The Cognitive Vulnerability to Depression (CVD) Project: Current Findings and Future Directions

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Research has suggested that depression often occurs following stressful life events (see Monroe & Hadjistavropoulos, 2002, for a review). However, individuals can vary widely in their responses to such events. Some may develop severe or long-lasting depression, whereas others do not become depressed at all or may experience mild dysphoria. Several factors have been proposed to explain such individual differences in response to life events. For example, the severity of a given negative life event, the amount of social support an individual receives in the face of a traumatic life event, or individual differences in one’s biological constitution or psychological characteristics may all modulate reactivity to stressful events. From a cognitive perspective, the meaning or interpretation individuals give to the life events they experience influences whether or not they become depressed and are vulnerable to recurrent, severe, or long-lasting episodes of depression. Two major cognitive theories of depression, the hopelessness theory (Abramson, Metalsky, & Alloy, 1989; Alloy, Abramson, Metalsky, & Hartlage, 1988) and Beck’s theory (Beck, 1967, 1987), reflect such vulnerability–stress models, in which variability in individual susceptibility to depression following stressful events is understood in terms of differences in cognitive patterns that affect how those events are...
interpreted. According to both theories, particular negative cognitive styles increase an individual's likelihood of developing episodes of depression after experiencing a negative life event—specifically, a cognitively mediated subtype of depression (Abramson & Alloy, 1990; Abramson et al., 1989). These theories propose that people who possess "depressogenic" cognitive styles are vulnerable to depression because they tend to generate interpretations of their experiences that have negative implications for themselves and their futures.

In the hopelessness theory (Abramson et al., 1989), people who exhibit a depressogenic inferential style are hypothesized to be vulnerable to developing episodes of depression, particularly a "hopelessness depression" subtype (HD), when they are exposed to negative life events. This depressogenic inferential style is characterized by a tendency to attribute negative life events to stable (likely to persist over time) and global (likely to affect many areas of life) causes, to infer that negative consequences will follow from a current negative event, and to infer that the occurrence of a negative event in one's life means that one is fundamentally flawed or worthless. People who exhibit such an inferential style should be more likely to make negative inferences regarding the causes, consequences, and self-implications of any stressful event they experience, thereby increasing the likelihood that they will develop hopelessness, the proximal sufficient cause of the symptoms of hopelessness depression.

In Beck's cognitive theory of depression (Beck, 1967, 1987; Beck, Rush, Shaw, & Emery, 1979), negative self-schemata involving themes of inadequacy, failure, loss, and worthlessness are hypothesized to contribute vulnerability to depression. These negative self-schemata are often represented as a set of dysfunctional attitudes, such as "If I fail partly, it is as bad as being a complete failure" or "I am nothing if a person I love doesn't love me." When people with such dysfunctional attitudes encounter negative life events, they are hypothesized to develop negatively biased perceptions of their self (low self-esteem), world, and future (hopelessness), which then lead to depressive symptoms. Although hopelessness theory and Beck's theory differ in terms of some of their specifics, both hypothesize that cognitive vulnerability operates to increase risk for depression through its effects on processing or appraisals of personally relevant life experiences. Despite this similarity, however, studies have suggested that the negative attributional style component of cognitive vulnerability as defined by the hopelessness theory and the dysfunctional attitude component of Beck's theory do represent distinct constructs (e.g., Gotlib, Lewinsohn, Seeley, Rohde, & Redner, 1993; Haefeli et al., 2003; Joiner & Rudd, 1996; Spangler, Simons, Monroe, & Thase, 1997).

A powerful strategy for testing these cognitive vulnerability hypotheses is the behavioral high risk design (e.g., Alloy, Lipman, & Abramson, 1992; DePue et al., 1981). Similar to the genetic high risk design, the behavioral high risk design involves studying individuals hypothesized to be at high or low risk for developing a particular disorder, but who do not currently have one. In a behavioral high risk design, however, individuals are selected based on hypothesized psychological, rather than genetic, vulnerability or invulnerability to the disorder. For example, in testing the cognitive theories of depression, researchers would want to select nondepressed individuals who either have or do not have the hypothesized depressogenic cognitive styles. These groups of cognitively high and low risk individuals can then be compared with respect to their likelihood of having had past occurrences of depression (retrospective design) and their likelihood of experiencing depression in the future (prospective design).

Studies using or approximating a behavioral high risk design have provided substantial support for the cognitive theories of depression. For example, Alloy et al. (1992) utilized a retrospective behavioral high risk design to test the attributional vulnerability hypothesis of the hopelessness theory. They examined the occurrence of major depressive disorder (MD) and HD during the previous 2 years in currently nondepressed undergraduates who either did or did not exhibit attributional vulnerability for depression (indicated by an internal, stable, and global attributional style for negative events). Consistent with the hopelessness theory, they found that attributionally vulnerable students were more likely to exhibit past MD and HD, experienced more episodes, and experienced more severe episodes of these disorders than attributionally invulnerable students. In addition, several other studies approximating a prospective behavioral high risk design have reported that people with negative cognitive styles are more likely to develop depressive moods or symptoms when they experience negative life events than are individuals without such negative styles (e.g., Alloy & Clements, 1998; Alloy, Just, & Panzarella, 1997; Metalsky, Halberstadt, & Abramson, 1987; Metalsky & Joiner, 1992; Metalsky, Joiner, Hardin, & Abramson, 1993; Nolen-Hoeksema, Girgus, & Seligman, 1986, 1992).

These positive results stand in contrast to those found when utilizing typical "remitted depression" designs, which generally have found little support for the cognitive vulnerability hypotheses (e.g., Burnett & Gotlib, 1988; Persons & Miranda, 1992; Segal & Ingram, 1994). In these studies, the cognitive styles of individuals who have recovered from depressive episodes are compared to the cognitive styles of individuals with no history of depression. However, there are several problems with using a remitted depression design to test cognitive vulnerability hypotheses (see Alloy, Abramson, & Just, 1995; Just, Abramson, & Alloy, 2001). For example, depressed individuals are a heterogeneous group and the cognitive theories of depression seek to account for only a subgroup of depressive
(i.e., only those with a cognitively mediated subtype of depression). Given that only a subset of such previously depressed individuals are likely to have had a cognitively mediated depression, such heterogeneity can result in equivocal findings when comparing a group of remitted depressed individuals to nondepressed individuals, some of whom may also have a cognitive vulnerability, but have not yet had a depressive episode.

Therefore, in keeping with the suggested methodology already presented, the Temple–Wisconsin Cognitive Vulnerability to Depression (CVD) project uses a prospective behavioral high risk design to test the cognitive vulnerability and other etiological hypotheses of the hopelessness theory and Beck’s theory of depression. The CVD project is a collaborative, two-site study that assesses, among other factors, individual’s cognitive styles, the occurrence of negative life events, and the occurrence of both depressive symptoms and clinically significant depressive episodes. This chapter reviews the major findings to date from the CVD project.

CVD Project Design

Participant Selection

Participants were selected for inclusion in the CVD project via a two-phase screening procedure. In the first phase, 5,378 freshmen (2,438 at Temple University, TU, and 2,940 at the University of Wisconsin–Madison, UW) completed two measures of cognitive style: the Cognitive Style Questionnaire (CSQ; Alloy et al., 2000), a modified version of the Attributional Style Questionnaire (ASQ; Peterson et al., 1982), which assesses individuals’ styles for inferring causes, consequences, and self-characteristics following the occurrence of positive and negative events, and a modified version of the Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978). The primary modifications made to the ASQ in designing the CSQ were that more hypothetical events were included (12 positive and 12 negative events), the hypothetical events were changed to more adequately reflect life events likely to be faced by college students, and the dimensions of consequences and self-characteristics are assessed in addition to the attributional dimensions of internality, stability, and globality. The DAS was modified by adding an additional 24 items that specifically assess dysfunctional beliefs in the achievement and interpersonal domains.

Individuals scoring in the highest (most negative) or lowest (most positive) quartile on both the DAS and the CSQ composite (stability + globality + consequences + self) for negative events were designated at high (HR) and low (LR) cognitive risk for depression, respectively (for more details, see Alloy & Abramson, 1999; Alloy et al., 2000). Thus, participants in the CVD project were selected based on the presence versus absence of vulnerability to depression as specified by both the hopelessness theory (Abramson et al., 1989) and Beck’s (1967, 1987) theory.

In the second phase of the screening process, a randomly selected subsample of HR and LR participants, who were under age 30, were administered an expanded version of the Schedule for Affective Disorders and Schizophrenia–Lifetime (SADS–L) diagnostic interview (Endicott & Spitzer, 1978). The SADS–L was expanded to allow for the Diagnostic and Statistical Manual (DSM–III–R; American Psychiatric Association, 1987), as well as Research Diagnostic Criteria (RDC; Spitzer, Endicott, & Robins, 1978) diagnoses, and the data were also recoded according to DSM–IV (American Psychiatric Association, 1994). Individuals were excluded from participation in the study if they exhibited any current Axis I disorder, current psychotic symptoms, past history of any bipolar-spectrum disorder, or any serious medical illness that would preclude participation in a longitudinal study. Participants who had a past unipolar mood disorder but had remitted for a minimum of 2 months were retained so as not to result in an unrepresentative sample of HR participants. More specifically, including only those HR participants with no prior history of depressive episodes may have yielded an unrepresentative group of HR participants (e.g., those who exhibit other protective factors, such as strong social support, that warded off the onset of depression). The final CVD project sample included 173 HR (83 at TU, 90 at UW) and 176 LR (87 at TU, 89 at UW) participants. Demographic and cognitive style characteristics of the final sample are presented in Table 2.1 (see Alloy & Abramson, 1999, and Alloy et al., 2000, for more details on the final sample’s characteristics and representativeness).

Project Assessments

After agreeing to participate in the study, all participants completed a Time 1 assessment that included measures of Axis II personality disorders and dimensions (Personality Disorders Examination, PDE; Loranger, 1988), self-referent information processing (SRIP Task Battery; Alloy, Abramson, Murray, Whitehouse, & Hogan, 1997), cognitive styles (CSQ, DAS, sociotropy-autonomy, self-consciousness), coping styles (rumination vs. distraction), social support, negative life events (with a combination questionnaire and semi-structured interview modeled after Brown and Harris, 1978), and hypothesized mediating cognitions (inferences for actual events and negative views of self, world, and future). After completing the Time 1 assessment, participants were followed longitudinally for 3½ years. For the first 2½ years of the follow-up, participants completed interview and questionnaire assessments every 6 weeks. For the remaining 3 years of the fol-
low-up, participants were interviewed and completed questionnaires every 4 months. During each assessment, questionnaires and interviews were used to assess the occurrence of negative life events, inferences for these events, the components of Beck’s (1967, 1987) negative cognitive triad (negative view of self, world, and future), coping styles, social support, and the onset and offset of symptoms and DSM-III-R and RDC episodes of depression and other psychopathology. Data from these assessments were also used to assess the onset and offset of symptoms and diagnoses of HD (see Table 2.2 for HD diagnostic criteria).

At the end of each year of follow-up, participants completed measures to reassess their inferential styles and dysfunctional attitudes, as well as their coping styles and self-referent information processing. Further, during the first 2½ years of follow-up, participants and their parents completed a number of measures assessing parents’ history of psychopathology, parents’ cognitive styles, inferential feedback, and parenting styles, as well as participants’ childhood life events and reports of childhood maltreatment. Finally, at the end of the 5½-year follow-up, participants completed a second PDE. For further details about the rationale, design, and methodology of the CVD project, see Alloy and Abramson (1999). Given that the majority of the CVD project data from the second 2½ years of follow-up have not yet been analyzed, this chapter focuses primarily on findings so far from the first 2½ years of follow-up.

CVD PROJECT FINDINGS

Do Negative Cognitive Styles Confer Vulnerability to Depression?

A primary hypothesis of the cognitive theories of depression is that certain negative cognitive styles confer vulnerability to symptoms and diagnoses of depression. Although cognitive styles are not immutable (Just et al., 2001) and are open to modification (e.g., through cognitive therapy; see DeRubeis & Hollon, 1995), these styles are typically viewed as relatively stable risk factors. Findings from the CVD project have supported the relative stability of cognitive styles. Specifically, the cognitive styles of our participants remained stable from before to during and after intervening episodes of major depression (Berreth, Alloy, & Abramson, 2004). In addition, participants’ attributions and inferences for particular negative life events they experienced remained stable over the 5-year follow-up (Raniere, 2000). Thus, cognitive styles appear to be a relatively traitlike vulnerability factor.

One method of testing the cognitive theories’ vulnerability hypothesis is to examine whether individuals who exhibit negative cognitive styles are more likely to have a history of depression than are individuals with positive cognitive styles. Thus, in the CVD project, HR participants were expected to have higher lifetime prevalence rates of episodic mood disorders (i.e., MD, minor depression [MiD], and HD) than were LR partici-
### TABLE 2.3
Lifetime Prevalence of Depressive and Other Disorders as a Function of Cognitive Risk Controlling for Age, Phase II Screening BDI Scores, and SADS-L Current Depressive Symptom Scores

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Low Risk % (n = 176)</th>
<th>High Risk % (n = 173)</th>
<th>(\Delta R^2)</th>
<th>OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major depression</td>
<td>17.0</td>
<td>38.7</td>
<td>9.48***</td>
<td>.025</td>
</tr>
<tr>
<td>(DSM-III-R or RDC)</td>
<td>(n = 50)</td>
<td>(n = 67)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Minor depression</td>
<td>11.9</td>
<td>22.0</td>
<td>3.03*</td>
<td>.008</td>
</tr>
<tr>
<td>(DSM-III-R or RDC)</td>
<td>(n = 21)</td>
<td>(n = 38)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hopelessness depression</td>
<td>11.9</td>
<td>39.9</td>
<td>22.41****</td>
<td>.059</td>
</tr>
<tr>
<td>(Project Diagnosis)</td>
<td>(n = 21)</td>
<td>(n = 69)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dysthymic disorder</td>
<td>2.3</td>
<td>3.5</td>
<td>0.37</td>
<td>.001</td>
</tr>
<tr>
<td>(DSM-III-R)</td>
<td>(n = 4)</td>
<td>(n = 6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intermittent dysthymic disorder</td>
<td>2.3</td>
<td>4.0</td>
<td>0.37</td>
<td>.001</td>
</tr>
<tr>
<td>(RDC)</td>
<td>(n = 4)</td>
<td>(n = 7)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression NOS</td>
<td>5.4</td>
<td>6.4</td>
<td>1.19</td>
<td>.003</td>
</tr>
<tr>
<td>(DSM-III-R)</td>
<td>(n = 5)</td>
<td>(n = 11)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Labile personality</td>
<td>1.1</td>
<td>8.1</td>
<td>0.75</td>
<td>.002</td>
</tr>
<tr>
<td>(RDC)</td>
<td>(n = 2)</td>
<td>(n = 14)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subaffective dysthymic disorder</td>
<td>0.0</td>
<td>3.5</td>
<td>1.55</td>
<td>.004</td>
</tr>
<tr>
<td>(RDC)</td>
<td>(n = 0)</td>
<td>(n = 6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any anxiety disorder</td>
<td>7.4</td>
<td>12.1</td>
<td>0.07</td>
<td>.000</td>
</tr>
<tr>
<td>(DSM-III-R or RDC)</td>
<td>(n = 13)</td>
<td>(n = 2)</td>
<td>(0.85-3.63)</td>
<td></td>
</tr>
<tr>
<td>Any substance use disorder</td>
<td>8.5</td>
<td>8.7</td>
<td>0.00</td>
<td>.000</td>
</tr>
<tr>
<td>(DSM-III-R or RDC)</td>
<td>(n = 15)</td>
<td>(n = 15)</td>
<td>(0.49-2.18)</td>
<td></td>
</tr>
<tr>
<td>Other psychiatric disorder</td>
<td>4.0</td>
<td>2.3</td>
<td>0.73</td>
<td>.002</td>
</tr>
<tr>
<td>(RDC)</td>
<td>(n = 7)</td>
<td>(n = 4)</td>
<td>(0.17-2.01)</td>
<td></td>
</tr>
</tbody>
</table>

Note: BDI = Beck Depression Inventory; SADS-L = Schedule for Affective Disorders and Schizophrenia-Lifetime Interview; OR = Odds Ratio; CI = Confidence Interval. Degrees of freedom are 1, 336 for major depression, hopelessness depression, labile personality, subaffective dysthymic disorder, any anxiety disorder, and other psychiatric disorder. Degrees of freedom are 1, 1,032 for all other disorders.

\*p < .05, \**p < .01, \***p < .001


Participants. Controlling for current levels of depressive symptoms, HR participants did indeed exhibit higher lifetime rates of DSM-III-R and RDC MD and HD than did LR participants (Alloy et al., 2000), as well as marginally higher lifetime rates of RDC MD (see Table 2.3). In fact, HR participants were approximately three times more likely to have experienced MD and almost five times more likely to have experienced HD than were LR participants. The HR-LR differences in lifetime prevalence rates of MD and HD were maintained when other hypothesized risk factors for depression were controlled (i.e., inferential style for positive events, sociotropy, autonomy, self-consciousness, stress-reactive rumination). Interestingly, the risk groups did not differ in lifetime rates of nondepressed mood disorders (i.e., DSM-III-R dysthymic disorder or RDC intermittent depressive disorder). Supporting the specificity of cognitive vulnerability to the depressive disorders, there were also no risk group differences in participants' lifetime histories of anxiety disorders, substance use disorders, or other psychiatric disorders. Following up on these findings, Haefel et al. (2003) used an unselected sample of undergraduates to "unpack" the generic cognitive vulnerability of the CVD project. Haefel et al. found that negative inferential styles, but not dysfunctional attitudes, uniquely predicted lifetime history of clinically significant depressive episodes and anxiety comorbidity with depression.

Despite the strengths of these findings, they do not adequately address whether negative cognitive styles serve as a vulnerability factor for depression, because the findings are equally supportive of the alternate hypothesis that negative cognitive styles are a consequence or "scar" left by the past experience of depression (see Lewinsohn, Steinmetz, Larson, & Franklin, 1981). Therefore, to adequately test the cognitive vulnerability hypothesis, data from the prospective portion of the CVD project are required. Results from the first 2½ years of follow-up in the CVD project indicate that risk group status predicted both first onsets and recurrences of both MD and HD during this time period (Alloy, Abramson, Whitehouse, et al., in press). Specifically, among individuals with no prior history of depression, HR participants were more likely than were LR participants to experience a first onset of MD, MID, and HD (see Table 2.4). These findings provide especially important support for the cognitive vulnerability hypothesis because they are based on a truly prospective test, uncontaminated by prior history of depression. In addition, among individuals with a past history of depression, HR participants were more likely to experience recurrences of MD, MID, and HD than were LR participants (see Table 2.4). Similar to the results of the retrospective analyses, there were no risk group differences in either first onsets or recurrences of anxiety disorders or other disorders. However, in the full sample, HR participants were more likely than LR participants to have an onset of anxiety disorder comorbid with depression, but not an anxiety disorder alone. Further, each of these results was maintained even after statistically controlling for participants' initial levels of depressive symptoms upon entering the study, as assessed by the Beck Depression Inventory (BDI; Beck et al., 1979).

In addition to contributing vulnerability to depression, the cognitive theories hypothesize that negative cognitive styles should confer risk for suicidality, ranging from suicidal ideation to completed suicides, and that
2. COGNITIVE VULNERABILITY TO DEPRESSION PROJECT

Table 2.4
CVD Project Prospective Rates of First Onsets and Recurrences of Depressive and Anxiety Disorders

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Low Risk</th>
<th>High Risk</th>
<th>OR</th>
<th>95% CI</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prospective Rates of Depressive and Anxiety Disorders: First Onsets (Subsample With No Prior Depression)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any major depression</td>
<td>2.7%</td>
<td>16.2%</td>
<td>7.4</td>
<td>1.6-34.8</td>
<td>.01</td>
</tr>
<tr>
<td>RDC minor depression</td>
<td>14.4%</td>
<td>45.9%</td>
<td>5.6</td>
<td>2.2-14.1</td>
<td>.001</td>
</tr>
<tr>
<td>Any episodic depression</td>
<td>16.2%</td>
<td>45.9%</td>
<td>4.9</td>
<td>2.6-12.2</td>
<td>.001</td>
</tr>
<tr>
<td>Hopelessness depression</td>
<td>3.6%</td>
<td>35.1%</td>
<td>11.6</td>
<td>3.3-41.3</td>
<td>.001</td>
</tr>
<tr>
<td>Any anxiety disorder</td>
<td>0.9%</td>
<td>6.8%</td>
<td>9.3</td>
<td>0.8-113.3</td>
<td>.09</td>
</tr>
<tr>
<td>Prospective Rates of Depressive and Anxiety Disorders: Recurrences (Subsample With Prior Depression)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any major depression</td>
<td>9.4%</td>
<td>28.6%</td>
<td>3.8</td>
<td>1.3-11.0</td>
<td>.02</td>
</tr>
<tr>
<td>RDC minor depression</td>
<td>32.8%</td>
<td>56.1%</td>
<td>3.1</td>
<td>1.4-7.0</td>
<td>.02</td>
</tr>
<tr>
<td>Any episodic depression</td>
<td>34.4%</td>
<td>62.2%</td>
<td>4.4</td>
<td>1.9-10.1</td>
<td>.001</td>
</tr>
<tr>
<td>Hopelessness depression</td>
<td>18.8%</td>
<td>50.0%</td>
<td>4.1</td>
<td>1.7-10.0</td>
<td>.002</td>
</tr>
<tr>
<td>Any anxiety disorder</td>
<td>4.7%</td>
<td>10.2%</td>
<td>4.0</td>
<td>0.8-21.5</td>
<td>.11</td>
</tr>
</tbody>
</table>

depressive disorders and suicidality. The results also provide support for the hypothesis that the subtype of HD exists in nature and conforms to theoretical description.

SPECIFICITY OF NEGATIVE COGNITIVE STYLES TO HOPELESSNESS DEPRESSION

The hopelessness theory (Abramson et al., 1989) proposes that negative cognitive styles confer vulnerability to HD, specifically, rather than to other subtypes of depression. Supporting this hypothesis, studies have found that negative cognitive styles, both alone and interacting with negative life events, are more strongly related to depressive symptoms hypothesized to be part of the HD symptom cluster (see Table 2.2) than to symptoms not part of the HD symptom cluster (Alloy & Clements, 1998; Alloy et al., 1997; Hankin, Abramson, & Siler, 2001; Joiner et al., 2001; Metalsky & Joiner, 1997) or to symptoms of other forms of psychopathology (Alloy & Clements, 1998). In addition, preliminary analyses based on the first 2½ years of prospective follow-up in the CVD project indicated that cognitive risk predicted first onsets and recurrences of HD (as described earlier), but not DSM melancholic depression.

RUMINATION AS A MEDIATOR AND MODERATOR OF COGNITIVE VULNERABILITY TO DEPRESSION

According to the response styles theory of depression (Nolen-Hoeksema, 1991), individuals who tend to ruminate in response to dysphoria will be at increased risk for experiencing more severe and prolonged depressions than will individuals who tend to distract themselves from their dysphoria. Rumination refers to "behaviors and thoughts that focus one’s attention on one’s depressive symptoms and on the implications of these symptoms" (Nolen-Hoeksema, 1991, p. 569), whereas distraction refers to active attempts to ignore depressive symptoms by focusing on pleasant or neutral activities. Several studies have found support for this theory, demonstrating that rumination is associated with a greater likelihood of major depression and longer and more severe episodes of depression (e.g., Just & Alloy, 1997; Morrow & Nolen-Hoeksema, 1990; Nolen-Hoeksema, 2000; Nolen-Hoeksema & Morrow, 1991; Nolen-Hoeksema, Morrow, & Frederickson, 1993; Nolen-Hoeksema, Parker, & Larson, 1994; Spasesjewic & Alloy, 2001). Abramson et al. (2002) described the expected relation between the cognitive vulnerabilities featured in hopelessness and Beck’s theories and rumination and hypothesized that rumination would mediate the ef-
fects of these cognitive vulnerabilities on the prospective development of depressive episodes. Consistent with this hypothesis, Spasojevic and Alloy (2001) found that a ruminative response style measured at Time 1 of the CVD project mediated the association between cognitive risk status and the development of prospective episodes of MD. Ruminati on also mediated the effects of other risk factors (past history of depression, maladaptive dependency, and self-criticism) for MD onset during the follow-up period.

Expanding on the response styles theory, Robinson and Alloy (2003) hypothesized that individuals who have negative inferential styles and, additionally, who tend to ruminate about these negative cognitions in response to the occurrence of stressful life events (stress-reactive rumination), may be more likely to develop episodes of depression in the first place. The idea is that negative cognitive styles provide the negative content, but this negative content may be more likely to lead to depression when it is "on one's mind" than when it is not. Accordingly, Robinson and Alloy proposed that stress-reactive rumination would exacerbate the association between negative cognitive styles and the onset of a depressive episode. Consistent with this hypothesis, using CVD project data, they found that stress-reactive rumination when assessed at Time 1 interacted with cognitive risk to predict prospective onsets of MD and HD episodes (see Fig. 2.1). Among the cognitively LR participants, there was no difference found in the likelihood of future onset of depression based on whether or not such individuals tended to evidence stress-reactive rumination. On the other hand, among the cognitively HR participants, individuals who were also high in stress-reactive rumination evidenced a higher prospective incidence of MD and HD than high risk individuals who did not tend to ruminate in response to stressors.

COGNITIVE VULNERABILITY-STRESS INTERACTION AND PROSPECTIVE DEVELOPMENT OF DEPRESSION

Given that the cognitive theories of depression are vulnerability-stress models in which depressogenic cognitive styles are proposed to confer vulnerability to depression when individuals confront negative life events, it is important to evaluate the interaction between cognitive style and the occurrence of negative life events in predicting onset/recurrence of depression. As indicated earlier, several previous studies have found support for the vulnerability-stress hypothesis (Alloy & Clements, 1998; Alloy et al., 1997; Anderson, 1990; Metalsky et al., 1987, 1993; Metalsky & Joiner, 1992; Nolen-Hoeksema et al., 1986, 1992; Panak & Garber, 1992;
Robinson, Garber, & Hilsman, 1995) in predicting depressive symptoms. However, there have been a handful of published studies that have found no support for this hypothesis (Cole & Turner, 1993; Joiner & Wagner, 1995; Tiggemann, Winifield, Winifield, & Goldney, 1991).

To date, little evaluation of the vulnerability-stress hypothesis has been conducted with the CVD project data. Preliminary investigation of this hypothesis using the Temple site data only, in which negative life events were coded as loss and/or danger events using the criterial definitions of Finlay-Jones and Brown (1981), revealed that negative cognitive style, loss events, and danger events all significantly predicted the occurrence of depressive episodes (Gifford & Alloy, 1999). However, there were no vulnerability-stress interactions found for episodes of either MD or HD. This particular study evaluated the effects of cognitive style and life events on the first depressive episode experienced during the course of the CVD project, controlling for past history of depression. Further analyses are needed to evaluate if this lack of significant findings is true when both sites’ data are used and for all negative life events or just for these two subgroups of life events. In a second preliminary evaluation of the occurrence of depressive episodes across the first 2½ years of follow-up in the CVD project (Robinson, 1997), also using Temple site data only, marginally significant to significant relations were found between the cognitive style × number of negative life events interaction and number of episodes of any depressive disorder, including both minor and major episodes. A third preliminary study of the occurrence of MD and HD episodes across the first 9 months of follow-up in the CVD project using only Temple site data (Panzarella, Alloy, & Whitehouse, in press) found that the interaction between cognitive risk and the number of negative life events predicted onsets of both MD and HD. Moreover, the cognitive vulnerability-stress interaction was further moderated by social support. HR participants with high stress and poor social support had a higher likelihood of developing a MD or HD episode than participants with 0, 1, or 2 of the 3 risk factors. As such, more definitive work remains to be done to evaluate the validity of the vulnerability-stress hypothesis in predicting depressive episodes in the CVD project data from both sites.

Characteristics of Cognitively Vulnerable Individuals

Self-Referent Information Processing. The cognitive theories of depression hypothesize that individuals who are cognitively vulnerable to depression tend to process information about themselves in a negatively biased manner. For example, Beck (1967, 1987) hypothesized that certain individuals possess negative self-schemata that negatively color their perception, interpretation, and memory of personally relevant experiences. Similarly, the hopelessness theory (Abramson et al., 1989) proposes that individuals with a negative cognitive style tend to infer negative self-characteristics following the occurrence of negative life events.

Data from the Self-Referent Information Processing (SRIP) Task Battery, administered at the Time 1 assessment of the CVD project, were used to test these hypotheses. Given Beck’s hypothesis that individuals with a negative self-schema should demonstrate biased information processing only for depression-relevant stimuli (i.e., stimuli related to themes of incompetence, worthlessness, and low motivation), Alloy et al. (1997) predicted that HR participants would demonstrate information-processing biases for depression-relevant, but not depression-irrelevant, self-referent adjectives. Partial support was obtained for this hypothesis. Specifically, as predicted, compared to LR participants, HR participants showed preferential self-referent processing compared to LR participants for depression-relevant material (e.g., words like “failure,” “passive,” and “useless”) as evidenced by relatively greater endorsement, faster processing, greater accessibility, better recall, and higher predictive certainty of this material. In addition, HR participants were less likely to process positive depression-relevant stimuli (e.g., words such as “resourceful,” “energetic,” and “important”) than were LR participants. Finally, although contrary to prediction, there were risk group differences for the depression-irrelevant material on two of the tasks. Specifically, LR participants were more likely than HR participants to judge positive depression-irrelevant stimuli as self-descriptive (e.g., words like “thoughtful”) and believed they were more likely to engage in future positive depression-irrelevant behaviors (e.g., giving up a seat on a bus for an elderly lady). However, the group differences were larger for depression-relevant than for depression-irrelevant stimuli even on these tasks. Importantly, all of the risk group differences were maintained even at postparticipation levels of depressive symptoms were statistically controlled. These findings are unique in demonstrating that the information-processing biases previously demonstrated in depressed individuals (see Ingram, Miranda, & Segal, 1998; Segal, 1988) also extend to nondepressed individuals at high cognitive risk for depression.

We also examined whether the negative self-referent processing exhibited by HR individuals mediated or moderated the predictive association between cognitive risk and prospective onsets of depressive episodes (Steinberg, Oelrich, Alloy, & Abramson, 2004). A composite of the five dependent measures from the SRIP Task Battery partially mediated the cognitive risk effects for prediction of HD episodes, but not MD or MDD episodes. This finding is interesting because HD is hypothesized specifically to be a cognitively mediated subtype of depression. In addition, the negative SRIP composite interacted with cognitive risk to predict first onsets,
but not recurrences, of MD and HD. Individuals who both exhibited negative cognitive styles and negative information processing about themselves were at increased risk for a first onset of depression compared to individuals with either of these risk factors alone.

**Cognitive Vulnerability and Personality Characteristics.** In addition to evaluating negative information processing about the self in individuals prone to depression, it is also important to evaluate the relation between cognitive vulnerability to depression and other personality characteristics and disorders. For example, it has been proposed that individuals with negative cognitive styles might be at increased risk for Axis II personality dysfunction (Smith et al., 2004). In support of this hypothesis, previous studies have indicated that comorbidity between depression and personality disorders is high, ranging from 30% to 70% (see Farmer & Nelson-Gray, 1990). In addition, depressed inpatients with comorbid personality disorders, especially borderline personality disorder, have been found to be more likely to exhibit negative cognitive styles than are depressed inpatients without comorbid personality disorders (Rose,Abramson, Hodulik, Halberstadt, & Leff, 1994). Finally, many personality disorders are partially defined by cognitive patterns that are consistent with definitions of depressogenic cognitive style (Beck et al., 1990). For example, the Cluster C personality disorders (Avoidant, Dependent, and Obsessive-Compulsive) were found to be associated with feelings of incompetence, helplessness, and weakness.

Although the relative specificity of depressogenic cognitive styles has been demonstrated for Axis I psychopathology (e.g., Alloy et al., 2000; Alloy, Abramson, Whitehouse, et al., in press), it has been proposed that such cognitive specificity would not be likely to occur for Axis II personality disorders (Smith et al., 2004). This hypothesized nonspecificity is due to the fact that personality disorders are frequently comorbid with each other and all three of the personality clusters have been associated with depression (Farmer & Nelson-Gray, 1990). In addition, all of the personality clusters are associated with cognitive, behavioral, and interpersonal characteristics consistent with those likely to be found in individuals who are cognitively vulnerable to depression (Smith et al., 2004). In contrast to this proposed nonspecificity between negative cognitive styles and the various personality disorders, there is some evidence that negative cognitive style and HD may be more specifically related to borderline and dependent personality functioning than to other personality disorders (Akhavan, 2000).

To further examine the relation between cognitive style and personality dysfunction, the Personality Disorder Examination (PDE; Loranger, 1988) was administered to all participants at the beginning and end of the CVD project. The PDE interview provides DSM-III-R categorical personality disorder diagnoses as well as dimensional scores for each disorder. In analyses of personality dysfunction, as assessed by the PDE at Time 1 of the CVD project, the cognitively high risk group had significantly more diagnosable personality disorders than the low risk group (5.4 vs. 1.7%; Smith et al., 2004). Although these percentages are low for both groups, it must be kept in mind that participants in the CVD project were quite young (mean age = 19 years) and, therefore, relatively unlikely to have full-blown personality disorder. For example, Loranger (1988) indicated that it is difficult to accurately diagnose personality disorder in individuals younger than age 25. Therefore, the fact that HR individuals were more than twice as likely to have a diagnosable personality disorder at such a young age is a significant finding. In addition, although there were not enough participants with diagnosable personality disorders to examine the rates of each personality disorder category separately, all three personality clusters were represented in those diagnosed.

When evaluating personality dysfunction using the dimensional scores, the HR group was rated higher than the LR group for Cluster A paranoid and schizotypal dimensions, Cluster B borderline, histrionic, and narcissistic dimensions, and Cluster C avoidant, dependent, obsessive-compulsive, and passive-aggressive dimensions. Schizoid, antisocial, and sadistic personality dysfunction were the only personality dimensions on which no significant risk group differences were found. The strength of these findings is bolstered by the fact that, except for the narcissistic, passive-aggressive, and self-defeating dimensions, these HR-LR differences remained after statistically controlling for the participants’ depressive symptom levels, based on their BDI scores. In addition, the risk group differences in the lifetime prevalence of episodic unipolar depressive disorders (major, minor, and HD) reported by Alloy et al. (2000) remained after statistically controlling for the effects of personality dysfunction. Therefore, although an association exists between negative cognitive style and personality disorder, personality dysfunction does not appear to be a sole mediator of the relation between cognitive vulnerability and depression.

Aside from DSM personality dysfunction, other personality characteristics have been linked to depression and deserve attention in regard to cognitive vulnerability for depression as well. For example, sociotropy and autonomy (Beck, 1983) represent two personality subtypes believed to confer vulnerability to depression when an individual experiences negative life events that are congruent with these personality traits. Sociotropy is believed to be a personality style characterized by concern about interpersonal relatedness and fear of rejection or abandonment. Individuals exhibiting this personality style are hypothesized to be prone to depression.
when they experience interpersonal stresses and losses, such as the breakup of a relationship or a fight with a friend. On the other hand, autonomy is believed to be a personality style characterized by concern for achievement, independence, and self-definition. Autonomous individuals are hypothesized to be at risk for depression when they experience failure to achieve goals they set for themselves, such as receiving a poor grade in school, or failing to get a promotion at work.

To date, there has been little published research on the relation between depressogenic cognitive styles and these two personality subtypes. In some analyses of the CVD project data, this relation was examined (Abramson, Alloy, & Hogan, 1997). HR participants showed greater sociopathy than did LR participants, even after controlling for their current level of depression. However, the opposite was found to be true with regard to autonomy. Specifically, HR individuals showed a trend toward less autonomy than LR individuals both before and after controlling for current depression levels. This suggests that HR individuals should be more prone to sociotropic, but not autonomous, depressions.

The aforementioned relations between cognitive vulnerability for depression and various personality dysfunction, disorders, and subtypes represent an important contribution to the continued evaluation and expansion of cognitive theories of depression. By incorporating an examination of personality and interpersonal functioning, these findings extend the growing body of research investigating the cognitive-behavioral-interpersonal configurations that confer risk for depression (e.g., Alloy, Fedderly, Kennedy-Moore, & Cohart, 1998; Gotlib & Hammen, 1992; Joiner, Alfano, & Metalsky, 1992; Panzarella et al., in press; Segrin & Abramson, 1994).

Developmental Antecedents of Cognitive Vulnerability to Depression

Evidence shows that negative cognitive styles do indeed confer vulnerability to future episodes of both depression and suicidality, so it is important to understand factors that may contribute to the development of such styles. Data from the CVD project allow an initial examination of several factors that may contribute to the development of these cognitive styles (see Alloy et al., 2004, for an in-depth review of the developmental findings from the CVD project). As part of the CVD project, we assessed the cognitive styles and lifetime history of psychopathology of 335 of our participants’ parents (217 mothers, 118 fathers). In addition, participants and their parents were asked to report the parents’ inferential feedback styles and parenting styles. Finally, participants’ reports of childhood maltreatment were assessed.

2. COGNITIVE VULNERABILITY TO DEPRESSION PROJECT

Parental Psychopathology. Given previous research suggesting that children of depressed parents are at increased risk for the development of negative attributional styles (e.g., Garber & Flynn, 2001; Hammen, 1992) and episodes of depression (e.g., Downey & Coyle, 1993), data from the CVD project were used to examine the relation between participants’ cognitive risk status and their parents’ history of depression. Parental history of depression was assessed using the reports of our participants (i.e., family history RDC method; Andreasen, Endicott, Spitzer, & Winokur, 1977), and from direct interviews with the parents themselves, using the expanded SADS-L.

Preliminary data from the CVD project suggest that there was a relation between participants’ cognitive risk group status and their parents’ histories of depression (Abramson et al., 2004). This relation, however, appears to be stronger for mothers’ than for fathers’ histories of depression. Specifically, HR participants, compared to LR participants, reported that their mothers were significantly more likely, and their fathers were marginally more likely, to have a past history of depression. In the direct interviews of the parents, mothers of HR participants were more likely to have had a past history of depression than were mothers of LR participants. There were no group differences, however, for fathers’ histories of depression. These findings are unique in demonstrating a relation between parents’ histories of depression and the cognitive styles of nondepressed individuals, and provide support for explorations of possible mediators of the association between parental depression and offspring’s cognitive vulnerability to depression.

Modeling and Parental Inferential Feedback. Parents may influence the cognitive styles of their children through modeling their own negative cognitive style or by providing negative inferential feedback regarding the causes and consequences of negative events in their children’s lives. However, studies have provided only limited support for a direct relation between parents’ and their children’s negative cognitive styles. For example, in the CVD project, the mothers of HR participants had more dysfunctional attitudes than did mothers of LR participants, even after controlling for the mothers’ levels of depressive symptoms (Alloy et al., 2001). In contrast, there were no risk group differences in mothers’ or fathers’ inferential styles or in fathers’ dysfunctional attitudes. In another study, however, third, fourth, and fifth graders’ attributional styles were significantly related to those of their mothers, but not their fathers (Seligman et al., 1984). Finally, in a third study, no relation was found between sixth graders’ attributional styles and those of their mothers (Garber & Flynn, 2001). Thus, although there is some evidence that children may model the cognitive styles of their parents, especially their mothers, future studies are
neither parents nor their children is driven by the belief that one can control one's emotional reactions. However, there are exceptions to this rule, and these exceptions may be related to the children's cognitive development. For example, children who are overprotected by their mothers and fathers may develop a sense of helplessness because they are unable to control their emotions. In contrast, children who are exposed to frequent negative events, such as parental rejection, may develop a sense of hopelessness because they believe that they are unable to control their emotional reactions. These differences in cognitive development may be related to the children's ability to control their emotions, which may be affected by their experiences and the environment in which they are raised.

Parenting Styles. Studies have suggested that certain parenting styles may also contribute to the development of a negative cognitive style in children. For example, both HR participants in the CVD project and their fathers reported that the fathers exhibited less warmth and acceptance (and more rejection) than did fathers of LR participants (Alloy et al., 2001). There were no group differences, however, for fathers' levels of either psychological autonomy versus control or firm versus lax control (discipline), nor were there any group differences in the parenting styles of mothers. Fathers' acceptance scores also predicted prospective onsets of MD, MiD, and HD episodes in their children, but only the prediction of HD episodes was mediated by the children's cognitive risk status (Alloy et al., 2001). In a longitudinal study of sixth graders and their mothers, higher levels of maternal psychological control were associated with increasing negativity of their children's attributional styles over a 1-year follow-up, even after controlling for the mothers' histories of mood disorders (Garber & Flynn, 2001). In this study, neither parental acceptance versus rejection nor firm versus lax control were related to changes in the children's attributional styles. Finally, undergraduates with a negative cognitive style reported less maternal care when growing up than did undergraduates with a positive cognitive style (Whisman & Kwon, 1992). In this study, undergraduates' cognitive styles were not related to the degree of maternal overprotection reported during childhood.

Thus, although these studies suggest a relation between certain parenting styles and children's cognitive styles, they do not agree as to which parenting styles are the most detrimental. In addition, only one study (Alloy et al., 2001) from the CVD project has examined the parenting practices of fathers. Future studies, therefore, should seek to clarify the relation between parenting practices and children's negative cognitive styles and should seek to include fathers in this evaluation.

Childhood Maltreatment. In extending the etiological chain of the hopelessness theory, Rose and Abramson (1992) proposed a developmental pathway by which negative life events, especially childhood maltreatment, may lead to the development of a negative cognitive style. Specifically, they suggested that when maltreatment occurs, individuals attempt to understand the cause, consequences, and meanings of the abuse so that future negative events may be avoided and hopelessness may be maintained. Thus, after the occurrence of maltreatment, children may initially make hopelessness-inducing attributions about its occurrence. For example, children may initially explain being beaten or verbally abused by their father by saying, "He was just in a bad mood today," which is an external, unstable, and specific explanation. With the repeated occurrence of maltreatment, however, these hopelessness-inducing attributions may be disconfirmed, leading children to begin making hopelessness-inducing attributions about its occurrence. For example, children may explain the maltreatment by thinking, "I'm a terrible person who deserves all the bad things that happen to me," which is an internal, stable, and global explanation that entails negative consequences and negative self-characteristics. Over time, these attributions may generalize to initially unrelated negative events. In this way, a relatively stable and global negative cognitive style may develop.
Researchers have only recently begun to evaluate the relation between childhood maltreatment and cognitive styles. These initial evaluations, however, have supported Rose and Abramson's hypotheses. For example, controlling for their levels of depressive symptoms, HR participants in the CVD project reported significantly higher levels of depression, but not physical or sexual maltreatment than did LR participants (Gibb, Alloy, Abramson, Rose, Whitehouse, Donovan, et al., 2001). In addition, participants' cognitive risk status fully mediated the relation between reported levels of childhood emotional maltreatment and the occurrence of DSM-III-R and RDC nonendogenous MD during the first 2½ years of follow-up. Further, participants' cognitive risk status fully mediated, and their average levels of hopelessness partially mediated, the relation between reported levels of childhood emotional maltreatment and the occurrence of HD during the first 2½ years of follow-up. To address the possibility that the association of childhood emotional maltreatment with negative cognitive styles is actually due to genetic influences or a negative family environment in general, Gibb, Abramson, and Alloy (2004) also examined the relation between emotional maltreatment by nonrelatives (i.e., peer victimization) during development and negative cognitive styles. Gibb et al. (2004) found that even when parental maltreatment and parental history of psychopathology were controlled, peer victimization still was significantly associated with cognitive HR status. These findings are not easily explained by third variable accounts such as genetic influence or a general negative family context.

Similarly, examining the CVD project participants' average levels of suicidality (both questionnaire- and interview-assessed) across the first 2½ years of follow-up, participants' cognitive risk status and average levels of hopelessness partially mediated the relation between reported levels of childhood emotional maltreatment and average levels of suicidality (Gibb, Alloy, Abramson, Rose, Whitehouse, & Hogan, 2001). The results of a recent cross-sectional study were also supportive of Rose and Abramson's developmental model. Specifically, the results were consistent with the hypothesis that high levels of childhood emotional maltreatment lead to more negative inferences about that maltreatment, which then lead to the development of a negative inferential style, and this inferential style then leaves one vulnerable to hopelessness and the symptoms of hopelessness depression (Gibb, Alloy, Abramson, & Marx, 2003).

In addition to supporting Rose and Abramson's (1992) developmental model, these results also support their hypothesis that childhood emotional maltreatment may be more likely than either childhood physical or sexual maltreatment to contribute to the development of a negative cognitive style. Specifically, Rose and Abramson hypothesized that, with childhood emotional maltreatment, the depressogenic cognitions are directly supplied to the child by the abuser. With childhood physical and sexual maltreatment, however, children must supply their own negative cognitions. In this way, childhood physical and sexual maltreatment may allow greater opportunity for the child to make less depressogenic attributions and inferences for the occurrence of maltreatment. Although these studies provide preliminary evidence for a relation between childhood emotional maltreatment and negative cognitive styles, future longitudinal research is needed that assesses the degree to which emotional maltreatment contributes to increased negativity in cognitive styles over time.

Many important theoretical issues remain to be addressed with the CVD project data. Although analyses to date have indicated that individuals with negative cognitive styles are at higher risk for experiencing clinically significant depression, future analyses will need to be done to evaluate whether nondepressed individuals at high cognitive risk are more likely than low risk individuals to develop depression only when they experience stressful life events, or whether cognitive risk may confer vulnerability to depression even in the absence of negative life events. In the CVD project, negative life events were assessed repeatedly (every 6 weeks for the first 2½ years of follow-up) and dated to the day they occurred, which makes prospective evaluation of this cognitive vulnerability-stress hypothesis possible. Although preliminary investigations of the vulnerability-stress hypothesis have been conducted, a more thorough evaluation is necessary. In addition, it will be important to test if any predictive effect of the cognitive risk x stress interaction for future depressive episodes is mediated by the occurrence of hopelessness, as predicted by the hopelessness theory, and if it is specific to HD as opposed to other possible subtypes of depression. Other environmental and individual difference variables that may serve as protective factors against the development of hopelessness and depression will also need to be explored. For example, there is substantial evidence that social support can help buffer against the occurrence of depression when people experience stressful events (e.g., Cohen & Wills, 1985; Panzarella et al., in press). Future analyses of the CVD project data will allow an investigation of these potential protective factors. There is much room for future research on cognitive vulnerabilities to depression outside of the CVD project as well. Most importantly, the CVD project combined both inferential styles and dysfunctional attitudes in defining negative cognitive style. Although this method of selecting participants provides the strongest possible test of the cognitive theories of depression, it does not allow examination of the unique contribution of inferential styles and dysfunctional attitudes in the prediction of depression. As such, the CVD project represents an important step in research.
examing cognitive vulnerability to depression. Future studies are needed, however, to more specifically test the predictions of Beck's theory and the hopelessness theory, separately (e.g., Haefeli et al., 2003). The role played by positive events should also be addressed in future studies. That is, do positive events provide a buffering effect, protecting against the occurrence of depression? Given the CVF project findings thus far, which have indicated a significant prospective relation between cognitive vulnerability and future depression, studies should continue to examine the therapeutic impact of modifying individuals' cognitive styles. For example, one study has suggested that cognitive behavioral therapists may reduce clients' depressive symptoms by reducing the negativity of clients' attributional styles (DeRubeis & Hollon, 1995). In addition, there is some evidence that training children to make less negative attributions about the negative events in their lives can help protect against future levels of depression (Gillham, Reivich, Jaycox, & Seligman, 1995; Jaycox, Reivich, Gillham, & Seligman, 1994). Given that negative cognitive styles may be especially likely to contribute vulnerability to depression when exacerbated by rumination, depressogenic cognitive styles may also be altered indirectly by training individuals in more effective methods of coping with stressful events, rather than directly trying to alter their cognitive style. Alternatively, it might be necessary to help cognitively vulnerable individuals decrease the stress in their environments.

Further, building positive cognitive styles in children by educating parents to model and provide feedback about more benign inferences for stressful events, as well as direct training in generating positive interpretations of stressful events in schools might help reduce the occurrence of negative cognitive style, and therefore, depression. Finally, parenting classes that teach parents less abusive ways of raising their children may also aid in the prevention of cognitive vulnerability to depression. All of these treatment and prevention models, based on the theorized existence of depressogenic cognitive styles, require further investigation if we are to more fully understand and utilize what we have learned from research on the cognitive theories of depression.

REFERENCES


