numerous cross-sectional and prospective studies indicate that negative events contribute to broad negative affect and depressive symptoms (see Compas, Grant, & Ey, 1994, for a review of the stress and depression literature in children and adolescents). Studies that prospectively assessed changes in negative affect after a naturally occurring stressor (e.g., academic exam, earthquake) have found that negative affect rises initially for most people after the negative event occurs, but this initial increase in negative affect persists only among cognitively vulnerable individuals (Metalsky, Halberstadt, & Abramson, 1987; Metalsky et al., 1993; Nolen-Hoeksema & Morrow, 1991; Ralph & Mineka, 1998). Consistent with our revision that the initial emotional response to negative events is broad negative affect, research investigating affective specificity by assessing depression along with other forms of negative affect has found that negative events are a nonspecific risk factor for general negative affect and a broad range of psychological symptoms, including anxiety (Cohen, Burt, & Bjorck, 1987; Compas et al., 1989; Lewinsohn, Seeley, &

Gotlib, 1997; Ralph & Mineka, 1998; Wagner & Compas, 1990; Watson, 2000) and externalizing behaviors (Hankin & Abramson, 2001; Lewinsohn et al., 1997; Robinson et al., 1995; Stanger et al., 1992; Windle, 1992).

The second, related consideration for including initial negative affect into the causal chain is to clarify the conundrum of comorbidity commonly observed with depression. As reviewed earlier, depression typically co-occurs with other psychological disorders, especially anxiety and behavioral disorders. Broad negative affect has been identified as the central common factor underlying the association between depression and co-occurring anxiety and externalizing symptoms (Brady & Kendall, 1992; Brown, Chorpita, & Barlow, 1998; Chorpita, Albano, & Barlow, 1998; Clark & Watson, 1991; Cole, Truglio, & Peeke, 1997; del Barrio, Moreno-Rosset, Lopez-Martinez, & Olmedo, 1997; Goodyer, Ashby, Altham, Vize, & Cooper, 1993; Joiner, Catanzaro, & Laurent, 1996; Krueger, Caspi, Moffitt, Silva, & McGee, 1996; Mineka, Watson, & Clark, 1998). By including initial elevations of general negative affect as a step in the causal chain, the elaborated model reflects the reality that depression commonly co-occurs with other negative emotions and psychopathological disorders. In this elaborated theory, then, depression comorbidity is not a problem because we explicitly posit that initial negative affect, common to depression and other disorders, results from the occurrence of a negative event. As is discussed, depression-specific cognitive vulnerability factors are included in the model to explain the development of depression specifically, as opposed to psychopathology in general.

Finally, our inclusion of a stage of initial general negative affect immediately following a negative event is consistent with work on the temporal dynamics of attribution and emotion. Specifically, Weiner (1985) has emphasized that emotional experience following an event is best conceived as a temporal sequence involving cognitions of increasing complexity that successively refine and differentiate emotional response. According to this view, once people perceive that an event has occurred, they initially may experience a primitive emotional response. These primitive emotions, which include happy for success and frustrated or sad for failure, are termed *outcome-dependent, attribution-independent* by Weiner because they are determined by the attainment or nonattainment of a desired goal and not by the causal attribution for the outcome. Following this immediate emotional reaction, a causal attribution will be sought (particularly if the outcome is negative, unexpected, or important), and a more differentiated set of emotions then will be generated by the chosen attribution (attribution-dependent emotions). Below, we describe how people exhibiting cognitive vulnerability select causal attributions (as well as other inferences) that ultimately lead to depression following the initial stage of general negative affect in response to a negative event. In other words, cognitive factors play a role in affect regulation by moderating an individual's initial response and maintenance/recovery from negative affect resulting from a negative event (see Davidson, 2000; Davidson, Jackson, & Kalin, 2000; for affect regulation models based on affective neuroscience).

### *Expanded Conceptualization of Cognitive Vulnerability to Depression*

The next step in the elaborated causal chain involves cognitive vulnerability factors moderating the likelihood that an individual

ultimately will experience increases in depression. Cognitive factors are hypothesized to interact with the occurrence of a negative life event to lead to a greater probability of experiencing eventual depression (as described by the generic cognitive vulnerability-stress section). For example, according to the hopelessness theory of depression (Abramson et al., 1989), individuals who exhibit a style to attribute negative events to stable, global causes are more likely to become hopeless, and in turn, depressed than individuals who do not. We also hypothesize that cognitive factors interact with the initial negative affect to amplify the affect, which can then contribute to increases in depressive symptoms.<sup>1</sup>

An important aspect of the cognitive vulnerability factors posited in the elaborated causal chain is that they are hypothesized to be more depression-specific than some other depression vulnerabilities. Supporting the specificity hypothesis, research with adolescents (Gladstone, Kaslow, Seeley, & Lewinsohn, 1997; Lewinsohn et al., 1997), examining risk factors for depression and other psychopathologies found that dysfunctional attitudes and negative attributional style were more strongly associated with clinical depression than with nondepressive disorders (e.g., anxiety and externalizing disorders). Also consistent with the hypothesized depression-specific vulnerability, Weiss, Susser, and Catron (1998) found that particular aspects of cognitive vulnerability (e.g., global and specific attributions) specifically predicted depression compared with comorbid anxiety and externalizing disorders among children. However, the specificity associated with depression among adults is less clear, as some studies have found that cognitive vulnerability predicts prospective increases in depression compared with anxiety (Hankin, Abramson, & Angeli, 1999; Metalsky & Joiner, 1992), whereas others have not (Ralph & Mineka, 1998). Last, cognitive vulnerability-stress studies with adolescents have found that the interaction between negative inferential style and negative events predicted depressive symptoms specifically, as opposed to externalizing problems (Hankin & Abramson, 2001; Robinson et al., 1995). Thus, these studies generally support the hypothesis that cognitive vulnerability more consistently and more strongly predicts depression compared with other comorbid conditions.

In contrast to the current generic cognitive vulnerability-stress model, the elaborated theory incorporates three particular revisions relevant to cognitive vulnerability. First, we include a ruminative response style (Nolen-Hoeksema, 1991), in addition to Beck's (1987) dysfunctional attitudes and Abramson et al.'s (1989) negative inferential style, as generic cognitive vulnerability factors. Second, we expand the points in the causal chain where these basic cognitive vulnerabilities can influence the likelihood of becoming depressed. Last, we emphasize the importance of assessing particular domains of cognitive vulnerability at different points in the lifespan, particularly dissatisfaction with body image and physical appearance among adolescents.

### *Ruminative Response Style*

Rumination is incorporated as a cognitive vulnerability factor within the elaborated causal chain because theoretical and empirical work suggests its importance in understanding depression generally and the gender difference in depression more specifically. Nolen-Hoeksema (1991) defined *rumination* as a stable, emotion-focused coping style that involves directing attention in-

wardly toward negative feelings and thoughts. The process of rumination maintains or exacerbates depression by augmenting accessibility and recall of negative events (Bower, 1981) and focusing attention on negative aspects of the self (Pyszczynski & Greenberg, 1987). Negative inferential styles and dysfunctional attitudes provide negative cognitive content, whereas rumination facilitates accessibility to or activation of this negative content. As the elaborated cognitive vulnerability-transactional stress model now explicitly includes elevations of initial negative affect, which includes depressed mood, we incorporate rumination as a general cognitive process that can lead to later increases in depressive symptoms after an individual begins experiencing initial negative affect. Similar to dysfunctional attitudes and negative inferential style, there are individual differences in the propensity to ruminate (ruminative response style) in response to a negative event or negative affect. Studies of adolescents (Broderick, 1998; Schwartz & Koenig, 1996) and adults (Butler & Nolen-Hoeksema, 1994; Just & Alloy, 1997; Morrow & Nolen-Hoeksema, 1990; Nolen-Hoeksema & Morrow, 1991, 1993; Nolen-Hoeksema, Morrow, & Fredrickson, 1993; Roberts, Gilboa, & Gotlib, 1998; Trask & Sigmon, 1999) support the hypothesis that rumination maintains or worsens depression.

### *Expanded Points in the Causal Chain*

With the inclusion of a ruminative response style as a cognitive vulnerability factor and initial negative affect as a link in the elaborated causal chain, the points in the causal chain where cognitive vulnerability can moderate the potential for depression are expanded. Moving beyond the existing generic cognitive vulnerability-stress model, the elaborated model posits that cognitive vulnerabilities (e.g., dysfunctional attitudes and negative inferential style) can interact with both negative events and elevations in initial negative affect to eventuate in increases in depression. Prospective research, such as the academic midterm studies (e.g., Metalsky et al., 1993; Robinson et al., 1995) have found that most individuals experience immediate elevations of negative affect (depressed mood) after a negative event (receiving a low exam grade), whereas only individuals with negative inferential styles continued to exhibit increases in depression several days later. Other prospective research (Rusting & Larsen, 1998) using experience sampling methods found that adults were more likely to make negative attributions for negative events when they were experiencing general negative affect. Also, in the expanded model, it is possible for rumination to interact with a negative event as well as with initial elevations in negative affect (Alloy et al., 2000). Last, by including a ruminative response style along with dysfunctional attitudes and negative inferential style as generic cognitive vulnerabilities, it is possible for these cognitive risk factors to affect each other. A ruminative response style has been found both to interact with a negative inferential style to predict depression (Alloy et al., 2000) as well as to mediate the association between cognitive risk factors (e.g., negative inferential style) and depression (Spasojevic & Alloy, 2001).

<sup>1</sup> We note the possibility that if negative interpretational and/or inferential styles (two types of cognitive vulnerability) are highly automatized, they also may contribute to the occurrence and form of the initial affective response to a negative event.



### *Contextual Domains of Cognitive Vulnerability to Depression*

So far, the cognitive vulnerabilities of dysfunctional attitudes, negative inferential style, and rumination have been conceptualized at a general level. However, we believe that more precise prediction can be achieved by considering particular domains of cognitive vulnerability. Both Beck (1987) and Abramson et al. (1989) have formulated a *specific vulnerability hypothesis*. According to this hypothesis, depression is more likely to result when there is a match in the domain (e.g., interpersonal or achievement) of the negative event and of the cognitive vulnerability compared with a mismatch (i.e., cognitive vulnerability and negative events are in different domains). The studies that have explicitly tested the domain match hypothesis with adults are generally supportive (Metalsky et al., 1987; Metalsky et al., 1993; Spangler, Simons, Monroe, & Thase, 1997; see Coyne & Whiffen, 1995, for a critical review).

Taking a developmental perspective, we suggest that there may be systematic changes in people's motivational strivings over the course of the life span (see Ryff, 1985). In this regard, Erikson (1974) has emphasized that people confront different tasks at different stages of the life span. Whereas adolescents confront the task of establishing an identity, elderly individuals question whether their lives had meaning. This view suggests that the domains most relevant for cognitive vulnerability may change over the life span. For example, health may be a more relevant domain for elderly people than for children.

The domain of perceived physical attractiveness and body satisfaction may be very motivationally significant for adolescents. This domain may not map neatly onto either the achievement or interpersonal domains but instead may cut across these domains or form a completely separate domain of vulnerability and stressor. The following example illustrates how the domain of physical attractiveness and body satisfaction may function similarly to other motivationally significant domains such as achievement in the elaborated cognitive vulnerability-transactional stress theory. Suppose an adolescent girl who exhibits cognitive vulnerability in the domain of physical attractiveness and body satisfaction encounters a negative event in this same domain such as a classmate making belittling comments about her weight and hairstyle. Given her cognitive vulnerability in the domain, the girl is likely to attribute these comments to stable and global causes ("I will always be fat and ugly and people will continue saying my hair looks bad"), infer other negative consequences will follow from the event ("I will never go to prom or get married"), and infer negative self-concept implications from the event ("I am worthless").

Last, it is important to consider the magnitude of the negative event and level of cognitive vulnerability required to contribute to depression. The concept of *titration* is important in the cognitive vulnerability-stress model (Abramson, Alloy, & Hogan, 1997). The severity of a negative event can range from hassles (e.g., being called fat by peers) to major negative events (e.g., death of parent), and similarly, cognitive vulnerability can extend from mild to extreme levels. Thus, everyday hassles can lead to initial negative affect and depressive symptoms, particularly if an individual has higher levels of cognitive vulnerability in that particular domain. Finally, note that discrepancies (Higgins, 1987) between an ideal self (e.g., thin, intelligent) and actual self (e.g., heavy, unin-

telligent) may function as negative events in the causal chain featured in our elaborated cognitive vulnerability-transactional stress theory.

### *Interpersonal Stress Generation*

The third revision we have made to the existing cognitive vulnerability-stress theory is to include a transactional stress-generation mechanism as part of the elaborated causal chain based on theoretical and empirical work from the interpersonal theories of depression (Hammen, 1991, 1999; Joiner & Coyne, 1999). In contrast to the generic cognitive vulnerability-stress model, which tends to conceptualize and investigate the stress-depression relationship in a static, unidirectional manner, a more likely depiction is through a transactional process in which increases in depression can contribute to the creation of further dependent negative events. It is important to distinguish between independent (fateful) events, which befall people outside their control (e.g., death of parent), and dependent events, to which individuals partly contribute and generate through aspects of their behavior and personality (e.g., romantic break up, fight with friend). Negative events can be assessed by multiple methods, including self-reports, interview, observation, documentation of naturalistic events, and experimental procedures (see Monroe & Simons, 1991).

Following the causal chain, both independent and dependent negative events lead to elevations in initial negative affect and depression, and increases in depression can lead to more dependent negative life events as depressed individuals seek reassurance excessively and are rejected by others (Joiner & Coyne, 1999). The net result of this transactional process is the further creation of dependent, negative events, which can restart the process at the beginning of the causal chain and, thus, lead to increases in depression over time.

Existing research supports the transactional, bidirectional relationship between stress and depression. Studies with adolescents (Cohen et al., 1987; Compas et al., 1989; Windle, 1992) have found that elevations in depressive symptoms lead to later increases in the overall number of negative events, even after controlling for initial levels of negative events. More recent studies have distinguished between independent and dependent negative events. Research with adults (Cui & Valliant, 1997; Daley, Hammen, Burge, & Davila, 1997; Davila, Bradbury, Cohan, & Tochluk, 1997; Davila, Hammen, Burge, & Paley, 1995; Harkness, Monroe, Simons, & Thase, 1999; Potthoff, Holahan, & Joiner, 1995) and adolescents (Adrian & Hammen, 1993; Daley et al., 1997; Williamson, Birmaher, Anderson, Al-Shabbout, & Ryan, 1995) has found that depressed individuals experience more dependent than independent negative events. Individuals who generated more dependent, interpersonal negative life events experienced increases in depression (Kendler, Karkowski-Shuman, & Prescott, 1999), had earlier onsets of depression, had poorer social skills, exhibited features of personality disorders, and had parents with pathology (see Hammen, 1999, for a review).

In addition, we hypothesize that other psychopathological symptoms and disorders co-occurring with depression provide another interpersonal mechanism contributing to the creation of additional dependent, negative events. For example, aggression within close, interpersonal relationships (relational aggression) is linked to depression (Crick et al., 1999). Within our model's framework,



individuals with depression overlapping with other symptoms (e.g., depression and relational aggression) would likely be creating dependent, interpersonal negative events as they are rejected by peers or romantic partners for their aggressive behavior within close peer relationships. Supporting this hypothesis, adolescent girls with comorbid depression and externalizing behaviors experienced more dependent negative life events compared with depressed-only or externalizing-only adolescents (Daley et al., 1997). Also, individuals who are sensitive to rejection are more likely to anxiously expect, perceive, and overreact to rejection, and this process can contribute to depressed mood and hostility (Downey, Khouri, & Feldman, 1997; Downey, Lebolt, Rincon, & Freitas, 1998). In addition, depression and eating disorders co-occur, and one connection between the two may involve interpersonal negative events (e.g., peers teasing a girl about her physical appearance), which can then lead to initial negative affect and depression as posited in the model, as well as, perhaps, eating disorders.

### *Developmental Factors*

So far, we have advanced a general cognitive vulnerability-transactional stress depression model that likely would apply equally well to adolescents and adults. In addition, we include particular developmental factors that can influence the elaborated causal chain and help explain why depression rises dramatically during middle adolescence, especially among girls. We emphasize particular developmentally sensitive factors, including the importance of pubertal onset as a transitional period affecting a rise in the number of negative life events in adolescence, the influence of preexisting vulnerabilities (e.g., genetic risk, personality, and environmental adversity), and the formation and consolidation of cognitive vulnerability through normal cognitive developmental processes.

### *Pubertal Onset*

Puberty is a time of transition, and transitions often are associated with moderate emotional upheaval and increases in negative life events (Caspi & Moffitt, 1991; Graber, Brooks-Gunn, & Petersen, 1996; Graber, Brooks-Gunn, & Warren, 1995). Starting at the beginning of puberty after age 13, both boys and girls report overall more negative events throughout adolescence compared with childhood (Ge et al., 1994; Rudolph & Hammen, 1999). Newcomb, Huba, and Bentler (1981) found that middle adolescence, after the pubertal transition, is the peak time for negative events. This developmental increase in the number of negative events, starting after puberty, closely parallels the surge in depressive symptoms and disorder reviewed earlier. Thus, the research indicates that more negative events occur after the pubertal transition, and the rise in the number of negative events continues throughout adolescence for both boys and girls.

What happens around puberty that may account for the general rise in negative events for boys and girls? Prior research has found differences in the kinds of negative events that typically occur for different age groups. Preadolescents most frequently report family events, adolescents report interpersonal, peer-related events, and young adults report academic/achievement events (Rudolph & Hammen, 1999; Wagner & Compas, 1990). Supporting the trans-

actional stress generation hypothesis, adolescents experience more dependent negative life events, whereas preadolescent children have more independent negative events occur (Rudolph & Hammen, 1999). A developmental rise in the number of dependent, especially interpersonal, negative events might occur as a result of adolescents taking a more active role in selecting their friends as their parents do less monitoring of their children's friends with the transition into adolescence (Rubin, Bukowski, & Parker, 1998).

Thus, there are developmental changes from childhood to adolescence in the number and domains of negative events. The pubertal transition into adolescence is marked by an increase in the number of dependent, interpersonal events from independent (mostly family) events during childhood. The overall increase in the number of negative events can lead to elevations in initial negative affect and depressive symptoms, according to the elaborated causal chain, and thus provides an explanation for the rapid rise in depression observed during adolescence. Also, consistent with the elaborated causal chain, an increase in the number of dependent negative events is observed during adolescence as levels of initial negative affect and depressive symptoms become more prevalent.

### *Preexisting Distal Vulnerabilities*

In addition to the effect that puberty has on the increase in negative events, the model also posits that particular distal vulnerabilities will enhance the likelihood of encountering more negative events and developing cognitive vulnerability throughout childhood and adolescence. We consider three particular preexisting distal vulnerabilities: genetic risk, personality, and environmental adversity. It is important to clarify that although these vulnerability factors are presented as discrete categories in the general model, it is recognized that these factors likely are interconnected and influence each other. For example, within the box representing distal preexisting vulnerabilities, genetic and environmental adversity serve as risk factors affecting other aspects of the model. However, these are not completely separate risk factors, as demonstrated by behavioral genetic studies showing that factors typically viewed as environmental adversity, such as family discord, are influenced by genetic factors (Deater-Deckard, Fulker, & Plomin, 1999; Neiderhiser, Reiss, Hetherington, & Plomin, 1999; Reiss et al., 1995). Thus, this general, integrative model is intended to reflect the reality that individuals' propensity to develop psychopathology occurs as part of a complex nature-nurture relationship unfolding over time.

*Genetic risk.* Behavioral genetic research with children and adolescents has shown that depression is a moderately heritable disorder (see Rutter, Silberg, O'Connor, & Simonoff, 1999, for a review). Other twin studies with children and adolescents have found that the genetic vulnerability to depression partly overlaps with the genetic liability for externalizing, conduct problems (O'Connor, Neiderhiser, Reiss, Hetherington, & Plomin, 1998) and anxiety disorders (Thapar & McGuffin, 1997). This common genetic liability to depressive and overlapping disorders may largely reflect negative affect and neuroticism. Also of interest are twin studies showing that depressive symptoms are heritable during adolescence (after age 11), whereas shared common family environment, but not genetic factors, was associated with depres-

sion in childhood (before age 11; Murray & Sines, 1996; Thapar & McGuffin, 1996).

We posit in the model that genetic risk for depression operates, at least partially, by increasing the likelihood that some individuals will experience more negative events. Supporting this hypothesis, behavioral genetic research with children and adolescents (Silberg et al., 1999; Thapar, Harold, & McGuffin, 1998) and adults (Kendler, 1995; Kendler, Neale, Kessler, Heath, & Eaves, 1993) finds that negative events are partially heritable. Also consistent with our emphasis on assessing domains for negative events, a twin study with adult women (Kendler & Karkowski-Shuman, 1997) found that depression was associated with exposure to particular, especially interpersonal, negative events. Moreover, consistent with our hypothesis that puberty is a developmental transition influencing the development of depression and an increase in negative events, a longitudinal twin study (Silberg et al., 1999) reported that genetic vulnerabilities increased the risk for depression and for encountering negative events for girls after, but not before, puberty. Taken together, the existing research supports our hypothesis that distal genetic liabilities influence the occurrence of negative events. However, genetic research has not pinpointed the mechanism through which this genetic risk for negative events operates, and it certainly seems implausible that there are genes that code directly for negative life events. One putative pathway may involve genes coding for neurotransmitters, which in turn may be linked to personality traits that increase the likelihood of exposure to negative life events. In support of this, molecular genetic research (Lesch et al., 1996) has linked neuroticism with serotonin transporter genes.

*Personality.* We suggest that one possible pathway from elevated genetic risk toward a propensity to experience more negative life events and depression may be through genetically influenced personality traits, and neuroticism in particular. Consistent with other theorists (Clark, Watson, & Mineka, 1994), we conceptualize neuroticism as a disposition to experience negative affective states, including anxious, angry, and depressive affects. There are multiple methods of measuring personality, including self-report questionnaires, observation, interview, and experimental methods (see Pervin & John, 1999).

Considerable research supports the notion that many personality traits, such as neuroticism, are heritable (Roberts & Kendler, 1999; see Caspi, 1998; Plomin & Caspi, 1999; for reviews). Research suggests that neuroticism likely is a vulnerability factor for depression (Kendler et al., 1993; Krueger et al., 1996; Trull & Sher, 1994; see Widiger, Verheul, & van den Brink, 1999, for review). For example, a prospective study found that inhibited 3-year-olds were more likely to develop clinical depression at age 21 compared with normal and undercontrolled children (Caspi, Moffitt, Newman, & Silva, 1998). However, the mechanism by which neuroticism confers risk to depression is unclear, and the specificity of neuroticism for depression may be unlikely.

We suggest that neuroticism confers distal risk to depression, at least in part, by influencing the likelihood that particular individuals will encounter negative life. Consistent with this hypothesis, research has found that neurotic individuals are more likely to encounter more negative events prospectively measured over time (Hankin et al., 1999; Van Os & Jones, 1999). In a 7-year prospective study, neuroticism predicted distress partially through the mediating link of dependent negative events (Ormel & Wohlfarth,

1991). Because these studies have assessed negative events via self-report, it is possible that neurotic individuals do not, in reality, encounter more events. Rather, they may perceive an event as more negative or respond more negatively to an event. More methodologically sophisticated studies using different methods to assess negative events (e.g., contextual threat interviews, Monroe & Simons, 1991; documented naturalistic stressors, Metalsky et al., 1993) could help ascertain the nature of the prospective association among neuroticism, negative events, and depression.

Last, it is important to note that the association among neuroticism, negative events, and depression is likely not strictly unidirectional. Supporting the notion of a dynamic, transactional process, a prospective study (Ge & Conger, 1999) found that personality traits, like neuroticism, measured in adolescence predicted the later onset of emotional and behavior problems, and vice versa (i.e., these problem behaviors and emotional distress measured in adolescence predicted prospective changes in adult neuroticism).

*Environmental adversity.* Last, we consider the impact that environmental adversity may have as a preexisting vulnerability factor. Conceptually, we consider environmental adversities as a constellation of distal negative events as opposed to the more proximally occurring events specified in the vulnerability-stress aspect of the model. Environmental adversity is likely a large and expansive factor, so we explicitly limit our focus to particular distal negative events occurring during childhood that can influence cognitive vulnerability to depression or experiencing more negative events (both independent and dependent). Examples of distal negative events as environmental adversity include abuse/maltreatment, death/illness of a parent, and so forth. Because the number of studies examining various kinds of environmental adversity is too vast to review here, we focus on abuse/maltreatment. Research examining the link between abuse/maltreatment and depression has found that depression is commonly associated with reports of childhood abuse (Andrews, 1995; Bifulco, Brown, & Adler, 1991; Boudewyn & Liem, 1995; Browne & Finkelhor, 1986; Levitan et al., 1998; McCauley et al., 1997; Mennen & Meadow, 1994; Pribor & Dinwiddie, 1992). Although the majority of this research has used samples of adults retrospectively reporting on these events occurring during childhood, research has detected little to no discernible retrospective bias in recalling abusive experiences (Brewin, Andrews, & Gotlib, 1993; Rutter & Maughan, 1997). More recently, research has progressed beyond establishing an association between distal adversities, like abuse, and depression, toward examining plausible mechanisms. Consistent with our hypothesis, adults reporting childhood maltreatment have shown elevated cognitive vulnerability (Gibb et al., 2001; Rose, Abramson, Hodulik, & Halberstadt, 1994) and have prospectively encountered more negative events as assessed over a two year interval (Hankin et al., 1999). Other research (Downey et al., 1997) has shown how adolescents and young adults who were maltreated as children are more likely to develop rejection sensitivity, which can lead to dependent negative life events within romantic relationships.

#### *Development of Cognitive Vulnerability to Depression*

The final developmentally sensitive factor to consider is the development and consolidation of cognitive vulnerability to de-

pression. The general cognitive vulnerability factors likely become more salient and operative as the child progresses through normal cognitive development. Although the precise mechanisms through which cognitive risk factors for depression develop are unknown, previous theory and research suggest that cognitive vulnerability may mediate the negative event–depression association earlier in childhood but moderate the event–depression relationship (as hypothesized in the generic cognitive vulnerability-stress model) starting around late childhood (Chorpita & Barlow, 1998; Cole & Turner, 1993). In other words, cognitive vulnerability to depression may result, in part, as a cognitively maturing child works to make sense of negative events that occur in an effort to control and predict his or her environment. Previous research indicates that cognitive vulnerability to depression appears to be actively on-line and functioning fully by early adolescence/puberty. Various studies have demonstrated that the cognitive vulnerability (negative inferential style)-stress interaction predicts elevations in depression as early as age 10 (Hilsman & Garber, 1995; Nolen-Hoeksema et al., 1992). For example, a 5-year longitudinal study (Nolen-Hoeksema et al., 1992) found that negative events directly contributed to depressive symptoms in 3rd and 4th graders, whereas the cognitive vulnerability-stress interaction predicted depressive symptoms starting in 5th grade. Broderick (1998) found that a ruminative response style was actively on-line and conferred vulnerability to depression by age 9, before the pubertal transition. In short, then, cognitive vulnerability to depression appears to be formed, consolidated, and ready to interact with negative events before the number of negative events rises after the pubertal transition.

The fact that cognitive vulnerability, which is hypothesized to be relatively depression-specific, is available and accessible by late childhood may resolve and explain, at least partially, the developmental patterns of comorbidity, especially anxiety preceding depression, noted earlier. Within the model, the fact that anxiety disorders often develop prior to depression is accounted for by negative events leading to negative, primarily anxious, affect. We posit that depression developing later in childhood, as opposed to anxiety onsets occurring earlier, is the result of depression-specific cognitive vulnerability having consolidated and being actively

on-line after a child has developed cognitively. Thus, the negative events increasing throughout adolescence can now interact with the available cognitive vulnerability to contribute to depression according to the elaborated causal chain. Anxiety would often co-occur alongside depression as the negative event engenders the elevation in initial levels of negative affect, and anxiety would be observed without comorbid depression if cognitive vulnerability does not interact with the negative event (e.g., a domain mismatch). To explain how cognitively immature children can develop depression, we posit that a sufficiently major, negative event, particularly one that indicates loss, can precipitate depression directly. This hypothesis is consistent with the titration model within the cognitive vulnerability-stress theory.

#### Explanation of the Development of the Gender Difference in Depression: Derivation From the Elaborated Cognitive Vulnerability-Transactional Stress Model

We have presented a general, integrative cognitive vulnerability transactional-stress model for depression, but how does it account for between group differences, such as why more girls than boys start becoming depressed, particularly after early adolescence? Also, how does the model explain individual differences in depression proneness, such as why some, but not all, girls (or boys) become depressed? We derive such between group and individual differences through processes explicated in the general model seen in Figure 2. Thus, in Figure 3 we depict how an explanation for the emergence of the gender difference in depression can be derived from factors and processes in the general, elaborated model.

In this section, we illustrate how the elaborated causal chain can account for more girls than boys becoming depressed after early adolescence. Specifically, we underscore those causal factors in the model on which girls show elevations relative to boys. Below, we present evidence that supports the following causal synopsis of the increase in depression among adolescent girls relative to adolescent boys, as seen in Figure 3. In brief, adolescent girls are more likely than boys to encounter negative life events, leading to greater elevations of initial negative affect, particularly depressed and anxious mood, among girls. Also, adolescent girls are more

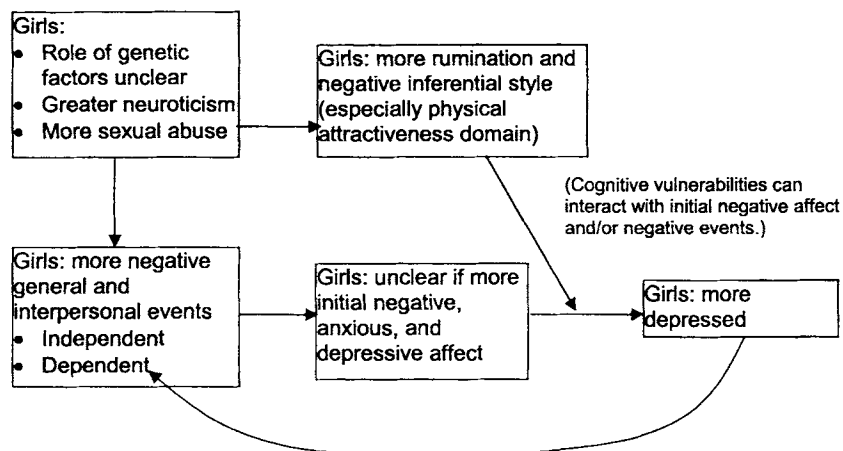


Figure 3. Schematic diagram illustrating the specific case of the emergence of the gender difference in depression derived from the elaborated cognitive vulnerability-transactional stress model of depression.

cognitively vulnerable to depression than boys, and this greater cognitive vulnerability enhances the likelihood of girls' experiencing later increases in depressive symptoms in response to negative events, initial negative affect, or both. Girls' higher levels of depressive symptoms, in turn, contribute transactionally to the generation of more dependent, negative life events, which in turn restart the causal chain leading to depression. Last, girls demonstrate elevations on some of the preexisting distal vulnerabilities compared with boys, including personality traits (e.g., neuroticism) and environmental adversity (e.g., sexual maltreatment), both of which contribute to girls encountering more negative events and having more negative cognitive vulnerability than boys.

Below, we review studies that have investigated whether there are gender differences in these components of our proposed elaborated causal chain to evaluate whether the model can explain the emergence of the gender difference in depression. No study has yet tested all of the hypothesized processes and factors of the general model in an integrative fashion. Moreover, many of the studies testing aspects of the model used cross-sectional designs. As a result, our review consists mainly of cross-sectional studies that have investigated the mechanisms and factors in isolation from other components in our proposed model. Because we have posited a developmentally sensitive, transactional model, it will be important for future research to test the hypothesized mechanisms and factors from the model in an integrative and prospective design.

#### *Gender Differences in Negative Events*

The general elaborated causal chain starts with the occurrence of negative events, so what is the evidence that girls encounter more stressful events than boys? Studies reporting statistics on negative events as a function of gender find that girls report more negative events than boys (Davies & Windle, 1997; Forteza, Salgado-de Snyder, Andrade, & Tapia, 1996; Garton & Pratt, 1995; Ge et al., 1994; Graber et al., 1995; Kearney, Drabman, & Beasley, 1993; Schraedley et al., 1999; Tubman & Windle, 1995). Studies that have separated the event domains have found that girls report more interpersonal (especially peer-related) negative events than boys (Larson & Ham, 1993; Rudolph & Hammen, 1999; Towbes, Cohen, & Glyshaw, 1989; Wagner & Compas, 1990; Windle, 1992), whereas boys report more negative academic, school events. Also, supporting the transactional stress generation mechanism, adolescent girls reported more dependent, interpersonal events (especially with family and peers), and adolescent boys experienced more dependent, noninterpersonal events (Rudolph & Hammen, 1999).

Other studies have found that negative events mediate the gender difference in depression during adolescence. In an investigation of negative events taking place within adolescents' families, Davies and Windle (1997) found that stressful events accounted for girls' higher levels of depressive symptoms. Within adolescents' peer relationships, girls' increased number of interpersonal negative events (peer rejections) mediated the gender difference in negative affect (Liu & Kaplan, 1999).

Cross-sectional studies (Rudolph & Hammen, 1999; Windle, 1992) show that overall negative life events correlated with depressive symptoms among girls, but not boys, with the strongest association found for interpersonal events. In a prospective study, Ge and colleagues (1994) reported that girls', but not boys',

depressed mood correlated with uncontrollable negative events. A second prospective study (Silberg et al., 1999) found that the impact of life events predicted clinical depression more strongly among pubertal girls compared with boys or prepubertal girls. Last, consistent with our emphasis on puberty as an important transition, research suggests that gender moderates the stress-emotional maladjustment association among middle adolescents (Horwitz & White, 1987; Newcomb et al., 1981), whereas no gender moderation is found among prepubertal children (Cohen et al., 1987). Thus, these studies suggest that gender moderates the effect of negative life events on depression. However, it is important to note that finding that gender moderates the association between negative life events and depression does not imply that there is a gender difference in the level of negative life events. As a result, the significance of this set of findings for explaining why more girls than boys become depressed is unclear.

Consistent with the developmental aspects of our model, studies investigating the developmental pattern of the gender difference in negative events have found that girls experience more negative events than boys after the pubertal transition (Ge et al., 1994; Graber et al., 1995; Rudolph & Hammen, 1999), especially interpersonal dependent events. Other research provides some suggestions why adolescent girls may encounter more negative, interpersonal events after puberty. Girls are especially aware of conflict in interpersonal friendships, and such conflict increases throughout adolescence (Laursen, 1996). Other research has shown that early-maturing girls choose more antisocial, deviant peers for friends compared with on-time or later maturing girls (Caspi & Moffitt, 1991; Ge et al., 1996), and associating with these more deviant peers may lead to the occurrence of more negative events and then future increases in depression. In support of this mechanism, Ge et al. (1996) found that early-maturing girls selected more deviant peers, and, in turn, association with peers prospectively predicted early maturing girls' increased anxious and depressive symptoms.

In addition to pubertal transition, the general model predicts that distal vulnerability factors lead to an increase in negative events. Preliminary support for this hypothesis comes from studies finding gender differences in some of the distal vulnerability factors (see distal vulnerability section later for review of studies).

#### *Gender Differences in Initial Negative Affect*

The general model posits that initial negative affect results from the occurrence of negative events. Knowing that girls experience more negative events than boys, what evidence is available that girls report more initial negative affect than boys? Our review earlier in the section on the descriptive timeline for the development of depression clearly shows that girls experience more negative affect than boys, particularly depression and anxiety. This review of descriptive studies also showed that anxiety often precedes depression and girls report more anxiety symptoms and disorders than boys. Given the strong general negative affective factor common to depression and anxiety (Brady & Kendall, 1992), this pattern suggests that girls experience more negative affect, particularly anxiety followed by depression.

However, this corpus of evidence does not directly address the difference between initial negative affect and subsequent increases in depression, as posited in our model. We located no studies testing whether girls show more immediate negative affect in

response to negative life events compared with boys. Thus, at this point, it is unknown whether girls are higher than boys on initial negative affect. An important avenue for future research is examining whether there is a gender difference in this link in the causal chain. Although the step of immediate initial negative affect is important for the general causal chain, it is important to note that an explanation for the emergence of the gender difference in depression, based on our general model, does not require girls to show more initial negative affect than boys because girls can become more depressed than boys through other processes in the model (e.g., more negative life events and greater levels of cognitive vulnerability).

In addition to the evidence for gender differences in overall levels of negative affect among adolescents, other research has shown that girls express depressive affect in response to negative events involving themselves (e.g., academic failures) and significant interpersonal others (e.g., family illness), whereas boys tend to react only to events that affect themselves (Gore, Aseltine, & Colten, 1993; Newcomb et al., 1981; Siddique & D'Arcy, 1984; Wagner & Compas, 1990). Gjerde (1995) found gender differences in the emotional symptom expression, in which boys responded to depressed mood in a more hostile, angry, and conduct-disorder manner, whereas girls responded to depressed mood in a more ruminative manner.

#### *Gender Differences in Cognitive Vulnerability to Depression*

In addition to a gender difference in negative events, our model posits that girls' greater cognitive vulnerability, in response to these negative events, contributes to more girls than boys becoming depressed. In the general depression model, dysfunctional attitudes, negative inferential style, and a ruminative response style are included as generic cognitive vulnerabilities. Research with adolescents has found gender differences in some aspects of cognitive vulnerability. No gender difference in dysfunctional attitudes has been found among adolescents (e.g., Lewinsohn et al., 1997). Most studies have not found a gender difference in attributional style (Hankin et al., 2001; Lewinsohn et al., 1997), although one study found that 10th-grade girls exhibited a more negative attributional style than boys (Nolen-Hoeksema & Girgus, 1995). This inconsistency in findings may be due to the poor internal reliability of the measure of attributional style used in these studies (see Gladstone et al., 1997; Hankin & Abramson, 2001). Indeed, a study with adolescents (Hankin & Abramson, 2001) using a more internally reliable measure revealed gender differences in negative inferential style (including negative inferences about the causes of events and implications for self-worth) but did not find gender differences in the previously used, less reliable measure of attributional style. Thus, preliminary evidence suggests that when inferential style is measured reliably, adolescent girls exhibit a more depressogenic inferential style than boys. Studies of ruminative response style with adolescents (Broderick, 1998; Hart & Thompson, 1996; Schwartz & Koenig, 1996) and adults (Butler & Nolen-Hoeksema, 1994; Nolen-Hoeksema et al., 1993; Nolen-Hoeksema, Larson, & Grayson, 1999; Nolen-Hoeksema, Parker, & Larson, 1994) have found gender differences with girls showing more rumination than boys.

Girls' greater levels of cognitive vulnerability than boys means that girls would be more likely to become depressed than boys even if there were no gender difference in negative life events. But with the rise in negative events throughout adolescence, especially for girls, the individuals who ruminate, negatively interpret, and make negative inferences about these negative life events are especially vulnerable to experiencing increases in depression. Thus, the emerging gender difference in depression can be explained, at least in part, by girls' greater cognitive vulnerability in combination with their rise in negative events throughout adolescence.

In addition, studies also have shown that cognitive vulnerability mediates the gender difference in adolescent depression. In a cross-sectional study of adolescent depression, Hankin and Abramson (2001) showed that negative inferential style mediated the gender difference in depressive and internalizing symptoms. Moreover, a ruminative response style partially mediated the gender difference in adolescent depressed mood (Hart & Thompson, 1996; Schwartz & Koenig, 1996).

#### *Development of Girls' Greater Cognitive Vulnerability to Depression*

Our review suggests that adolescent girls have more cognitive vulnerability to depression than boys, but how do girls develop such cognitive risk factors? As we have indicated, girls are more likely to experience negative events, especially interpersonal ones, than adolescent boys. Drawing on Kelley's work (1967), researchers (e.g., Dykman & Abramson, 1990; Just, Abramson, & Alloy, 2001; Metalsky & Abramson, 1981) have hypothesized that individuals confronted with repeated occurrences of negative life events in a wide variety of domains should develop a more stable, global attributional style for negative events over time and, hence, increases in cognitive vulnerability to depression. Coyne and Whiffen (1995) similarly argued that people's current social contexts may exert a powerful influence on their cognitive vulnerability to depression. Thus, girls' greater likelihood of experiencing negative events may contribute to greater cognitive vulnerability to depression.

In addition to encountering more negative events, girls also encode these events in greater detail in large associative cognitive networks connected to affective nodes. Two recent studies (Davis, 1999; Seidlitz & Diener, 1998) have shown that females encode life events in more detail than males. For example, Davis (1999) revealed gender differences in autobiographical memory, with females (both children and adults) recalling more childhood emotional memories and recalling these emotional memories more quickly than males. No gender difference was found for nonemotional memories. These cognitive findings can be integrated with other research from gender socialization. Parents discuss emotional material in more detail with their daughters compared to sons (Brody & Hall, 1993). Mothers discussed sadness in longer and more detailed conversations with daughters than sons, whereas anger was talked about more with sons than daughters (Fivush, 1991). Also, research has found that mothers are more controlling of their daughters than sons, and this pattern of gender socialization partially mediated girls' more negative self-evaluations, especially their tendency to take responsibility for failure compared with boys (Pomerantz & Ruble, 1998). Such findings suggest that

females' greater cognitive vulnerability than males may derive, in part, from parent's gender socialization of their children and from girls' encoding negative events in more emotional detail than boys.

These studies have all examined general cognitive vulnerability. Existing cognitive theories posit that a domain (e.g., interpersonal or achievement) match between the cognitive vulnerability and a negative event will heighten the likelihood of depression. No study has examined yet whether girls experience more domain matches between cognitive vulnerability and negative life events than boys. However, girls are more cognitively vulnerable in the interpersonal domain than boys (Bandura, Pastorelli, Barbaranelli, & Caprara, 1999; Leadbeater, Blatt, & Quinlan, 1995; Leadbeater, Kuperminc, Hertzog, & Blatt, 1999).

### *Role of Negative Cognitions About Attractiveness and Body Image*

Starting in 3rd grade and by the end of high school, Caucasian girls show less satisfaction with their physical appearance than do boys (Harter, 1999). Over 80% of adolescent girls (age 12–18), compared with 40% of boys, reported dissatisfaction with their body image (Kostanski & Gullone, 1998; see also Paxton et al., 1991; Rierdan et al., 1989; Wertheim et al., 1992). Adolescent girls express dissatisfaction with their weight even when they are within normal weight range for their height (Casper & Offer, 1990; Dornbusch, 1984; Drenowski & Yee, 1987). Early maturing girls are at higher risk for negative perceptions of their body image and weight compared with on-time peers (Graber, Brooks-Gunn, Paikoff, & Warren, 1994).

Girls who derived their self-worth from physical appearance reported low self-esteem and high levels of depressive affect (Harter, 1999). A cross-sectional study (Kostanski & Gullone, 1998) found that perceived body dissatisfaction was positively associated with both anxious and depressive symptoms especially for girls. Another study (Cole et al., 1998) found that 4th and 5th graders' beliefs that they were physically unattractive predicted depressed mood for girls more than for boys. Last, initial body dissatisfaction, dietary restraint, and bulimic symptoms predicted onset of depressive episodes for girls over a prospective 4-year period (Stice, Hayward, Cameron, Killen, & Taylor, 2000).

Other studies have found that negative perceptions of attractiveness mediated the gender difference in adolescent depression. One cross-sectional study (Allgood-Merten, Lewinsohn, & Hops, 1990) of high school adolescents showed that girls' excessive body dissatisfaction mediated the gender difference in depressed mood. Hankin and Abramson (2001) found that negative cognitive style in the domain of perceived attractiveness mediated the gender difference in depressive and internalizing symptoms. Moreover, adolescent girls' greater actual-ideal self-discrepancies partially mediated the gender difference in depressive, but not anxious, symptoms (Hankin, Roberts, & Gotlib, 1997). Such actual-ideal self discrepancies may include differences in the kind of body shape and physical appearance girls would ideally like to have compared with the body shape and physical appearance they believe they actually have. Last, compared with boys, adolescents girls' excessive dissatisfaction with their body shape was associated with increases in depressed mood around age 13 through decreases in self-esteem and disappointment with physical appearance (Wichstrom, 1999).

Taken together, these findings suggest that adolescent girls, compared with boys, have negative cognitions about their physical attractiveness and bodies that, in turn, contribute to their elevated levels of depression. How can we understand these findings from the perspective of our elaborated cognitive vulnerability-transactional stress theory?

As we noted earlier, adolescent girls exhibit elevations on some aspects of cognitive vulnerability including negative inferential styles about the causes of negative events and their implications for self-worth in the domain of physical attractiveness and body satisfaction (Hankin & Abramson, 2001). Such cognitive vulnerability should put adolescent girls at risk for negative cognitions about their physical attractiveness and bodies when they encounter negative events in this domain (e.g., a boyfriend telling her there is something wrong with her appearance). Thus, the tendency for adolescent girls to exhibit negative cognitions in this domain is predicted by their elevated cognitive vulnerability in the domain. The elaborated cognitive vulnerability-transactional stress theory further predicts that these negative cognitions, in turn, will contribute to the formation of depressive symptoms, consistent with the findings above.

It is intriguing that negative cognitions about physical attractiveness and body satisfaction may be even more predictive of depression among girls than boys (Cole et al., 1998). We speculate that this occurs because the domain of personal physical attractiveness and body satisfaction may be more motivationally significant for girls than boys. Both evolutionary (Buss, 1994) and socialization (Eagly & Wood, 1999) theories of mate selection are consistent with this view. Females tend to value financial security, status, and commitment in their mates, whereas males tend to find physical attractiveness and youth more valuable in their mates. Thus, the domain of personal physical attractiveness and body satisfaction is likely to be of greater motivational significance for adolescent girls than adolescent boys. It may be no surprise, then, that Hankin and Abramson (2001) reported that adolescent girls are more likely than adolescent boys to make negative inferences about their self-worth when they confront negative events in the domain of physical appearance. Such events may signal lower potential mate value for a female than for a male. Of interest, physical attractiveness and youth may be less controllable, harder to achieve, and more ephemeral than financial security and status. Supporting this hypothesis, recent research (Ben Hamida, Mineka, & Bailey, 1998) showed that the traits of physical attractiveness and youth, which were especially important for female mate value, were rated as more uncontrollable than security and status. Thus, females, especially after puberty, may likely experience increased helplessness, anxiety, low self-worth, and depression if they believe that physical attractiveness and youth are the only, or most likely, ways to find a mate.

Given the potential importance of the domain of physical attractiveness and body image in the emergence of gender differences in depression, it is important to explore the origins of females' cognitive vulnerability in this domain. For example, are females socialized to make stable, global attributions and infer negative consequences and self-characteristics when confronted with negative events related to physical attractiveness? And once adolescent females' cognitive vulnerability about physical attractiveness and body image is in place, do they confront more negative events in this domain than do adolescent males? For

instance, in heterosexual couples, do females receive more negative comments about their attractiveness and bodies from their boyfriends than their boyfriends receive from them? The answers to such questions will contribute to further understanding of the emergence of gender differences in depression.

Last, the uniqueness and cultural importance of body dissatisfaction and negative perceptions of attractiveness as depression vulnerabilities should be considered. The increased body fat accumulated during puberty is more disappointing for Caucasian girls than for African American girls (Casper & Offer, 1990; Halpern, Udry, Campbell, & Suchindran, 1999; Parker, Nichter, Nichter, & Vuckovic, 1995). In addition, Caucasian girls believe that a slim body importantly affects the likelihood of dating (Paxton et al., 1991) and popularity among peers (Parker et al., 1995). In our model, not having a romantic partner and being unpopular with peers are conceptualized as potent interpersonal, negative life events that can contribute to depression. However, findings suggesting that negative perceptions of body satisfaction and physical attractiveness influence the gender difference in depression need to be interpreted cautiously because these studies have been conducted with mostly Caucasian samples. Future studies should determine if these results apply to more ethnically diverse children. A final important question for further research is how unique this domain of cognitive vulnerability is for depression, particularly given prior research showing that body dissatisfaction is also linked to eating disorders as well as depression (Stice et al., 1998; Stice et al., 2000).

#### *Distal Preexisting Vulnerability Factors*

In the general elaborated causal chain, we included genetic risk, personality, and environmental adversity as individual difference developmental factors that distally influence the likelihood that more negative events will be encountered and cognitive vulnerability to depression will develop. In this section we review whether there are gender differences in these distal risk factors. But beyond simply identifying whether there are gender differences in these distal factors, it will be important for future research to test the model's hypothesis that girls showing elevations in these risk factors compared with boys leads to more negative events and greater cognitive vulnerability among girls, which in turn, would eventuate in girls' increased depression. However, no study has yet investigated this more elaborate hypothesis.

#### *Gender Difference in Genetic Heritability*

Adult (Kendler & Prescott, 1999; Lyons et al., 1998) and child/adolescent (Eaves et al., 1997; Rutter et al., 1999) twin studies have obtained nearly identical genetic heritability estimates for males and females. However, Kendler and Prescott (1999) further reported that men and women do not share identical genes for risk for depressive disorders, nor do they not share any genes together, because the genetic risk for depressive disorder for men and women was moderately correlated ( $r = .57$ ). This moderate genetic overlap toward depression liability between men and women suggests that men and women may have different, but somewhat overlapping, pathways toward depressive disorders.

In contrast to these studies, Jacobson and Rowe (1999) found a gender difference such that the genetic contribution for depressed

mood was greater among adolescent girls than boys. They also reported that the genetic vulnerability contributing to the association between depressed mood and family connectedness was higher among adolescent girls than boys. Another child/adolescent twin study (Silberg et al., 1999) found a genetic heritability for depressive disorders only among pubertal girls, with no significant heritability for boys or prepubertal girls. The moderate heritability effects for adolescent girls included a common genetic factor that influenced the liability to experience both increases in depression and negative life events. Moreover, latent genetic factors mediated the long-term stability of depressive disorders among pubertal girls. Silberg et al. (1999) concluded that the emergence of a genetic liability to depressive disorders combined with life events (which are also partially genetically mediated) during adolescence accounted for the onset of depressive disorders that was more evident among pubertal girls. Although these gender differences in the heritability of depression among adolescents are interesting, their significance for explaining elevations of depression among adolescent girls remains unclear.

#### *Gender Difference in Personality: Neuroticism*

Various studies show that adolescent girls report higher levels of neuroticism than boys (e.g., del Barrio, Moreno-Rosset, Lopez-Martinez, & Olmedo, 1997; Goodyer et al., 1993). Also, as several personality theorists consider neuroticism as being on a continuum with general negative affect (e.g., Clark & Watson, 1991), the evidence reviewed above concerning gender differences in negative affect also may support this hypothesis.

#### *Gender Difference in Environmental Adversity: Child Maltreatment*

Research has found gender differences in childhood sexual abuse (CSA) but not physical or emotional maltreatment (Kaplan, Pelcovitz, & Labruna, 1999). In an adult sample of clinic patients, 17% of the depressed adults reported CSA, and 87% of this CSA subsample were women (Gladstone et al., 1999). Finally, a meta-analysis (Rind, Tromovitch, & Bauserman, 1998) of adults retrospectively recalling CSA concluded that 14% of men and 27% of women reported such events. Moreover, indicative of moderation, women showed a stronger CSA—emotional maladjustment relationship than men. Whiffen and Clark (1997) found that a history of CSA completely mediated the gender difference in adult depression.

Although these studies provide support for the model's hypothesis that females experience more distal environmental adversity than males, this research does not directly address the issue of the emergence of the gender difference in depression. The participants from such studies (e.g., Whiffen & Clark, 1997) are adults reporting on their lifetime histories of depression, mostly occurring during adulthood, so these studies apply most precisely to the adult gender difference in depression. To explain the emergence of the gender difference in adolescence, future research must either use adolescents reporting on their past and current depression or have adults recall the onset date of depression more precisely while also assessing the timing of childhood adversities. In addition, most studies of adults retrospectively recalling abusive experiences do not clearly consider when the abuse occurred as a potential factor.



### Biological Explanations

In our model and review of evidence, we have not explicitly included biological risk factors. Biological factors have not been excluded because we think they are unimportant in understanding the development of gender differences in depression. Instead, we have not focused on them because there is currently less evidence that they account for the emergence of the gender difference in depression. Also, it is important to note that we do not view biological risk factors as incompatible or mutually exclusive with our model (see Abramson et al., in press, for an integration). We review biological factors that have been linked to depression to see whether there are gender differences in such vulnerabilities and whether they account for the gender difference in depression (see Bebbington, 1998, for a review).

#### *Hormones*

A review of mood changes during puberty generally concluded that hormonal changes, by themselves, may not lead to significant alterations in mood or poor adjustment (Buchanan, Eccles, & Becker, 1992). In addition, little consistent evidence exists to support the hypothesis that rising levels of female hormones (e.g., progesterone, estrogen) mediate the gender difference in depression. Brooks-Gunn and Warren (1989) found the effect of one sex hormone (estradiol) among girls (ages 10–14) was minimal compared with the influence of social factors (negative life events) in predicting depressed mood. Of interest, however, at the initial evaluation and 1 year later, depressive affect was highest for girls with rising estradiol levels, although these levels were below adult hormonal levels (Paikoff, Brooks-Gunn, & Warren, 1991). Other studies (e.g., Susman, Dorn, & Chrousos, 1991; Susman, Nottelman, Inoff-Germain, Dorn, & Chrousos, 1987) found no gender differences in adrenal hormones. We found only one study (Angold, Costello, Erkanli, & Worthman, 1999) supporting the hypothesis that female hormonal levels mediated girls' increased levels of depression in an all female sample. But, conclusions cannot be made from this study about whether sex hormones mediate the emergence of the gender difference in depression because boys were not included in the analyses.

In addition, results of studies investigating depressive affect associated with girls' menstrual cycle have been inconsistent. Golub and Harrington (1981) found no significant relationship between negative affect and menstrual cycle phase. Other research has found that girls' premenstrual syndrome is associated with emotional distress (particularly anxiety) during adolescence (Freeman, Rickels, & Sondheimer, 1993; Raja, Feehan, Stanton, & McGee, 1992). Also potentially informative is the literature on mood changes associated with postpartum depression (Hendrick & Altshuler, 1999; O'Hara, 1997; O'Hara & Swain, 1996). For example, Troutman and Cutrona (1990) found that pregnant adolescents had higher rates of depression compared with nonpregnant girls. Focusing research attention on biological factors associated with adolescent girls' postpartum depression could provide important information relevant to the hormonal factors contributing to girls' rising levels of depression during adolescence.

#### *Biological Stress Response*

Results of adult studies examining the biological stress response of the hypothalamic-pituitary-adrenal (HPA) axis have found in-

creases in the stress hormone cortisol and corticotropin-releasing hormone (CRH) among depressives compared with controls (Thase, 2000). However, research with depressed adolescents has not found HPA axis dysfunction or elevations of cortisol compared with controls (Dahl, Siegel, & Williamson, 1992; Dorn et al., 1996; Dorn & Chrousos, 1997). Other research (Susman et al., 1999) found no association between plasma cortisol and depression during pregnancy or the postpartum period among a group of adolescent girls, although lower levels of CRH were associated with depressive symptoms during pregnancy. In addition, some studies have found no gender difference in levels of cortisol (Susman et al., 1987, 1991). Moreover, contrary to research with adults showing that healthy women have higher cortisol responses compared with healthy men (Gallucci et al., 1993), research with adolescents has found that boys, compared with girls, had significantly higher levels of cortisol (Dahl et al., 1992; Dorn et al., 1996) and noradrenaline (Elwood, Ferguson, & Thakar, 1986). To date, only one study (Susman, Dorn, Inoff-Germain, Nottelman, & Chrousos, 1997) found that increased cortisol levels were associated with higher emotional distress for girls compared with boys, and this was obtained only when the blood samples were collected under the most novel and uncertain conditions.

#### *Other Biological Factors*

Although depression has been associated with various neurotransmitters, such as lowered serotonin levels in children and adolescents (Hughes, Petty, Sheikha, & Kramer, 1996), no gender difference has been found in serotonin (Sallee et al., 1998) or other neurotransmitter metabolites in depressed inpatients (Mokrani, Duval, Crocq, Bailey, & Macher, 1997). Last, no gender difference has been found in natural killer cell activity (Birmaher et al., 1994).

#### *Summary*

Existing studies of biological explanations have provided little consistent evidence that hormone, cortisol, or neurotransmitter levels mediate the gender difference in depression (see Seeman, 1997, for a similar conclusion in a review of adults). Nevertheless, there are few studies in this area, and many of them have used small samples sizes, so not finding statistically significant gender differences may result from a lack of statistical power. A meta analysis across the various studies could discern whether there exists a meaningful gender difference in various biological vulnerabilities, such as hormonal or cortisol levels. Finally, most of the studies have considered only simple linear models in which biological changes (e.g., hormonal; Brooks-Gunn et al., 1994) directly influence negative affect, although it is unlikely that such a direct model would capture the complexity and interdependence of the biological system. A transactional model involving bidirectional effects among biological changes, negative affect, pubertal timing, and social events is likely to best represent how biological factors might influence negative affect (Brooks-Gunn et al., 1994). A complementary model suggests that affective dysregulation during female biological transitions, such as menarche, may be associated with the neurobiology of stress (Dorn & Chrousos, 1997). Further examination of these hypotheses with more sophisticated methods (e.g., cortisol and hormonal assays with assessments of

negative life events throughout the menstrual cycle) appears warranted given the existing limitations in studies.

### Conclusion

This article reviewed studies to construct a descriptive timeline of the development of the gender difference in depression. Both boys' and girls' rates of depression rise dramatically throughout middle adolescence, but girls show a more dramatic increase than boys starting after age 13 or middle puberty. This timeline for the emergence of the gender difference in depression appears relatively unique to depression despite its overlap with other psychiatric disorders.

We emphasized the scientific value of having a general model of depression and then explaining the emergence of gender differences in depression as a special case of the general model. Starting from existing general cognitive vulnerability-stress theory for depression, we elaborated on this causal chain by incorporating initial elevations of negative affect, integrating a transactional stress-generation mechanism, and expanding general cognitive vulnerability. We also posited particular developmentally sensitive factors (e.g., puberty, distal risk factors) that contribute to the dramatic rise in depression rates throughout adolescence, especially for girls. Evidence was reviewed that provides preliminary support for this elaborated cognitive vulnerability-transactional stress causal chain as a general model of depression for children, adolescents, and adults.

To explain how more girls than boys become depressed starting in early adolescence, we advocated using our general depression model and then determining which factors in the model show gender differences. Our review of studies indicates that there are gender differences among adolescents in negative life events, particularly interpersonal events; aspects of cognitive vulnerability; dependent negative events created through the stress generation mechanism; and some of the distal preexisting vulnerability factors. Such studies finding gender differences in many of the proposed model's factors and processes along with the supportive evidence for the general model provide preliminary evidence that our elaborated causal chain may account, at least in part, for the development of the gender difference in depression.

In addition to providing a theoretical framework for understanding the etiology and course of the developing gender difference in depression, our model also suggest points for treatment and intervention. Efforts can be aimed at the more distal vulnerability factors, such as reducing levels of environmental adversity and implementing coping strategies to combat preexisting anxiety that precedes depression. In addition, interventions such as cognitive behavioral treatment (CBT; e.g., Gillham, Reivich, Jaycox, & Seligman, 1995) can address the more proximal risk factors to prevent the formation and consolidation of cognitive vulnerability to depression. In particular, adding interventions for cognitive vulnerability in the domain of physical attractiveness to the CBT course could be particularly helpful for girls. These are a few possibilities that may have an effect at reducing the rising rates of depression throughout adolescence, especially for girls.

We believe that the general, elaborated cognitive vulnerability-transactional stress model of depression offers an integrative model capable of accounting for the general development of depression and for specific derivable cases, such as the emergence of

the gender difference in depression. We hope this proposed model stimulates further research into this important and exciting area. We look forward to further studies testing the model's hypotheses.

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### Call for Nominations

The Publications and Communications Board has opened nominations for the editorships of *Journal of Experimental Psychology: Animal Behavior Processes*, *Journal of Personality and Social Psychology: Personality Processes and Individual Differences*, *Journal of Family Psychology*, *Psychological Assessment*, and *Psychology and Aging* for the years 2004–2009. Mark E. Bouton, PhD, Ed Diener, PhD, Ross D. Parke, PhD, Stephen N. Haynes, PhD, and Leah L. Light, PhD, respectively, are the incumbent editors.

Candidates should be members of APA and should be available to start receiving manuscripts in early 2003 to prepare for issues published in 2004. Please note that the P&C Board encourages participation by members of underrepresented groups in the publication process and would particularly welcome such nominees. Self-nominations are also encouraged.

Search chairs have been appointed as follows:

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- Sara Kiesler, PhD, for *JPS: PPID*
- Susan H. McDaniel, PhD, and Mark I. Appelbaum, PhD, for the *Journal of Family Psychology*
- Lenore W. Harmon, PhD, for *Psychological Assessment*
- Randi C. Martin, PhD, and Joseph J. Campos, PhD, for *Psychology and Aging*

To nominate candidates, prepare a statement of one page or less in support of each candidate. Address all nominations to the appropriate search committee at the following address:

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The first review of nominations will begin December 14, 2001.