Prospective Incidence of First Onsets and Recurrences of Depression in Individuals at High and Low Cognitive Risk for Depression

Lauren B. Alloy
Temple University

Lyn Y. Abramson
University of Wisconsin—Madison

Wayne G. Whitehouse
Temple University

Michael E. Hogan
University of Wisconsin—Madison

Catherine Panzarella
Mental Health Association of Southeastern Pennsylvania,
Philadelphia

Donna T. Rose
Pacquette Center

Do negative cognitive styles provide similar vulnerability to first onsets versus recurrences of depressive disorders, and are these associations specific to depression? The authors followed for 2.5 years prospectively college freshmen ($N = 347$) with no initial psychiatric disorders at high-risk (HR) versus low-risk (LR) for depression on the basis of their cognitive styles. HR participants had odds of major, minor, and hopelessness depression that were 3.5–6.8 times greater than the odds for LR individuals. Negative cognitive styles were similarly predictive of first onsets and recurrences of major depression and hopelessness depression but predicted first onsets of minor depression more strongly than recurrences. The risk groups did not differ in incidence of anxiety disorders not comorbid with depression or other disorders, but HR participants were more likely to have an onset of anxiety comorbid with depression.

Keywords: cognitive vulnerability, depression, first onsets, recurrences, specificity

Two women at the same company fail to receive an expected promotion. One becomes seriously depressed; the other one suffers only mild discouragement. Why are some people vulnerable to depression, whereas others never seem to become depressed? Investigators have attempted to understand such individual differences in vulnerability to depression in response to stress from both biological and psychological perspectives. According to the cognitive theories of depression, the ways people typically construe events in their lives, their cognitive styles, importantly influence their vulnerability to depression. Both hopelessness (Abramson, Metalsky, & Alloy, 1989) and Beck's (1967) theories of depression contain a "cognitive vulnerability hypothesis" in which indi-

viduals who exhibit particular maladaptive thinking patterns are at increased risk for depression when they experience negative life events.

Specifically, according to the hopelessness theory (Abramson et al., 1989), people who exhibit characteristic styles of inferring stable (enduring) and global (widespread) causes, negative consequences, and negative self-characteristics in response to a negative life event are more likely to develop depression—particularly, hopelessness depression (HD)—than people who do not exhibit these negative inferential styles. The logic is that people who exhibit these hypothesized depressogenic inferential styles should be more likely to make negative inferences about the causes, consequences, and self-implications of any particular negative life event they experience, thereby increasing the likelihood that they will develop hopelessness, the proximal cause of the symptoms of depression, particularly HD.

In Beck's (1967) theory, negative self-schemata revolving around themes of failure, loss, inadequacy, and worthlessness are hypothesized to provide cognitive vulnerability to depression. Such negative self-schemata are often represented as a set of dysfunctional attitudes, such as that one's worth depends on being perfect or on others' approval. When they confront negative life events that impinge on these beliefs, individuals who possess dysfunctional attitudes are hypothesized to develop negatively biased construals of the self, world, and future (hopelessness) and, in turn, depression. Thus, both hopelessness and Beck's theories hypothesize that people with negative cognitive styles are at

greater risk for depression onset than people with positive cognitive styles because of the former group's tendency to appraise life events negatively.

Cognitive Vulnerability to Clinical Depression and the Behavioral High-Risk Design

Do negative cognitive styles actually contribute vulnerability to clinically significant depression? Elsewhere we (e.g., Alloy et al., 2000; Just, Abramson, & Alloy, 2001) have argued that the most powerful and direct method of testing the cognitive vulnerability hypotheses of depression is with a behavioral high-risk design (Alloy, Lipman, & Abramson, 1992; Depue et al., 1981; see also Zuroff, Mongrain, & Santor, 2004, for additional benefits of the behavioral high-risk design). In this design, individuals are studied who do not currently have the disorder of interest (e.g., depression) but who are hypothesized to be at high versus low risk for developing the disorder because of their status on a behavioral vulnerability to the disorder. Thus, to test the cognitive vulnerability hypotheses in the present prospective portion of the Temple–Wisconsin Cognitive Vulnerability to Depression (CVD) Project (Alloy & Abramson, 1999; Alloy et al., 2000), we selected nondepressed participants at high versus low risk for depression based on the presence versus absence of the depressogenic cognitive styles. We then compared these cognitive high-risk (HR) and low-risk (LR) groups on their likelihood of developing depression during a 2.5-year prospective follow-up period.

Consistent with the cognitive vulnerability hypotheses, three prior studies (Alloy et al., 1992, 2000; Haeffel et al., 2003), including the retrospective portion of the CVD Project (Alloy et al., 2000), through the use of a retrospective version of the behavioral high-risk design found that nondepressed participants with the hypothesized depressogenic cognitive styles had higher rates of past major depression (MD), minor depression (MiD), and HD than did participants who did not exhibit these negative cognitive styles. Although the increased past history of depressive disorders among cognitive HR individuals obtained in these studies is predicted by the cognitive vulnerability hypotheses, these studies have a major conceptual limitation. The direction of the association between negative cognitive styles and increased past rates of depressive disorders is ambiguous because of the retrospective nature of the design. The negative cognitive styles could have developed as a consequence of the past depression (Lewinsohn, Steinmetz, Larson, & Franklin, 1981) rather than temporally preceding and contributing vulnerability to the onset of the past depression. Thus, a more definitive test of the cognitive vulnerability hypotheses of depression requires a prospective behavioral high-risk design.

Prior prospective investigations with children (Abela, 2001; Abela & Sarin, 2002; Conley, Haines, Hilt, & Metalsky, 2001; Dixon & Ahrens, 1992; Hilsman & Garber, 1995; Nolen-Hoeksema, Girgus, & Seligman, 1992; Robinson, Garber, & Hilsman, 1995), adolescents (Hankin, Abramson & Siler, 2001), and adults (Alloy & Clements, 1998; Alloy, Just, & Panzarella, 1997; Alloy, Reilly-Harrington, Fresco, Whitehouse, & Zechmeister, 1999; Brown, Hammen, Craske, & Wickens, 1995; Dykman & Johll, 1998; Hankin, Abramson, Miller, & Haeffel, 2004; Joiner, Metalsky, Lew, & Klocek, 1999; Klocek, Oliver, & Ross, 1997; Kwon & Oei, 1992; Lewinsohn, Joiner, & Rohde, 2001; Metal-

sky, Halberstadt, & Abramson, 1987; Metalsky & Joiner, 1997; Metalsky, Joiner, Hardin, & Abramson, 1993; Olinger, Kuiper, & Shaw, 1987; Reilly-Harrington, Alloy, Fresco, & Whitehouse, 1999) have provided considerable support for the cognitive vulnerability hypotheses of hopelessness or Beck's (1967) theories (but see Ingram, Miranda, & Segal, 1998, for a review of nonsupportive studies as well). Moreover, a recent review of the empirical status of broader cognitive–personality factors in depression has supported their role as vulnerabilities for depression (Zuroff et al., 2004). However, with two exceptions (Hankin et al., 2004, Study 2; Lewinsohn et al., 2001), the prior prospective tests of hopelessness and Beck's theories demonstrated that dysfunctional attitudes or negative attributional (or inferential) styles provide vulnerability to depressive mood or symptoms. Thus, little is known as yet about whether negative cognitive styles also provide vulnerability to full-blown, clinically significant depressive disorders. Testing the cognitive vulnerability hypotheses for depressive disorders is particularly important given previous critiques (e.g., Coyne & Gotlib, 1983) that the cognitive theories are relevant to mild depressive symptoms but not to clinically significant depression. Consequently, the present study uses a prospective behavioral high-risk design to test the cognitive vulnerability hypotheses of hopelessness and Beck's theories jointly for clinically significant depressive disorders.

Cognitive Vulnerability and First Onsets Versus Recurrences of Clinical Depression

A second goal of our study was to examine whether negative cognitive styles provide vulnerability similarly for first onsets versus recurrences of depression. According to hopelessness (Abramson et al., 1989) and Beck's (1967) theories, negative inferential styles and dysfunctional attitudes, respectively, are relatively stable vulnerabilities that should increase risk for both first and subsequent episodes of depression. However, it is possible that distal vulnerability factors such as negative cognitive styles are primarily important for increasing risk for first episodes, and then, once an episode has occurred, more proximal factors such as priming (e.g., Persons & Miranda, 1992; Riskind & Rholes, 1984; Scher, Ingram, & Segal, 2005) become more important in contributing risk for additional episodes. Moreover, some theories and empirical work on depression suggest that different risk factors may be involved in first onsets versus recurrences (Daley, Hammen, & Rao, 2000; Lewinsohn, Allen, Seeley, & Gotlib, 1999). For example, on the basis of Teasdale's (1983, 1988) differential activation hypothesis, which suggests that prior experience with depression leads to a greater likelihood of mutual activation of dysphoria and depressive thinking, Lewinsohn and colleagues (1999) predicted and found that dysphoria and dysfunctional attitudes interacted to predict recurrences, but not first onsets, of depression.

Given the high rate of recurrence of depression (Judd, 1997), coupled with the tendency for first onsets to occur in adolescence or early adulthood (Burke, Burke, Regier, & Rae, 1990; Hankin et al., 1998), most prior research on risk factors for depression has really examined predictors of recurrence rather than first onset. Thus, inasmuch as the CVD Project included a late adolescent sample of initially nondepressed individuals, approximately half of whom had no prior history of depression, we examined whether

negative cognitive styles differentially predicted first onsets versus recurrences of depressive disorders. The determination of whether the cognitive vulnerability hypothesis holds for first onsets versus subsequent episodes has important implications for understanding the etiology of depression and how it evolves over the course of repeated episodes. Such understanding, in turn, will inform cognitive–behavioral interventions aimed at preventing initial onset versus recurrence of depression.

Cognitive Vulnerability and Specificity to Depression

A third goal of this study was to examine the specificity of the hypothesized depressogenic cognitive styles as vulnerabilities for depressive disorders. Given that anxiety disorders are frequently comorbid with depression (Alloy, Kelly, Mineka, & Clements, 1990; Mineka, Watson, & Clark, 1998), it is important to examine whether negative cognitive styles confer specific risk for depressive disorders or more general vulnerability to anxiety and other disorders. Indeed, in their helplessness–hopelessness theory of comorbidity, Alloy et al. (1990) suggested that the negative inferential styles featured as vulnerabilities in the hopelessness theory of depression (Abramson et al., 1989) were likely to increase risk for comorbid anxiety and depression, as well as depression itself, because these styles would increase the likelihood of developing the expectation of helplessness held with certainty, a proximal cause of mixed depression and anxiety symptoms. However, these negative inferential styles should not increase risk for pure anxiety disorders or other Axis I disorders.

A majority of prior studies have found that negative cognitive styles are associated with depression but not anxiety (see Mineka, Pury, & Luten, 1995, for a review; Alloy et al., 2000; Gladstone, Kaslow, Seeley, & Lewinsohn, 1997; Lewinsohn, Gotlib, & Seeley, 1997; Weiss, Susser, & Catron, 1998), although this is not always the case (Haeffel et al., 2003; Luten, Ralph, & Mineka, 1997). In addition, some prospective studies have found that the Cognitive Vulnerability × Stress combination of the hopelessness theory predicts depressive symptoms more specifically than anxiety symptoms or symptoms of other disorders (Alloy & Clements, 1998; Alloy et al., 1997; Hankin et al., 2004; Metalsky & Joiner, 1997; Robinson et al., 1995), whereas other studies did not find this specificity (Cole & Turner, 1993; Hammen, Adrian, & Hiroto, 1988; Luten et al., 1997; Ralph & Mineka, 1998).

A limitation of these prior studies is that they did not test the specificity of the negative cognitive styles at the level of clinically significant disorders. Lewinsohn et al. (2001) found that the Cognitive Vulnerability × Stress interaction predicted depressive disorder, but not nonmood disorders, among adolescents. However, the specificity of cognitive vulnerability for depressive versus anxiety disorders could not be determined in this study because all nonmood disorders were grouped together. In contrast, Hankin et al. (2004, Study 2) found that the Cognitive Vulnerability × Stress combination from both hopelessness and Beck's (1967) theories prospectively predicted depressive disorders specifically and not anxiety disorders. However, the Hankin et al. study was based on a small sample size and did not consider the comorbidity of anxiety and depressive disorders. Consequently, further tests of the specificity of negative cognitive styles as prospective vulnerabilities for clinically significant depressive disorders versus anxiety disorders and other Axis I disorders needed.

The Present Study

In this article, we provide the first presentation of diagnostic data from the prospective portion of the Temple–Wisconsin CVD Project (Alloy & Abramson, 1999) based on both sites. In the CVD Project, university freshmen who were nondepressed and had no other current Axis I disorders at the start of the study, but who were selected to be at high- or low-cognitive risk for depression on the basis of the presence versus absence of dysfunctional attitudes and negative inferential styles, were followed prospectively with structured diagnostic interview assessments of Axis I psychopathology. Here, we report on the prospective incidence of depressive and other Axis I disorders in the cognitive HR and LR groups during the first 2.5 years of follow-up. On the basis of the cognitive vulnerability hypotheses of hopelessness and Beck's (1967) theories of depression, we predicted that (a) HR participants would exhibit higher prospective incidence (probability of occurrence) of both first onsets and recurrences of depressive disorders, including the subtype of HD, than would LR participants and (b) there would be no risk group differences in prospective incidence of other Axis I disorders, with the exception of comorbid anxiety and depression.

In following Lewinsohn et al. (2001), we controlled for current depressive symptoms at the time of assessment of cognitive vulnerability for two reasons. First, current depression is an established risk factor for future depression (Hammen et al., 1988; Lewinsohn et al., 1994), and, thus, we wanted to rule out initial dysphoria associated with HR status as a plausible explanation for any risk group differences in prospective onsets of depression obtained. Second, previously depressed individuals may show elevated residual depressive symptoms that could confound the interpretation of findings regarding the effect of a prior history of depression (and thus, the interpretation of risk group differences in recurrences of depression). In addition, to provide a stringent test of the specificity hypothesis for cognitive vulnerability, we also controlled for the prospective occurrence of anxiety disorders when predicting prospective onsets of depressive disorders, and we controlled for the prospective occurrence of depressive disorders when predicting prospective onsets of anxiety disorders or other disorders. Finally, we also examined whether any risk group differences in prospective onsets of anxiety or depressive disorders was explained by anxiety–depression comorbidity.

Method

Participants and procedure. Freshmen at Temple University (TU) and the University of Wisconsin (UW) were selected for the CVD Project on the basis of a two-phase screening procedure. Details of the selection procedures are provided in Alloy and Abramson (1999) and Alloy et al. (2000). In brief, in Phase I, we gave the Cognitive Style Questionnaire (CSQ; Alloy et al., 2000) and Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978), measures of cognitive styles featured as vulnerabilities for depression in hopelessness and Beck's (1967) theories, respectively, to 5,378 freshmen. Those who scored in the highest quartile (most negative) on both the DAS and CSQ composite (stability, globality, consequences, and self dimensions) for negative events were potential HR participants, whereas those who scored in the lowest quartile (most positive) on both instruments were potential LR participants (see Alloy et al., 2000, for HR and LR cutpoints on the CSQ and DAS and Phase I screening sample means).

Table 1
Final CVD Project Sample: Demographic and Cognitive Style Characteristics

| Characteristic | High-risk (n = 172) | | Low-risk (n = 175) | | Comparison |
	M	SD	M	SD	Statistical test
Age (in years)	18.59	1.04	19.20	2.31	$t(345) = -3.20, p = .01$
Parent education (in years)[a]	14.54	2.42	14.26	2.40	$t(337) = 1.07, p = .29$
Combined parent income[a]	$67,449	$80,353	$57,341	$46,009	$t(268) = 1.25, p = .21$
Gender (%)					$\chi^2(345) = 0.04, p = .97$
Female	67.3		67.0		
Male	32.7		33.0		
Race					$\chi^2(345) = 1.60, p = .11$
Caucasian	82.4		75.3		
Other	17.6		24.7		
DAS item score	4.45	0.50	2.20	0.31	$t(345) = 50.79, p < .01$
CSQ–N item score	5.10	0.43	2.75	0.40	$t(345) = 52.39, p < .01$

Note. DAS = Dysfunctional Attitudes Scale; CSQ–N = Cognitive Style Questionnaire—Negative Events Composite.
[a] Some participants did not provide parental education or income data, so degrees of freedom are smaller.

In Phase II, a random subset of freshmen who were <30 years old and met the Phase I criteria for the HR or LR groups were given an expanded Schedule for Affective Disorders and Schizophrenia—Lifetime (SADS–L) interview (Endicott & Spitzer, 1978) by interviewers who were blind to risk status. Participants also completed the Beck Depression Inventory (BDI; Beck, Rush, Shaw, & Emery, 1979). On the basis of the expanded SADS–L interview and *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.; *DSM–III–R*; American Psychiatric Association, 1987) and research diagnostic criteria (RDC; Spitzer, Endicott, & Robins, 1978), Phase II participants were excluded if they exhibited any current mood or other Axis I disorder, psychotic symptoms, bipolar disorder, or any serious medical illness. Participants were retained if they met diagnostic criteria for a past depressive disorder but had remitted for a minimum of 2 months (to ensure that any depression onsets during the prospective phase were new episodes and not relapses). On average, the most recent past episode of depression was 2.31 years (SD = 2.44 years.) before Phase I. Our logic in including participants who were nondepressed at the start of the project but had a past depression is that exclusion of such individuals might lead to a biased test of our main hypotheses. If the cognitive vulnerability hypotheses are correct, then HR participants, by virtue of their negative cognitive styles, should be continually at risk and thus more likely to have experienced past depression than LR participants—a result we did obtain (Alloy et al., 2000). If we excluded such individuals, we might be left with an unrepresentative HR group consisting of participants who, despite possessing very negative cognitive styles, do not readily become depressed, perhaps because they have other protective factors (see Panzarella, Alloy, & Whitehouse, in press).

The final CVD Project sample included 172 HR and 175 LR participants (see Table 1). The cohorts at the two sites were comparable on gender and cognitive styles, but the TU cohort had a higher percentage of minority participants, $\chi^2(1) = 47.97, p < .001$; was older, $F(1, 341) = 24.50, p < .001$; and had lower mean parental education, $F(1, 331) = 33.57, p < .001$, and income,[1] $F(1, 261) = 16.06, p < .001$, than did the UW cohort. To the extent that the present findings replicate across the sites, the site differences in socioeconomic status and ethnic composition should increase the generalizability of our results. The HR and LR groups did not differ on gender or ethnicity; however, the LR group was older than the HR group, $F(1, 341) = 10.91, p < .002$. Inasmuch as the likelihood of experiencing disorders increases with age, the fact that the LR group was older worked against our hypothesis of higher rates of prospective depressive episodes in the HR group. Nevertheless, we controlled for age in our analyses. The final sample was representative of the original Phase I screening sample on

age and ethnicity, but had a higher proportion of women, $\chi^2(1) = 9.86, p < .01$, than did the Phase I screening sample. In addition, the final sample did not differ from Phase II eligible freshmen who did not participate. Further details of the study methodology are available from Alloy and Abramson (1999) and Alloy et al. (2000). Within 1 month of Phase II, the final sample completed a Time 1 assessment and then began the prospective phase.

Assessment of Axis I disorders. Lifetime diagnoses were based on the expanded SADS–L interviews administered at the Phase II screening. Prospective diagnoses were derived from expanded SADS—Change (SADS–C) interviews administered every 6 weeks during the 2.5 years of follow-up. All SADS–L and SADS–C interviewers were blind to participants' cognitive risk status. The original SADS–L and SADS–C interviews were expanded to allow for *DSM–III–R* as well as RDC diagnoses, more detailed assessment of depression and HD symptoms, and improved assessment of anxiety disorders (see Alloy & Abramson, 1999, and Alloy et al., 2000, for further details). In addition, the SADS–C interview was further expanded to allow for *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*; American Psychiatric Association, 1994) diagnoses as well. Moreover, we also incorporated features of the Longitudinal Interval Follow-up Evaluation (LIFE II; Shapiro & Keller, 1979) into our expanded SADS–C interview to allow us to track symptoms daily. Analyses reported here are based on *DSM–IV* and RDC criteria. We also established project criteria for diagnosing the hypothesized subtype of HD (see Appendix). Our diagnostic interviewer training program is described in Alloy et al. (2000). Throughout the project, we calibrated our diagnoses across interviewers within and between sites as well as with recognized diagnostic experts (see Alloy & Abramson, 1999, and Alloy et al., 2000, for further details). An interrater reliability study on a randomly chosen subset of 80 of the SADS–L and 125 of the SADS–C interviews yielded kappas ≥.90 for all project diagnoses. Further, a test–retest reliability study of 80 SADS–C interviews in which different interviewers blindly and independently interviewed the same participant with the SADS–C within 2 days for the same 6-week interval yielded a mean correlation of .97 between interviewers for day-by-day dating of depressive episodes.

For depression diagnoses, in order to ensure that episodes were sufficiently severe to have clinical significance, episodes also were required to meet a minimum severity criterion on the BDI (also given every 6 weeks

---

[1] The degrees of freedom for the risk-group comparison on parental income are small because many participants did not provide this information.

for each 2-week period in the interval)[2] as well as the relevant *DSM–IV* or RDC diagnostic criteria. For major depression (MD) episodes, the BDI criterion was ≥16; for minor depression (MiD) and hopelessness depression (HD) episodes, the BDI criterion was ≥10.[3]

Assessment of depressive symptoms. The BDI (Beck et al., 1979) is a 21-item self-report questionnaire that assesses the presence and severity of cognitive, motivational, affective, and somatic symptoms of depression. Previous research has shown that the BDI is internally consistent ($\alpha = .81$ for nonpsychiatric samples; Beck, Steer, & Garbin, 1988) and valid with both psychiatric and undergraduate samples (e.g., Beck et al., 1988). Test–retest reliability of the BDI is also adequate (Beck et al., 1988). The BDI was administered at the Phase II screening and at each 6-week prospective assessment. It was used to assess initial levels of depressive symptoms at the start of the prospective follow-up and as an additional severity criterion for MD, MiD, and HD episodes.

Assessment of cognitive vulnerability. Cognitive risk was assessed with the CSQ and an expanded version of Form A of the DAS, created specifically for the CVD Project by Lauren B. Alloy and Lyn Y. Abramson. The CSQ assessed the internality, stability, and globality of causal attributions as well as inferred consequences and self-worth implications for hypothetical positive and negative events. We used an additive composite score of the stability, globality, consequences, and self dimensions for negative events to select HR and LR participants (along with the DAS) as described above. Coefficient alpha ($n = 5,378$) for the negative event composite was .88, and retest stability over 1 year ($n = 349$) was $r = .80$ (Alloy et al., 2000). An expanded DAS, Form A, assessed dysfunctional attitudes regarding concern with others' approval and perfectionism. The expansion involved adding an additional 24 items to the original 40-item DAS to better capture dysfunctional beliefs in the achievement and interpersonal domains specifically. Coefficient alpha ($n = 5,378$) for the expanded DAS was .90, and retest stability over 1 year ($n = 349$) was $r = .78$ (Alloy et al., 2000).

Data analyses. All analyses controlled for the effects of age, Phase II BDI scores (to control for any initial HR–LR differences in depressive symptoms as well as residual depressive symptoms associated with a past history of depression), and length of follow-up (in months) on prospective onsets of depressive and other Axis I disorders. To test the cognitive vulnerability hypotheses for prospective onsets of depressive episodes, hierarchical logistic regression analyses were conducted with predictors entered in the following order: the three covariates (Step 1), prior depression (presence vs. absence of at least one prior MD or MiD episode [Step 2]), any anxiety disorder (presence vs. absence of at least one prospective anxiety disorder [Step 3]), gender (Step 4), site (Step 5), cognitive risk (Step 6), the Risk × Prior Depression interaction (Step 7), the Risk × Gender interaction (Step 8), and the Risk × Site interaction (Step 9).[4] When either prospective onsets of anxiety disorders or other Axis I disorders was the dependent variable, any depression (presence vs. absence of at least one prospective depressive disorder) was entered in Step 3 rather than "any anxiety disorder". Note that the test of the cognitive risk effect was very conservative in that we only examined the effect of risk after controlling for initial depressive symptoms, prior depression history, and prospective onsets of anxiety disorders (as well as age, gender, site, and duration of follow-up). The effect of the Risk × Prior Depression interaction (Step 7) allowed us to test whether cognitive vulnerability differentially predicts first onsets versus recurrences of depression.

Results

In the full sample ($N = 347$), the overall incidence of onset of various disorders during the prospective follow-up was 49 (14.1%) for MD, 126 (36.3%) for MiD, 91 (26.2%) for HD, 19 (5.5%) for any anxiety disorder, and 10 (2.9%) for any other disorder (the disorders in this category were mostly alcohol and drug use disorders). Of the 91 onsets of HD episodes, 47 (52%) also met

criteria for MD and 31 (34%) for MiD. There were no significant gender differences in prospective onsets of any depressive or anxiety disorders; however, there was a gender difference for prospective onsets of any other disorder, $t(331) = -2.15$, $p < .04$ (see Table 4). Men had a greater likelihood than women (6.1% vs. 1.3%) of having an onset of any other disorder. There were no site differences in prospective onsets of any disorders examined.

Prospective onsets of depressive disorders as a function of cognitive risk and prior depression. Table 2 displays the results of the hierarchical regression analyses for prospective onsets of MD, MiD, and HD. As can be seen in Table 2, both initial BDI scores and a prior history of a depressive episode significantly predicted the likelihood of a prospective onset of MD, MiD, and HD. A history of a past episode of depression increased the likelihood of a recurrence of MD, MiD, and HD during the 2.5-year prospective follow-up by 2.66, 2.77, and 2.81 times, respectively. The prospective onset of any anxiety disorder did not predict the likelihood of onset of any of the depressive disorders. Most important, consistent with the cognitive vulnerability hypotheses, controlling for age, gender, site, duration of follow-up, initial BDI scores, prior history of depression, and prospective onset of any anxiety disorders, cognitive risk predicted the likelihood of a prospective onset of MD, MiD, and HD significantly. Indeed, the odds of an onset of MD, MiD, and HD during the college years were 6.66, 3.53, and 6.77 greater, respectively, for HR compared with LR individuals (see Table 3 for actual rates of depressive disorders as a function of risk group and prior depression status).

The Risk × Prior Depression interaction is relevant to examining whether cognitive vulnerability predicts first onsets versus recurrences of depressive disorders differentially. As can be seen in Table 2, controlling for age, gender, site, duration of follow-up, initial BDI scores, prior history of depression, prospective onset of any anxiety disorders, and cognitive risk status, this interaction was not significant in predicting prospective onsets of MD or HD, although there was a trend for the Risk × Prior Depression

---

[2] For feasibility reasons, it was not possible to actually administer the BDI every 2 weeks throughout the prospective phase of the project. Thus, participants completed the BDI every 6 weeks for each 2-week period in the 6-week interval. However, we conducted a validity substudy in which 15 HR and 15 LR participants completed daily ratings of depressive symptoms for a month and then completed a BDI at the end of the month for each 2-week period in the 1-month interval. This substudy obtained a high correlation between participants' BDI scores for each 2-week period at the end of the month and the average of their daily-rated symptoms for the same 2-week period ($r = .88$).

[3] These BDI cutoffs were based on Kovacs and Beck's (1977) standard depth of depression cutpoints for the BDI. The cutoff of a BDI score ≥16 for major depression represents at least moderate depression according to Kovacs and Beck, whereas the cutoff of a BDI score ≥10 for minor depression and HD represents at least mild depression according to Kovacs and Beck.

[4] There were not enough prospective episode onsets for major depression, any anxiety disorder, or any other disorder to allow entry of three-way and four-way interactions among predictors in the final steps of the analyses. However, no three-way or four-way interactions were significant for prediction to minor depression or HD onsets. Thus, for consistency, we do not present these higher order interactions in Tables 2 and 4 displaying results from the hierarchical regression analyses.

Table 2

Hierarchical Regression Analyses for Prospective Onsets of Depressive Disorders

Step and predictor	Estimate	t	p	OR	95% CI
		Major depression			
1					
Age	−0.13	−0.95	.34	0.88	0.67–1.15
Phase 2 BDI	0.07	3.78	.00	1.07	1.03–1.11
Duration of follow-up (months)	−0.01	−0.23	.82	0.99	0.92–1.07
2					
Prior depression (PD)[a]	0.98	2.66	.01	2.66	1.29–5.45
3					
Any anxiety disorder[b]	0.82	1.44	.15	2.27	0.75–6.92
4					
Gender	−0.09	−0.23	.82	0.92	0.45–1.89
5					
Site	−0.33	−0.91	.37	0.72	0.35–1.47
6					
Cognitive risk (CR)[c]	1.90	3.50	.00	6.66	2.31–19.24
7					
CR × PD	−1.32	−1.10	.27	0.27	0.02–2.81
8					
CR × Gender	0.30	0.29	.77	1.34	0.18–10.04
9					
CR × Site	−2.03	−1.66	.10	0.13	0.01–1.44
		Minor depression			
1					
Age	−0.12	−1.34	.18	0.89	0.74–1.06
Phase 2 BDI	0.07	4.69	.00	1.07	1.04–1.11
Duration of follow-up (months)	−0.00	−0.06	.95	1.00	0.94–1.06
2					
Prior depression (PD)[a]	1.02	3.89	.00	2.77	1.66–4.63
3					
Any anxiety disorder[b]	0.56	1.13	.26	1.76	0.66–4.71
4					
Gender	0.23	0.82	.41	1.26	0.73–2.17
5					
Site	−0.19	−0.72	.47	0.82	0.49–1.40
6					
Cognitive risk (CR)[c]	1.26	3.84	.00	3.53	1.85–6.71
7					
CR × PD	−1.47	−2.44	.02	0.23	0.07–0.75
8					
CR × Gender	−0.84	−1.28	.20	0.43	0.12–1.56
9					
CR × Site	0.12	0.21	.83	1.13	0.36–3.53
		Hopelessness depression			
1					
Age	−0.14	−1.55	.12	0.87	0.72–1.04
Phase 2 BDI	0.06	4.23	.00	1.07	1.04–1.10
Duration of follow-up (months)	−0.01	−0.23	.82	0.99	0.94–1.05
2					
Prior depression (PD)[a]	1.03	3.96	.00	2.81	1.69–4.89
3					
Any anxiety disorder[b]	0.84	1.67	.09	2.32	0.87–6.20
4					
Gender	0.45	1.59	.11	1.57	0.90–2.74
5					
Site	−0.37	−1.36	.17	0.69	0.41–1.18
6					
Cognitive risk (CR)[c]	1.91	5.52	.00	6.77	3.43–13.33
7					
CR × PD	−1.17	−1.82	.07	0.31	0.09–1.09
8					
CR × Gender	0.13	0.20	.84	1.14	0.31–4.23
9					
CR × Site	−0.63	−1.03	.30	0.53	0.16–1.76

Note. BDI = Beck Depression Inventory; OR = odds ratio; CI = confidence interval.
[a] *Prior depression* refers to the presence or absence of a prior history of major or minor depression. [b] *Any anxiety disorder* refers to the presence or absence of a prospective onset of any anxiety disorder. [c] *Cognitive risk* refers to cognitive risk status (i.e., high risk vs. low risk).

Table 3
Prospective Rates of Depressive, Anxiety, and Other Disorders as a Function of Cognitive Risk and Prior History of Depression

	High risk/ No prior depression ($n = 74$)		Low risk/ No prior depression ($n = 111$)		High risk/ Prior depression ($n = 98$)		Low risk/ Prior depression ($n = 64$)	
Disorder	n	%	n	%	n	%	n	%
Major depression[a]	12	16.2	3	2.7	28	28.6	6	9.4
Minor depression[b]	34	45.9	16	14.4	55	56.1	21	32.8
Hopelessness depression[c]	26	35.1	4	3.6	49	50.0	12	18.8
Any anxiety disorder[a]	5	6.8	1	0.9	10	10.2	3	4.7
Any other disorder[a]	3	4.1	1	0.9	5	5.1	1	1.6

[a] *DSM–IV* or research diagnostic criteria (RDC) diagnosis. [b] RDC diagnosis only. [c] Project criteria.

interaction to predict HD ($p < .068$). Thus, first onsets and recurrences of MD and HD were similarly predicted by cognitive vulnerability. However, the Risk × Prior Depression interaction did significantly predict onsets of MiD (see second portion of Table 2). To examine the pattern of this interaction, we conducted hierarchical regression analyses separately for participants with and without a prior history of depression. Among the 185 participants (74 HR, 111 LR) with no lifetime history of depression, controlling for age, gender, site, duration of follow-up, initial BDI scores, and prospective onset of any anxiety disorders, cognitive risk predicted the likelihood of a first onset of MiD significantly, $t(181) = 3.40$, $p < .001$, odds ratio [OR] = 7.13, 95% confidence interval [CI] = 2.30–22.13; see Table 3 for actual rates of MiD. In contrast, among the 162 participants (98 HR, 64 LR) with a prior history of depression, controlling for age, gender, site, duration of follow-up, initial BDI scores, and prospective onset of any anxiety disorders, cognitive risk only marginally predicted the likelihood of a recurrence of MiD, $t(160) = 1.89$, $p < .06$, OR = 2.21, 95% CI = 0.97–5.03; see Table 3 for rates of MiD.

Prospective onsets of anxiety and other Axis I disorders as a function of cognitive risk and prior depression. Inasmuch as the onset rates of other individual Axis I disorders were small, we combined individual anxiety diagnoses (*DSM–IV* or RDC panic disorder, generalized anxiety disorder, specific phobia, social phobia, obsessive–compulsive disorder, posttraumatic stress disorder) into an any anxiety disorder category and all other Axis I disorders into an any other disorder category for analysis. Table 4 displays the results of the hierarchical regression analyses for prospective onsets of any anxiety disorder and any other disorder.

Initial BDI scores, prior history of depression, and prospective onset of any depressive disorder did not predict the likelihood of prospective onset of any anxiety or other disorders. Consistent with the specificity hypothesis for cognitive vulnerability, controlling for age, gender, site, duration of follow-up, initial BDI scores, prior history of depression, and prospective onset of any depressive disorder, cognitive risk did not predict the likelihood of onset of any other disorder (see last portion of Table 4). However, cognitive risk did predict significantly the likelihood of an onset of any anxiety disorder (see the first portion of Table 4). The odds of an onset of any anxiety disorder were 4.12 times greater among HR participants than among LR participants (see Table 3 for the rates of any anxiety disorder). However, further analyses indicated

that this effect was attributable to anxiety disorders comorbid with depression. When we conducted the hierarchical regression analysis for any anxiety disorder with comorbidity of prospective onsets of anxiety and depression as a predictor instead of prospective onset of depression, cognitive risk no longer predicted the likelihood of an onset of any anxiety disorder, $t(341) = -0.10$, $p = .925$. In contrast, when we conducted the hierarchical regression analyses for MD, MiD, and HD with comorbidity of prospective onsets of anxiety and depression as a predictor instead of prospective onset of anxiety, cognitive risk was still a significant predictor of the likelihood of onset of MD, MiD, and HD: $t(341) = 3.29$, $p < .001$, OR = 6.03, 95% CI = 2.07–17.58; $t(341) = 3.37$, $p < .001$, OR = 3.08, 95% CI = 1.60–5.90; $t(341) = 5.17$, $p < .000$, OR = 6.08, 95% CI = 3.07–12.05, respectively. Moreover, we divided participants with an onset of any anxiety disorder into two groups: those with ($n = 11$; 10 HR, 1 LR) and without ($n = 8$; 5 HR, 3 LR) a comorbid depressive episode. In the full sample, controlling for age, gender, site, duration of follow-up, initial BDI scores, prior history of depression, and prospective onset of any depressive disorder, cognitive risk significantly predicted onset of anxiety disorder comorbid with depression (OR = 19.6; 95% CI = 2.1–187.0; $p < .01$), but not of anxiety disorder alone (OR = 1.4; 95% CI = 0.2–9.4; $p < .80$).

Discussion

Consistent with the cognitive vulnerability hypotheses of hopelessness (Abramson et al., 1989) and Beck's (1967) theories, nondepressed individuals who exhibited negative inferential styles and dysfunctional attitudes were more likely to experience prospective onsets of major, minor, and hopelessness depression than were nondepressed individuals who did not exhibit these negative cognitive styles. Indeed, among HR participants, the risk for onset of major depression and hopelessness depression was almost 7 times greater, and for minor depression 3.5 times greater, than that of LR participants. As such, these findings extend prior work (Alloy et al., 1992, 2000; Haeffel et al., 2003) demonstrating that negative cognitive styles are associated with a greater likelihood of past major, minor, and hopelessness depression, as well as prior prospective studies (Abela, 2001; Abela & Sarin, 2002; Alloy & Clements, 1998; Alloy et al., 1997, 1999; Brown et al., 1995; Conley et al., 2001; Dixon & Ahrens, 1992; Dykman & Johll,

Table 4
Hierarchical Regression Analyses for Prospective Onsets of Anxiety and Other Disorders

Step and Predictor	Estimate	t	p	OR	95% CI
Any anxiety disorder					
1					
Age	0.10	0.94	.35	1.11	0.90–1.37
Phase 2 BDI	0.03	1.34	.18	1.03	0.98–1.09
Duration of follow-up (months)	0.05	0.83	.41	1.05	0.93–1.18
2					
Prior depression (PD)[a]	0.75	1.41	.16	2.13	0.75–6.06
3					
Any depression[b]	−0.34	−1.33	.19	0.71	0.43–1.18
4					
Gender	−0.03	−0.07	.95	0.97	0.36–2.61
5					
Site	−0.04	−0.07	.94	0.96	0.34–2.70
6					
Cognitive Risk (CR)[c]	1.42	2.05	.04	4.12	1.06–16.00
7					
CR × PD	−1.09	−0.84	.40	0.34	0.03–4.36
8					
CR × Gender	−0.70	−0.54	.59	0.50	0.04–6.44
9					
CR × Site	−1.13	−0.86	.39	0.32	0.02–4.25
Any other disorder					
1					
Age	0.15	1.17	.24	1.16	0.90–1.50
Phase 2 BDI	0.04	1.38	.17	1.04	0.98–1.11
Duration of follow-up (months)	0.05	0.57	.57	1.05	0.89–1.23
2					
Prior depression (PD)[a]	0.42	0.62	.54	1.52	0.40–5.76
3					
Any depression[b]	1.13	1.56	.12	3.10	0.75–12.82
4					
Gender	−1.54	−2.15	.03	0.21	0.05–0.87
5					
Site	−0.88	−1.09	.27	0.41	0.08–2.01
6					
Cognitive Risk (CR)[c]	1.12	1.13	.26	3.07	0.44–21.44
7					
CR × PD	−0.23	−0.14	.89	0.80	0.03–19.66
8					
CR × Gender	−1.13	−0.68	.50	0.32	0.01–8.43
9					
CR × Site[d]					

Note. BDI = Beck Depression Inventory; OR = odds ratio; CI = confidence interval. [a] *Prior depression* refers to the presence or absence of a prior history of major or minor depression. [b] *Any depression* refers to the presence or absence of a prospective onset of any depressive disorder. [c] *Cognitive risk* refers to cognitive risk status (i.e., high risk vs. low risk). [d] The analysis could not provide the statistics for the CR × Site interaction because there were too few cases of any other disorder at one of the sites.

1998; Hankin et al., 2001, 2004; Hilsman & Garber, 1995; Joiner et al., 1999; Klocek et al., 1997; Kwon & Oei, 1992; Lewinsohn et al., 2001; Metalsky et al., 1987, 1993; Metalsky & Joiner, 1997; Nolen-Hoeksema et al., 1992; Olinger et al., 1987; Reilly-Harrington et al., 1999; Robinson et al., 1995) obtaining an association between negative cognitive styles and subsequent depressive symptoms. Inasmuch as we assessed depression with structured diagnostic interviews and standardized diagnostic criteria rather than with depressive symptom questionnaires, our findings suggest that negative cognitive styles indeed confer vulnerability to onsets of clinically significant depressive disorders. This is important because a criticism sometimes leveled at the cognitive

theories of depression is that they apply only to mild depressive symptoms (Coyne & Gotlib, 1983). Moreover, our finding that the risk group differences in onsets of major, minor, and hopelessness depression replicated across sites (there were no Risk × Site interactions), despite substantial site differences in ethnicity and socioeconomic status, provides evidence for the generalizability of our findings.

We obtained support for the cognitive vulnerability hypotheses despite controlling for initial depressive symptoms (BDI scores) and prior history of depressive disorder (see Lewinsohn et al., 2001). Consequently, initial dysphoria associated with HR status or residual depressive symptoms associated with prior depression

are unlikely to be plausible explanations for our cognitive risk effects. Our controls for initial BDI scores and prior history of depression provide a very (probably overly) conservative test of the cognitive vulnerability hypotheses because any variance in depressive diagnoses shared between cognitive styles and initial or past depression is allocated to initial symptoms and the past depression, even though the cognitive theories predict that such shared variance should exist (Alloy, Abramson, Raniere, & Dyller, 1999). Therefore, the magnitudes of the cognitive risk effects we obtained may be underestimates of the true effect sizes in nature. That negative cognitive styles predicted onsets of major, minor, and hopelessness depression during the follow-up period despite controlling for initial depressive symptoms and any past depressive disorder provides an especially important test of the cognitive vulnerability hypotheses because the test is truly prospective, uncontaminated by initial depression or prior history of depression.

Our findings also suggest that similar cognitive vulnerabilities may underlie the first and subsequent episodes of major depression and hopelessness depression, inasmuch as we did not obtain significant Risk × Prior Depression interactions in predicting these disorders. This is consistent with hopelessness (Abramson et al., 1989) and Beck's (1967) theories, which suggest that negative inferential styles and dysfunctional attitudes, respectively, are relatively enduring vulnerabilities (although not immutable, see Just et al., 2001) that should continually maintain individuals' increased risk for depression across first episodes and recurrences. It is interesting to note, however, that the Risk × Prior Depression interaction was significant in predicting the likelihood of an onset of RDC minor depression. The pattern of this interaction indicated that the magnitude of the risk group differences was greater for first onsets than recurrences of minor depression (see also Table 3). It may be that for milder forms of depression, such as RDC minor depression, subsyndromal depressive symptoms may play a more important role in contributing to recurrent episodes than to first onsets. This hypothesis is consistent with Lewinsohn et al.'s (1999) finding that dysfunctional attitudes combined with depressive symptoms predicted recurrences of depression but not first onsets. However, given that cognitive risk marginally predicted ($p < .06$) recurrences of minor depression among participants with a past history of depression, this finding for minor depression needs to be replicated before any definitive conclusions are drawn about cognitive vulnerability's differential prediction of first onsets versus recurrences for milder forms of depressive disorder.

Whereas the HR group was more likely than the LR group to experience prospective onsets of depressive disorders, the risk groups did not differ in the likelihood of onset of anxiety disorders without comorbid depression or other Axis I disorders. HR participants were more likely than LR participants to have an onset of anxiety comorbid with depression. This specificity is impressive given that the effect was obtained despite controlling for prospective onset of any anxiety disorder in the analyses, again a very conservative test. These findings suggest that the maladaptive cognitive styles featured as vulnerabilities in hopelessness and Beck's (1967) theories confer specific risk for depression but not other disorders unless they co-occur with depression. As such, the findings are consistent with the prediction from the helplessness–hopelessness theory of anxiety–depression comorbidity (Alloy et al., 1990), as well as with many (Alloy & Clements, 1998; Alloy et al., 1997, 2000; Gladstone et al., 1997; Hankin et al., 2004;

Lewinsohn et al., 1997, Metalsky & Joiner, 1997; Robinson et al., 1995; Weiss et al., 1998), but not all (Cole & Turner, 1993; Haeffel et al., 2003; Hammen et al., 1988; Luten et al., 1997; Ralph & Mineka, 1998), prior studies that have found specificity of cognitive vulnerability or the Cognitive Vulnerability × Stress interaction for depression. One limitation of many of these prior studies is that they used symptom measures saturated with high levels of negative affect and that contain a mix of anxiety and depression symptoms (but see Hankin et al., 2004, and Ralph & Mineka, 1998, for exceptions). Given our finding that negative cognitive styles predicted prospective onsets of anxiety disorders comorbid with depression, but not pure anxiety disorders, it is not surprising that some of these prior studies found that negative cognitive styles predicted anxiety as well as depression.

It is important to comment on the fact that we did not obtain gender differences in the likelihood of onset of any of the depressive disorders. Although epidemiological findings document a 2:1 ratio of depression in women versus men from adolescence through adulthood (Hankin et al., 1998; Hankin & Abramson, 2001; Nolen-Hoeksema, 1987, 1990), this usual gender difference is often not found in college student samples such as ours (Nolen-Hoeksema, 1987, 1990). Moreover, a potentially important reason why we did not obtain gender differences in prospective onsets of depression in our sample is that participants were selected for the CVD Project on the basis of extreme scores on measures of negative cognitive styles. Thus, within the HR and LR groups, women and men had extremely negative or positive attitudes and inferential styles, respectively. Given that cognitive styles were a powerful predictor of prospective onsets of depression and that the selection procedures resulted in female and male participants being equivalent on cognitive styles, there was little possibility of obtaining gender differences in incidence of prospective depression during the follow-up in this sample.

Despite the many strengths of the CVD Project and the use of the powerful behavioral high-risk design, there are several important limitations that should be noted as well. First, although the prospective design can demonstrate temporal precedence for negative cognitive styles as vulnerabilities to depression, it cannot establish a causal role for these styles. Studies are needed to determine whether manipulation of these cognitive styles in the context of preventive interventions causally affects the likelihood of future depression.

This study also cannot determine whether the vulnerability to depression associated with HR status is attributable to negative inferential styles, dysfunctional attitudes, or both. This is because cognitive risk status in the CVD Project was based on both the CSQ and DAS and, therefore, negative inferential styles and dysfunctional attitudes were too highly correlated in this sample to examine their separate effects. For the same reason, we also cannot examine the separate predictive power of the components (inferences about causes, consequences, or self) of the negative inferential style. In prior studies that have examined the two types of cognitive vulnerability (negative inferential styles and dysfunctional attitudes) separately, Haeffel et al. (2003) found that negative inferential styles were the more powerful of the two vulnerabilities in predicting lifetime depression whereas Lewinsohn et al. (2001) obtained greater support for dysfunctional attitudes in combination with stress in predicting prospective depression. In yet a

third set of studies, Hankin et al. (2004) found that negative inferential styles and dysfunctional attitudes were equally predictive of prospective depression in combination with stressful events and neither cognitive vulnerability–stress component uniquely predicted depression when controlling for the other. Thus, both types of maladaptive cognitive patterns may be important vulnerabilities for depression, and resolution of this issue awaits further prospective studies designed to pit the two vulnerabilities against each other.

Another potential limitation of our study is the reliance on self-report measures of cognitive vulnerability. However, Alloy, Abramson, Murray, Whitehouse, and Hogan (1997) found that the HR and LR participants differ in their self-referent information processing on a battery of behavioral tasks and, thus, provide construct validity for the self-report measures of cognitive styles. Finally, the present findings are based on a sample of university freshmen, chosen because they are at an age of risk for first onsets of depression. The generalizability of these findings to other community samples remains an open question.

In conclusion, our findings provide important new evidence that negative inferential styles and dysfunctional attitudes confer significant vulnerability to first onsets and recurrences of depressive disorders. Moreover, the vulnerability associated with negative cognitive styles has some specificity to depressive episodes. An important next step is to examine whether these negative cognitive styles combine with prospectively occurring negative life events to predict prospective onsets of depressive episodes.[5] Thus, there is need for further development of interventions, such as cognitive–behavioral therapy, designed to prevent the formation and consolidation of negative cognitive styles or to remediate these styles once fully formed.

---

[5] We have not included tests of the cognitive vulnerability–stress hypothesis in this article for three reasons. First, the CVD Project was specifically designed to test the cognitive vulnerability hypotheses of hopelessness and Beck's (1967) theories on their own, as well as the vulnerability–stress hypothesis. Thus, presentation of the prospective vulnerability results should precede presentation of any vulnerability–stress findings. Second, the present examination of whether cognitive vulnerability predicts clinically significant depressive disorders, first onsets versus recurrences of depressive episodes, and the specificity of the cognitive vulnerability effect to depression versus other disorders are all major issues in and of themselves. Finally, the role of stressful life events and consideration of the type of vulnerability–stress combination (synergistic vs. titration model) that best predicts depressive episodes is very important and complicated and deserves full exploration in a separate article.

References

Abela, J. R. Z. (2001). The hopelessness theory of depression: A test of the diathesis-stress and causal mediation components in third and seventh grade children. *Journal of Abnormal Child Psychology, 29,* 241–254.

Abela, J. R. Z., & Sarin, S. (2002). Cognitive vulnerability to hopelessness depression: A chain is only as strong as its weakest link. *Cognitive Therapy and Research, 26,* 811–829.

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

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

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

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

Alloy, L. B., Abramson, L. Y., Raniere, D., & Dyller, I. M. (1999). Research methods in adult psychopathology. In P. C. Kendall, J. N. Butcher, & G. N. Holmbeck (Eds.), *Handbook of research methods in clinical psychology* (2nd ed., pp. 466–498). New York: Wiley.

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

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

Alloy, L. B., Kelly, K. A., Mineka, S., & Clements, C. M. (1990). Comorbidity in anxiety and depressive disorders: A helplessness-hopelessness perspective. In J. D. Maser & C. R. Cloninger (Eds.), *Comorbidity in anxiety and mood disorders* (pp. 499–543). Washington, DC: American Psychiatric Press.

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

Alloy, L. B., Reilly-Harrington, N., Fresco, D., Whitehouse, W. G., & Zechmeister, J. S. (1999). Cognitive styles and life events in subsyndromal unipolar and bipolar disorders; Stability and prospective prediction of depressive and hypomanic mood swings. *Journal of Cognitive Psychotherapy: An International Quarterly, 13,* 21–40.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., revised). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Beck, A. T. (1967). *Depression: Clinical, experimental, and theoretical aspects.* New York: Harper & Row.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression.* New York: Guilford Press.

Beck, A. T., Steer, R. A., & Garbin, M. G. (1988). Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. *Clinical Psychology Review, 8,* 77–100.

Brown, G. P., Hammen, C. L., Craske, M. G., & Wickens, T. D. (1995). Dimensions of dysfunctional attitudes as vulnerabilities to depressive symptoms. *Journal of Abnormal Psychology, 104,* 431–435.

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

Cole, D. A., & Turner, J. E., Jr. (1993). Models of cognitive mediation and moderation in child depression. *Journal of Abnormal Psychology, 102,* 271–281.

Conley, C. S., Haines, B. A., Hilt, L. M., & Metalsky, G. I. (2001). The Children's Attributional Style Interview: Developmental tests of cognitive diathesis-stress theories of depression. *Journal of Abnormal Child Psychology, 29,* 445–463.

Coyne, J. C., & Gotlib, I. H. (1983). The role of cognition in depression: A critical appraisal. *Psychological Bulletin, 94,* 472–505.

Daley, S. E., Hammen, C., & Rao, U. (2000). Predictors of first onset and recurrence of major depression in young women during the 5 years following high school graduation. *Journal of Abnormal Psychology, 109,* 525–533.

Depue, R. A., Slater, J., Wolfstetter-Kausch, H., Klein, D., Goplerud, E., & Farr, D. (1981). A behavioral paradigm for identifying persons at risk for bipolar depressive disorder: A conceptual framework and five validation studies [Monograph]. *Journal of Abnormal Psychology, 56,* 431–445.

Dixon, J. F., & Ahrens, A. H. (1992). Stress and attributional styles as predictors of self-reported depression in children. *Cognitive Therapy and Research, 16,* 623–634.

Dykman, B. M., & Johll, M. (1998). Dysfunctional attitudes and vulnerability to depressive symptoms: A 14-week longitudinal study. *Cognitive Therapy and Research, 22,* 337–352.

Endicott, J., & Spitzer, R. A. (1978). A diagnostic interview: The Schedule for Affective Disorders and Schizophrenia. *Archives of General Psychiatry, 35,* 837–844.

Gladstone, T. R. G., Kaslow, N. J., Seeley, J. R., & Lewinsohn, P. M. (1997). Sex differences, attributional style, and depressive symptoms among adolescents. *Journal of Abnormal Child Psychology, 25,* 297–305.

Haeffel, G. J., Abramson, L. Y., Voelz, Z. R., Metalsky, G. I., Halberstadt, L., Dykman, B. M., et al. (2003). Cognitive vulnerability to depression and lifetime history of Axis I psychopathology: A comparison of negative cognitive styles (CSQ) and dysfunctional attitudes (DAS). *Journal of Cognitive Psychotherapy: An International Quarterly, 17,* 3–22.

Hammen, C., Adrian, C., & Hiroto, D. (1988). A longitudinal test of the attributional vulnerability model in children at risk for depression. *British Journal of Clinical Psychology, 27,* 37–46.

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

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

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

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

Hilsman, R., & Garber, J. (1995). A test of the cognitive diathesis-stress model of depression in children: Academic stressors, attributional style, perceived competence, and control. *Journal of Personality and Social Psychology, 69,* 370–380.

Ingram, R. E., Miranda, J., & Segal, Z. V. (1998). *Cognitive vulnerability to depression.* New York: Guilford Press.

Joiner, T. E., Metalsky, G. I., Lew, A., & Klocek, J. (1999). Testing the causal mediation component of Beck's theory of depression: Evidence for specific mediation. *Cognitive Therapy and Research, 23,* 401–412.

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

Just, N., Abramson, L. Y., & Alloy, L. B. (2001). Remitted depression studies as tests of the cognitive vulnerability hypotheses of depression onset: A critique and conceptual analysis. *Clinical Psychology Review, 21,* 63–83.

Klocek, J. W., Oliver, J. M., & Ross, M. J. (1997). The role of dysfunctional attitudes, negative life events, and social support in the prediction of depressive dysphoria: A prospective longitudinal study. *Social Behavior and Personality, 25,* 123–136.

Kovacs, M., & Beck, A. T. (1977). Empirical-clinical approach toward a definition of childhood depression. In J. G. Schulterbrandt & A. Raskin (Eds.), *Depression in childhood: Diagnosis, treatment, and conceptual models* (pp. 1–25). New York: Raven Press.

Kwon, S. M., & Oei, T. P. (1992). Differential causal roles of dysfunctional attitudes and automatic thoughts in depression. *Cognitive Therapy and Research, 16,* 309–328.

Lewinsohn, P. M., Allen, N. B., Seeley, J. R., & Gotlib, I. H. (1999). First onset vs recurrence of depression: Differential processes of psychosocial risk. *Journal of Abnormal Psychology, 108,* 483–489.

Lewinsohn, P. M., Gotlib, I. H., & Seeley, J. R. (1997). Depression-related psychosocial variables: Are they specific to depression in adolescents? *Journal of Abnormal Psychology, 106,* 365–375.

Lewinsohn, P. M., Joiner, T. E., & Rohde, P. (2001). Evaluation of cognitive diathesis-stress models in predicting major depressive disorder in adolescents. *Journal of Abnormal Psychology, 110,* 203–215.

Lewinsohn, P. M., Roberts, R. E., Seeley, J. R., Rohde, P., Gotlib, I. H., & Hops, H. (1994). Adolescent psychopathology: II. Psychosocial risk factors for depression. *Journal of Abnormal Psychology, 103,* 302–315.

Lewinsohn, P. M., Steinmetz, J. L., Larson, D. W., & Franklin, J. (1981). Depression-related cognitions: Antecedent or consequence? *Journal of Abnormal Psychology, 90,* 213–219.

Luten, A., Ralph, J., & Mineka, S. (1997). Depressive attributional style: Is it specific to depression vs. anxiety vs. negative affect? *Behaviour Research and Therapy, 35,* 703–719.

Metalsky, G. I., Halberstadt, L. J., & Abramson, L. Y. (1987). Vulnerability to depressive mood reactions: Toward a more powerful test of the diathesis-stress and causal mediation components of the reformulated theory of depression. *Journal of Personality and Social Psychology, 63,* 667–675.

Metalsky, G. I., & Joiner, T. E. (1997). The Hopelessness Depression Symptom Questionnaire. *Cognitive Therapy and Research, 21,* 359–384.

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

Mineka, S., Pury, C., & Luten, A. (1995). Explanatory style in anxiety versus depression. In G. Buchanan & M. E. P. Seligman (Eds.), *Explanatory style* (pp. 135–158). Hillsdale, NJ: Erlbaum.

Mineka, S., Watson, D., & Clark, L. A. (1998). Comorbidity of anxiety and unipolar mood disorders. *Annual Review of Psychology, 49,* 377–412.

Nolen-Hoeksema, S. (1987). Sex differences in unipolar depression: Evidence and theory. *Psychological Bulletin, 101,* 259–282.

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

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

Olinger, L. J., Kuiper, N. A., & Shaw, B. F. (1987). Dysfunctional attitudes and stressful life events: An interactive model of depression. *Cognitive Therapy and Research, 11,* 25–40.

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

Persons, J. B., & Miranda, J. (1992). Cognitive theories of vulnerability to depression: Reconciling negative evidence. *Cognitive Therapy and Research, 16,* 485–502.

Ralph, J., & Mineka, S. (1998). Attributional style and self-esteem: The prediction of emotional distress following a midterm exam. *Journal of Abnormal Psychology, 107,* 203–215.

Reilly-Harrington, N. A., Alloy, L. B., Fresco, D. M., & Whitehouse, W. G. (1999). Cognitive styles and life events interact to predict bipolar and unipolar symptomatology. *Journal of Abnormal Psychology, 108,* 567–578.

Riskind, J. H., & Rholes, W. S. (1984). Cognitive accessibility and the

capacity of cognitions to predict future depression: A theoretical note. *Cognitive Therapy and Research, 8,* 1–12.

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

Scher, C. D., Ingram, R. E., & Segal, Z. V. (2005). Cognitive reactivity and vulnerability: Empirical evaluation of construct activation and cognitive diatheses in unipolar depression. *Clinical Psychology Review, 25,* 487–510.

Shapiro, R. W., & Keller, M. B. (1979). *Longitudinal Interval Follow-up Evaluation* (LIFE). Boston: Massachusetts General Hospital.

Spitzer, R. A. L., Endicott, J., & Robins, E. (1978). Research diagnostic criteria: Rationale and reliability. *Archives of General Psychiatry, 35,* 773–782.

Teasdale, J. D. (1983). Negative thinking in depression: Cause, effect or reciprocal relationship? *Advances in Behaviour Research and Therapy, 5,* 3–25.

Teasdale, J. D. (1988). Cognitive vulnerability to persistent depression. *Cognition and Emotion, 2,* 247–274.

Weiss, R., Susser, K., & Catron, T. (1998). Common and specific features of childhood psychopathology. *Journal of Abnormal Psychology, 104,* 3–14.

Weissman, A., & Beck, A. T. (1978). *Development and validation of the Dysfunctional Attitudes Scales: A preliminary investigation.* Paper presented at the meeting of the American Educational Research Association, Toronto, Ontario, Canada.

Zuroff, D. C., Mongrain, M., & Santor, D. A. (2004). Conceptualizing and measuring personality vulnerability to depression: Comment on Coyne and Whiffen (1995). *Psychological Bulletin, 130,* 489–511.

Appendix

Diagnostic Criteria for Depressive Disorders

DSM–IV major depression: (a) depressed mood or loss of interest ≥2 weeks (for definite) or ≥1 week (for subthreshold) for 6 out of 7 days of each week; (b) depressed mood or loss of interest ≥90% (definite) or ≥75% (subthreshold) of each depressed day; (c) ≥ 4 (definite) or ≥3 (subthreshold) criterial symptoms present, overlapping 6 out of 7 days of each week for ≥2 weeks (definite) or ≥1 week (subthreshold); and (d) symptoms cause clinically significant distress or impairment in functioning.

RDC major depression: Same as *DSM–IV* major depression except that one additional criterion symptom is required for definite and probable diagnoses.

RDC minor depression: (a) depressed ≥2 weeks (definite) or ≥1 week (probable) for 6 out of 7 days of each week; (b) depressed ≥90% (definite) or ≥50% (probable) of each depressed day; (c) ≥ 2 criterion symptoms present (definite and probable), overlapping 6 out of 7 days of each week for ≥2 weeks (definite) or ≥1 week (probable); and (d) impairment in functioning.

Hopelessness depression: (a) hopelessness ≥2 weeks (definite) or ≥1 week (probable) for 6 out of 7 days of each week; (b) ≥ 5 (definite) or ≥4 (probable) criterion symptoms present, overlapping 6 out of 7 days of each week for ≥2 weeks (definite) or ≥1 week (probable); and (c) onset of hopelessness must precede the onset of the Criterion 2 symptoms by at least 1 day and by no more than 1 week. The criterion symptoms of hopelessness depression are sadness, retarded initiation of voluntary responses, suicidality, sleep disturbance (initial insomnia), low energy, self-blame, difficulty in concentration, psychomotor retardation, brooding/worrying, lowered self-esteem, and dependency.