

The Temple-Wisconsin Cognitive Vulnerability to Depression Project: Lifetime History of Axis I Psychopathology in Individuals at High and Low Cognitive Risk for Depression

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The authors tested the cognitive vulnerability hypotheses of depression with a retrospective behavioral high-risk design. Individuals without current Axis I diagnoses who exhibited either negative or positive cognitive styles were compared on lifetime prevalence of depressive and other disorders and the clinical parameters of depressive episodes. Consistent with predictions, cognitively high-risk participants had higher lifetime prevalence than low-risk participants of major and hopelessness depression and marginally higher prevalence of minor depression. These group differences were specific to depressive disorders. The high-risk group also had more severe depressions than the low-risk group, but not longer duration or earlier onset depressions. The risk group differences in prevalence of depressive disorders were not mediated by current depressive symptoms.

Psychological research on depression over the past 20 years has focused on the potential role of maladaptive cognitive patterns as vulnerability factors for depression. Both Beck's (1967, 1987) theory and the hopelessness theory (Abramson, Metalsky, & Alloy, 1989; Alloy, Abramson, Metalsky, & Hartlage, 1988) of depression, as well as its predecessor, the reformulated helplessness theory of depression (Abramson, Seligman, & Teasdale, 1978), contain a "cognitive vulnerability hypothesis" in which individuals who exhibit dysfunctional cognitive styles are hypoth-

esized to be at increased risk for onset of depression when they experience negative life events. Specifically, according to the hopelessness theory, episodes of depression, particularly the subtype of "hopelessness depression" (HD), are more likely to develop when people interpret negative life events as being caused by stable (enduring) and global (widespread) factors, as likely to lead to other negative consequences or outcomes, and as implying that they are flawed, unworthy, or deficient than when people do not make such inferences. Similarly, in Beck's theory, people who

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possess depressive self-schemas containing dysfunctional attitudes, such as that their worth depends on being perfect or on others' approval, are hypothesized to be vulnerable to depressive episodes when they encounter negative events that impinge on these beliefs.

Cognitive Vulnerability and the Behavioral High-Risk Design

Thus, according to both of these cognitive theories of depression, people with negative cognitive styles are at greater risk for depression onset than people with positive cognitive styles. We believe that the most direct and compelling way to test this cognitive vulnerability hypothesis is with a behavioral high-risk design (Alloy, Lipman, & Abramson, 1992; Depue et al., 1981). Thus, in a retrospective version of this design, we examined whether nondisordered participants selected to be at high versus low risk for depression based on the presence versus absence of the hypothesized depressogenic cognitive styles differed in their likelihood of exhibiting depression in the past.

The logic behind this retrospective version of the behavioral high-risk design is based on two arguments. First, individuals who exhibit the hypothesized cognitive vulnerabilities for depression presumably developed these maladaptive cognitive patterns sometime in the past, thereby increasing their risk for past episodes of depression whenever these patterns were present. Consistent with this argument is evidence that attributional styles exhibit some stability over the life span (Burns & Seligman, 1989). Second, given that a past history of depression is an established predictor of future depression (e.g., Belsher & Costello, 1988), the finding of an association between increased rates of past depression and the hypothesized cognitive vulnerabilities among persons not currently in a depressive episode would provide support for the vulnerability status of these negative cognitive styles. However, there is an interpretational ambiguity inherent in the retrospective high-risk strategy. It is not possible to distinguish with certainty whether increased lifetime prevalence of depression among cognitively high-risk participants is due to the negative cognitive styles contributing to the cause of the past episodes of depression or to the past depression leading to the development of the negative cognitive styles as a consequence or "scar" (Lewinsohn, Steinmetz, Larson, & Franklin, 1981; Rohde, Lewinsohn, & Seeley, 1990; Zeiss & Lewinsohn, 1988) of the earlier depression. Nonetheless, the finding of increased lifetime prevalence of depression among those who exhibit the putative cognitive vulnerabilities for depression but are not currently in a depressive episode is consistent with, but not exclusive to, the hopelessness and Beck theories, and thus it would provide some support for these theories' cognitive vulnerability hypotheses.

In a previous study involving the retrospective behavioral high-risk design, Alloy et al. (1992) found that nondepressed undergraduates with the hypothesized depressogenic attributional style had experienced more frequent and severe episodes of major depression and HD in the previous 2 years than nondepressed students who possessed a nondepressogenic attributional style. Complementing Alloy et al.'s (1992) retrospective high-risk strategy, studies that have used the prospective high-risk design or modified versions of this design (see Alloy, Abramson, Whitehouse, et al., 1999) have tended to provide support for the vulner-

ability hypotheses of the cognitive theories of depression (e.g., Alloy & Clements, 1998; Alloy, Just, & Panzarella, 1997; Kwon & Oei, 1992; Lewinsohn et al., 1994; Metalsky, Halberstadt, & Abramson, 1987; Metalsky & Joiner, 1992; Metalsky, Joiner, Hardin, & Abramson, 1993; Nolen-Hoeksema, Girgus, & Seligman, 1986, 1992; Olinger, Kuiper, & Shaw, 1987; but see Follette & Jacobson, 1987, and Lewinsohn, Hoberman, & Rosenbaum, 1988, for examples of nonsupport). However, most of these prospective studies have demonstrated that depressogenic attributional styles or dysfunctional attitudes provide vulnerability to depressive symptoms; little is known as yet about whether these cognitive styles also provide vulnerability to clinically significant depressive disorders (but see Abramson, Alloy, Hogan, et al., 1999, and Alloy, Abramson, Whitehouse, et al., 1999).

In the present study, we sought to expand on Alloy et al.'s (1992) earlier retrospective findings in three major ways. First, we extended the retrospective time frame from the previous 2 years to lifetime history of disorder. Second, we examined whether negative cognitive styles were associated with increased lifetime prevalence of disorders that are frequently comorbid with depression (e.g., anxiety disorders and substance use disorders). An important issue for the Beck (1967, 1976) and hopelessness (Abramson et al., 1989; Alloy, Kelly, Mineka, & Clements, 1990) theories is whether dysfunctional attitudes and depressogenic inferential styles act as specific vulnerabilities for depression rather than general vulnerabilities to other forms of psychopathology as well. Finally, we explored whether five additional cognitive styles hypothesized to also confer risk for depression (described subsequently) were associated with lifetime history of depressive disorders either alone or in interaction with cognitive risk status based on dysfunctional attitudes and inferential styles for negative events.

Role of Additional Cognitive Styles in Vulnerability to Depression

Aside from negative inferential styles and dysfunctional attitudes, several theorists have proposed other cognitive or personality styles as possible additional risk factors for depression. For example, Needles and Abramson (1990) proposed that individuals who infer that positive events are due to unstable, specific causes; will not lead to further positive consequences; and do not mean that they are competent or worthy will be less likely to obtain emotional benefits from the occurrence of positive events. Thus, they will experience longer episodes of depression than individuals with an enhancing inferential style for positive events. In an extension of this reasoning, Lapkin (1995) argued that a pessimistic inferential style for positive events (i.e., unstable, specific, etc.) might also contribute additional vulnerability to depression onset by decreasing one's ability to gain hope and positive affect from the occurrence of positive events, thereby increasing one's vulnerability to the depression-triggering effects of negative life events. If this is the case, then individuals who possess a pessimistic inferential style for both negative and positive events should be more vulnerable to depression onsets than those with only the negative style for negative events.

In Beck's theory (1983, 1987), individual differences in the subjective value people place on various life experiences also contribute vulnerability for depression. People who are high in

sociotropy place great importance on intimacy, social relationships, and acceptance from others, whereas those high in autonomy value achievement, freedom, and independence. We explored whether high levels of sociotropy or autonomy (or both) would be associated with increased lifetime prevalence of depression alone or in combination with negative cognitive styles.

Much evidence supports an association between self-focused attention and depression (see Ingram, 1990, and Musson & Alloy, 1988, for reviews). Several theorists (e.g., Ingram, 1990; Pyszczynski & Greenberg, 1987) have hypothesized that chronically high levels of self-focused attention or private self-consciousness (Fenigstein, Scheier, & Buss, 1975) may increase individuals' risk for onset, maintenance, or exacerbation of depression by increasing the salience of negative aspects of oneself. Similarly, according to Nolen-Hoeksema (1991), individuals who tend to ruminate when they become depressed, focusing on internally generated thoughts and emotions associated with depression, are more likely to experience prolonged and severe depression than those who tend to distract themselves from their depressed mood, a hypothesis supported by several studies (e.g., Just & Alloy, 1997; Nolen-Hoeksema & Morrow, 1991; Nolen-Hoeksema & Morrow, 1993; Nolen-Hoeksema, Morrow, & Fredrickson, 1993; Nolen-Hoeksema, Parker, & Larson, 1994). In an extension of Nolen-Hoeksema's logic, Robinson (1997; see also Zullo & Seligman, 1990) argued that individuals who both tend to make negative inferences and ruminate about these negative cognitions in response to the occurrence of stressful life events (stress-reactive rumination) may be more likely to develop an episode of depression in the first place. Thus, we also examined whether the tendency to engage in self-focused attention (private self-consciousness) or stress-reactive rumination would moderate the association between negative cognitive styles and lifetime prevalence of depression.

Study Overview and Hypotheses

Consequently, in this article, we present data from the retrospective portion of the Temple-Wisconsin Cognitive Vulnerability to Depression Project (CVD Project; Alloy & Abramson, 1999). In the CVD Project, university freshmen who did not meet criteria for any current Axis I disorder at the outset of the study were selected to be at high or low cognitive risk for depression on the basis of the presence versus absence of depressogenic inferential styles for negative events and dysfunctional attitudes. Here we report on the lifetime history of depressive and other Axis I disorders in the high-risk and low-risk participants at the outset of the project. On the basis of the cognitive vulnerability hypotheses of hopelessness and Beck's theories of depression and the logic of the retrospective high-risk design (Alloy et al., 1992), we predicted that the high-risk group (HR group) would exhibit higher lifetime prevalence (probability of occurrence) of depressive disorders and of the subtype of HD than would the low-risk group (LR group).¹ In contrast, we predicted no risk group differences in lifetime prevalence of other Axis I disorders.

In addition to these two main hypotheses, we also conducted two other sets of exploratory analyses. Although the cognitive theories of depression are silent about whether individuals' cognitive styles will influence the clinical parameters of their depressive episodes, we compared the age of onset and severities and

durations of past episodes of depression in cognitively low-risk versus high-risk participants. We also explored whether inferential styles for positive events, sociotropy, autonomy, private self-consciousness, or stress-reactive rumination were associated with lifetime prevalence of depression either alone or in interaction with cognitive risk status based on dysfunctional attitudes and inferential style for negative events.

Method

Participants

Sample selection. Between September 1990 and June 1992, 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 administered equivalently at both sites. Details of the selection procedure and rationale for use of a freshman sample are provided by Alloy and Abramson (1999). In brief, in Phase 1 of screening, 5,378 freshmen (2,438 at TU and 2,940 at UW) completed the Cognitive Style Questionnaire (CSQ), a revision of the Attributional Style Questionnaire (ASQ; Peterson, 1991; Seligman, Abramson, Semmel, & von Baeyer, 1979) that assesses styles for inferring causes, consequences, and characteristics about the self for hypothetical positive and negative life events; a revised version of the Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978); and a personal data sheet that requested demographic data and contact information. The CSQ and DAS were designed to assess the vulnerabilities featured in the hopelessness and Beck theories, respectively, and were used to identify freshmen at high or low generic cognitive risk for depression on the basis of both theories. Inasmuch as these two theories share many similarities (Alloy & Abramson, 1999), the overall goal of the CVD Project was to test the etiological hypotheses of the theories simultaneously rather than to pit them against one another. On the basis of the Phase 1 screening, we identified 619 potential high-risk (261 at TU and 358 at UW) and 585 potential low-risk (234 at TU and 351 at UW) participants. The criteria for the two risk groups were as follows.

To be included in the HR group, individuals at each site separately had to have scores in the highest quartile (most negative) of the Phase 1 screening sample on both the DAS (high-risk item score cut point ≥ 3.69 at TU and ≥ 3.81 at UW) and the composite of the stability, globality, consequences, and self dimensions for negative events on the CSQ (high-risk item score cut point ≥ 4.43 at TU and ≥ 4.50 at UW). To be included in the LR group, individuals at each site separately had to have scores in the lowest quartile (most positive) of the Phase 1 screening sample on both the DAS (low-risk item score cut point ≤ 2.60 at TU and ≤ 2.86 at UW) and the CSQ composite for negative events (low-risk item score cut point ≤ 3.30 at TU and ≤ 3.47 at UW). The overall Phase 1 sample item score means were 3.19 ($SD = 0.80$) and 3.35 ($SD = 0.74$) for the DAS at TU and UW, respectively, and 3.85 ($SD = 0.86$) and 3.98 ($SD = 0.77$) for the CSQ negative event composite at TU and UW, respectively.

A random subset of freshmen who were less than 30 years old and met the Phase 1 criteria for the HR group ($n = 313$: 167 at TU and 146 at UW) or the LR group ($n = 236$: 130 at TU and 106 at UW) were invited for the Phase 2 screening. In Phase 2, participants were administered an expanded version of the Schedule for Affective Disorders and Schizophrenia-

¹ Given that Abramson et al. (1989) specified that the HD syndrome cuts across currently diagnosed categories of clinical depression (e.g., major depression, minor depression, and dysthymia) and is distinguished only from certain other hypothesized subtypes of depression (e.g., DSM melancholic depression or "endogenomorphic depression"; Klein, 1974) not assessed retrospectively in this study, we predicted risk group differences in lifetime prevalence of all depressive disorders assessed in the present study.

Lifetime (SADS-L) interview (Endicott & Spitzer, 1978) and also completed a number of self-report measures of depression and other psychopathology, including the Beck Depression Inventory (BDI; Beck, Rush, Shaw, & Emery, 1979). The SADS-L interviews were conducted by research assistants unaware of participants' risk group status. Phase 2 participants were excluded from the final sample if they had any of the following disorders based on the expanded SADS-L interview and the application of criteria of the *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): (a) current *DSM-III-R* or RDC diagnosis of any episodic mood disorder (e.g., major or minor depressive disorder, manic or hypomanic episode, or bipolar disorder with a current episode of major depressive disorder, mania, or hypomania) or any chronic mood disorder (e.g., dysthymia, intermittent depressive disorder, or cyclothymia), (b) current *DSM-III-R* or RDC diagnosis of any other Axis I psychiatric disorder (e.g., anxiety disorder or substance use disorder), (c) current psychotic symptoms, (d) past history of mania, hypomania, bipolar disorder, or cyclothymia, and (e) serious medical illness that would preclude participation in a longitudinal study.

In addition to participants with no lifetime history of any Axis I disorder, those with a past mood disorder (e.g., past major or minor depressive disorder) but who had remitted for a minimum of 2 months were also retained in the final sample. The minimum 2-month remission from a past depression was designed to ensure that participants' cognitive styles were assessed in a nondepressed state and to ensure that any depression onsets during the prospective phase of the project were new episodes and not relapses of prior depression. In fact, among 139 participants with at least one past episode of major or minor depressive disorder, 102 (73.4%) had experienced this episode more than 6 months before the Phase 1 screening, 32 (23.0%) had experienced the episode 4–6 months before, and 5 (3.6%) had experienced the episode 2–3 months before. These percentages did not differ between the HR and LR groups, $\chi^2(2, N = 139) = 3.34, ns$. On average, the most recent past episode of depression was 843 days (2.31 years; $SD = 891$ days) before Phase 1.

Our logic in also including participants who were not currently in a depressed episode but had experienced past depression is that by excluding such people, 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. However, we controlled for any current depressive symptoms that might be associated with cognitive high-risk status at screening in all tests of the cognitive vulnerability hypotheses. We did exclude participants with a past history of bipolar spectrum disorders because Beck's theory and hopelessness theory were originally designed to be applicable to unipolar depression (but see Alloy, Reilly-Harrington, Fresco, Whitehouse, & Zechmeister, 1999; and Reilly-Harrington, Alloy, Fresco, & Whitehouse, 1999).

The 209 eligible high-risk (114 at TU and 95 at UW) and 207 eligible low-risk (110 at TU and 97 at UW) freshmen who met all inclusion and exclusion criteria at the end of Phase 2 were invited to participate in the prospective phase of the CVD Project. Eighteen eligible high-risk (14 at TU and 4 at UW) and 13 eligible low-risk (9 at TU and 4 at UW) participants refused to take part in the prospective phase, and another 19 high-risk (17 at TU and 2 at UW) and 18 low-risk (14 at TU and 4 at UW) participants were dropped (as a result of inability to locate, five or more missed appointments, or poor English-speaking ability).² The final CVD Project sample included 173 high-risk (83 at TU and 90 at UW) and 176 low-risk (87 at TU and 89 at UW) freshmen. The present investigation was based on this final sample. Within 1 month of the Phase 2 screening, members of the final sample were administered a Time 1 assessment that included completion of the Sociotropy Autonomy Scales (SAS; Beck, Epstein, Harrison, & Emery, 1983), the Self-Consciousness Scale (SCS; Fenigstein et al., 1975), and the Stress-Reactive Rumination Scale (SRRS;

Robinson, 1997; Robinson & Alloy, 1999),³ and then they entered the prospective phase of the project.

Sample demographic and cognitive style characteristics. Table 1 shows the demographic and cognitive style characteristics of the final samples at each site. With respect to site differences, the two cohorts were comparable in regard to gender ratio and DAS, CSQ, SAS, and SCS Private subscale scores but differed on ethnic composition, socioeconomic status (SES), age, and screening BDI scores. The TU cohort had a higher proportion of minority participants (37.1%) than did the UW cohort (6.1%), $\chi^2(1) = 47.97, p < .001$. The TU cohort also had lower mean parental education, $F(1, 331) = 33.57, p < .001$, and income, $F(1, 261) = 16.06, p < .001$, and was older, $F(1, 341) = 24.50, p < .001$, than the UW cohort. Finally, the TU cohort had higher initial BDI scores than the UW cohort, $F(1, 340) = 16.16, p < .001$ (see Table 1 for means). To the extent that the present findings replicate across the sites, the site differences in SES and ethnic composition should increase the generalizability of our results.

With regard to risk group and sex differences, the HR and LR groups did not differ on gender or ethnic composition; however, the combined (across sites) LR group was older than the combined HR group, $F(1, 341) = 10.91, p < .002$, and the men were older than the women, $F(1, 341) = 6.14, p < .02$. In addition, there was a Risk \times Site interaction for age, $F(1, 341) = 9.83, p < .002$; the LR group at TU was older than the LR group at UW and the HR groups at both sites (see Table 1). Given that the likelihood of experiencing depressive or other disorders increases with age, the fact that the LR group was older than the HR group works against the hypothesis of higher rates of past depression in the HR group. Nevertheless, we controlled for age differences in our statistical analyses. The HR group also had higher screening BDI scores than the LR group, $F(1, 340) = 174.76, p < .001$ (see Table 1); thus, we controlled for these current depressive symptom differences in all analyses as well.

Although they were not selected on the basis of the additional cognitive style measures, the risk groups also differed significantly on sociotropy, $F(1, 340) = 266.70, p < .001$; private self-consciousness, $F(1, 340) = 26.21, p < .001$; stress-reactive rumination, $F(1, 151) = 33.81, p < .001$; and the CSQ positive events composite, $F(1, 340) = 40.07, p < .001$. The HR group was more sociotropic, self-conscious, and ruminative than the LR group but had a more enhancing inferential style for positive events (see Table 1 for means). In addition, women ($M = 64.98, SD = 21.02$) were more sociotropic than men ($M = 60.16, SD = 19.65$), $F(1, 340) = 5.20, p < .05$. Table 2 displays the correlations among risk status, BDI scores, and each of the additional cognitive style scores. Given some overlap between risk status and the additional cognitive style measures, in the analyses that explored the associations of these other cognitive styles with lifetime history of depression, we examined the unique effects of each cognitive style independent of cognitive risk status based on the DAS and CSQ composite for negative events.

Sample representativeness. The final sample was representative of the original Phase 1 screening sample on age and ethnic composition but had a higher proportion of women (67.1% at TU and 68.2% at UW) than did the Phase 1 sample (56.8% at TU and 60.7% at UW), $\chi^2(1) = 9.86, p < .01$. In addition, participants in the final sample did not differ significantly from Phase 2 eligible participants who either refused participation or were dropped on demographics or cognitive style (CSQ and DAS scores). Thus,

² The number of eligible individuals who refused participation in the prospective phase was higher at TU than at UW because UW participants were given the opportunity to refuse participation even before their eligibility was established at Phase 2. The number of eligible participants dropped was also greater at TU than UW primarily as a result of poor English-speaking ability.

³ The SRRS was administered only at the TU site; thus, analyses involving this measure included only the TU cohort.

Table 1
Final Sample: Demographic and Cognitive Style Characteristics

| Characteristic | High risk | | | Low risk | | |
|-------------------------------|-----------|-----------|------|----------|-----------|------|
| | <i>M</i> | <i>SD</i> | % | <i>M</i> | <i>SD</i> | % |
| Temple site ^a | | | | | | |
| Age (years) | 18.45 | 1.40 | | 19.57 | 2.98 | |
| Parental education (years) | 13.76 | 2.47 | | 13.43 | 2.26 | |
| Combined parental income (\$) | 48,061 | 36,013 | | 39,354 | 25,819 | |
| Female | | | 67.5 | | | 66.7 |
| Caucasian | | | 68.3 | | | 57.7 |
| BDI total score | 15.32 | 9.76 | | 5.00 | 4.44 | |
| DAS item score | 4.39 | 0.55 | | 2.17 | 0.29 | |
| CSQ NEG COMP item score | 5.05 | 0.47 | | 2.71 | 0.43 | |
| CSQ POS COMP item score | 5.42 | 0.79 | | 5.04 | 0.78 | |
| SAS SOC total score | 74.80 | 16.91 | | 49.18 | 15.18 | |
| SAS AUT total score | 72.86 | 14.47 | | 75.06 | 12.36 | |
| SCS Private subscale score | 25.94 | 5.26 | | 23.46 | 5.42 | |
| SRRS NEG INF total score | 427.49 | 199.49 | | 248.32 | 165.81 | |
| Wisconsin site ^b | | | | | | |
| Age (years) | 18.67 | 0.37 | | 18.77 | 1.14 | |
| Parental education (years) | 15.20 | 2.17 | | 15.03 | 2.27 | |
| Combined parental income (\$) | 82,911 | 100,473 | | 71,782 | 53,219 | |
| Female | | | 68.9 | | | 67.4 |
| Caucasian | | | 95.6 | | | 92.1 |
| BDI total score | 12.11 | 7.14 | | 3.20 | 2.44 | |
| DAS item score | 4.50 | 0.44 | | 2.23 | 0.33 | |
| CSQ NEG COMP item score | 5.15 | 0.40 | | 2.78 | 0.37 | |
| CSQ POS COMP item score | 5.58 | 0.51 | | 5.00 | 0.79 | |
| SAS SOC total score | 79.66 | 13.55 | | 50.26 | 15.72 | |
| SAS AUT total score | 69.40 | 11.19 | | 72.58 | 10.71 | |
| SCS Private subscale score | 25.75 | 5.15 | | 22.51 | 5.07 | |
| SRRS NEG INF total score | | | | | | |

Note. The SRRS was not given at the Wisconsin site. BDI = Phase I Beck Depression Inventory; DAS = Dysfunctional Attitudes Scale; CSQ NEG COMP and POS COMP = Cognitive Style Questionnaire negative events and positive events composites, respectively; SAS SOC and AUT = Sociotropy Autonomy Scales Sociotropy and Autonomy subscales, respectively; SCS = Self-Consciousness Scale; SRRS NEG INF = Stress-Reactive Rumination Scale Negative Inferences subscale.
^a High-risk *n* = 83, low-risk *n* = 87. ^b High-risk *n* = 90, low-risk *n* = 89.

the final sample was generally representative of the population from which it was drawn on demographics (but, obviously, not on cognitive styles) and appeared to be unbiased in important respects relative to other eligible persons who did not participate (see Alloy & Abramson, 1999, for further details).

Diagnostic Procedure

Diagnostic interview. Lifetime diagnoses and their clinical parameters (e.g., severity and duration) were based on information obtained from the expanded SADS-L interviews given at the Phase 2 screening, with decision rules specified by RDC and *DSM-III-R* criteria. Interviewers were unaware of participants' risk group status. The original SADS-L interview (Endicott & Spitzer, 1978) was expanded for the CVD Project in the following ways: (a) We added additional probes to allow for the assignment of *DSM-III-R* as well as RDC diagnoses; (b) we added additional probes that assessed the precise number of days participants felt depressed and the percentage of waking hours of each depressed day they felt depressed; (c) we expanded and improved on the probes in the anxiety disorders section by incorporating aspects of the Anxiety Disorders Interview Schedule (DiNardo & Barlow, 1988); and (d) we grouped together all items relevant to a given diagnosis and presented items relevant to assess-

ing past episodes of a disorder immediately after the items for a current episode of that disorder (participants found this modified format less confusing). We also constructed an interview-based index of current depressive symptoms from the current depression section of the SADS-L and used this index (along with the screening BDI) as an additional covariate in our analyses.

In consultation with Jean Endicott's group at the New York State Psychiatric Institute (the developers of the SADS), we specified the degree of persistence of depressed mood or pervasive loss of interest and the minimum number of days of overlapping symptoms required more explicitly than did the RDC and *DSM-III-R*.⁴ Thus, our diagnoses may be somewhat stricter than the RDC and *DSM-III-R* for depressive disorders. We also established explicit project criteria for diagnosing the hypothesized subtype of HD (Abramson et al., 1989). The Appendix shows the specific criteria for each depressive disorder. Note that whereas some of the symptoms hypothesized to be part of the HD syndrome (e.g., sadness and

⁴ We developed these more explicit persistence and symptom overlap criteria in consultation with Endicott's group because the RDC and *DSM-III-R* criteria were ambiguous with regard to these dimensions.

Table 2
Correlations Among Cognitive Risk, Other Cognitive Styles, and Depressive Symptoms

| Variable | 1 | 2 | 3 | 4 | 5 | 6 | 7 |
|------------|---------|---------|----------|--------|---------|---------|---|
| 1. Risk | — | | | | | | |
| 2. CSQ POS | .32**** | — | | | | | |
| 3. SOC | .67**** | .34**** | — | | | | |
| 4. AUT | -.11** | -.06 | -.27**** | — | | | |
| 5. SCPRIV | .27**** | .19**** | .27**** | .14*** | — | | |
| 6. SRRS | .44**** | .22*** | .51**** | -.11 | .20*** | — | |
| 7. BDI | .59**** | .03 | .41**** | -.04 | .24**** | .28**** | — |

Note. $n = 349$ for all correlations except those involving the SRRS, for which $n = 159$. Risk (0 = low risk, 1 = high risk) = cognitive risk; CSQ POS = Cognitive Style Questionnaire positive events composite; SOC = Sociotropy Autonomy Scales Sociotropy subscale; AUT = Sociotropy Autonomy Scales Autonomy subscale; SCPRIV = Self-Consciousness Scale Private subscale; SRRS = Stress-Reactive Rumination Scale Negative Inferences subscale; BDI = Beck Depression Inventory.
** $p < .05$. *** $p < .01$. **** $p < .001$.

suicidal ideation) are completely overlapping with symptoms that are part of DSM and RDC criteria for major depression, others only partially overlap with symptoms for major depression (e.g., retarded initiation of voluntary responses). In addition, some symptoms currently described as part of major depression, such as anhedonia, irritability, guilt, and appetite disturbance, are not hypothesized to be part of the HD syndrome (Abramson et al., 1989; Alloy & Clements, 1998).

Diagnostic training, calibration, and reliability. Diagnostic interviewers completed an intensive training program for administering the SADS-L interviews and for assigning DSM-III-R and RDC diagnoses that was modeled after ideal programs (Amenson & Lewinsohn, 1981; Gibbon, McDonald-Scott, & Endicott, 1981). The training program consisted of approximately 200 hr of didactic instruction and homework, training on case vignettes and videotaped interviews, role-playing, extensive practice conducting live interviews, and regular exams that had to be passed. Throughout the project, interviewers received extensive individual feedback. In addition, we calibrated our diagnoses across interviewers within and between sites, as well as with recognized diagnostic experts⁵ (see Alloy & Abramson, 1999, for further details). We conducted an interrater reliability study on approximately 15% ($n = 80$) of the SADS-L interviews. On the basis of joint ratings of these 80 randomly selected interviews, the kappa coefficient (Cohen, 1960) was .90 or above for all project diagnoses.

Assessment of Dependent Variables

For each dependent measure derived from the SADS-L interviews, we combined definite and probable diagnoses of each disorder. Lifetime prevalence was operationalized as the probability of having had at least one episode of the disorder during the participant's lifetime. Severity of episodes was operationalized in two ways: (a) the number of RDC, DSM-III-R, or HD criterial symptoms the participant exhibited during the episode (e.g., of 8 symptoms that form the symptom criterion for DSM-III-R major depressive disorder, the number the participant showed) and (b) the total number of symptoms of a disorder (both criterial and noncriterial) the participant exhibited (e.g., of 17 possible depression symptoms). Duration of episodes was calculated in weeks. Age of onset was defined as the participant's age at the time of the earliest lifetime episode of any category of disorder (e.g., the earliest age of any diagnosed depressive disorder).

Instruments for Assessing Independent Variables

Cognitive Style Questionnaire. Participants' inferential styles for positive and negative events were assessed with the CSQ (Abramson, Metalsky, & Alloy, 1999), a modified version of the ASQ (Peterson et al., 1982;

Seligman et al., 1979). The ASQ is a well-established instrument with good reliability and validity (Peterson, 1991) that assesses people's attributions for hypothetical positive and negative events on the internality, stability, and globality dimensions. The CSQ was modified from the ASQ by increasing the number of events to 12 positive and 12 negative (6 achievement and 6 interpersonal events of each valence) events and by including ratings (on 7-point scales) of the probable consequences of each event (e.g., "How likely is it that the other person no longer wanting a romantic relationship with you will lead to other negative things happening to you?") and the implications of each event for the self (e.g., "To what degree does your receiving a negative evaluation of your job performance mean to you that you are flawed in some way?"). In the CVD Project, a composite score for negative events based on a sum of the stability, globality, consequences, and self dimensions was used (along with the DAS) to select HR and LR groups as described earlier. The same composite for positive events was used as the measure of individuals' inferential style for positive events. Alpha coefficients based on the Phase 1 screening sample ($n = 5,378$) for the negative and positive event composites were .88 and .86, respectively. Both retest stabilities over a 1-year interval based on the final sample ($n = 349$) were .80.

Dysfunctional Attitudes Scale. The DAS (Weissman & Beck, 1978) contains 40 items that assess dysfunctional attitudes regarding perfectionistic standards of performance, concern with evaluation by others, causal attributions, expectations about the likelihood of desired outcomes, and attachment of high importance to particular goals. For the CVD Project, we expanded the DAS by adding 24 items of relevance to college students that measured dysfunctional beliefs in achievement and interpersonal domains specifically (e.g., "If I fail in school or work, then I am a failure as a person" and "I am a nobody if my closest friend stops liking me"). The expanded DAS score was used (along with the CSQ) to select HR and LR groups. Reliability and validity for the original 40-item DAS are adequate (Hammen & Krantz, 1985; Weissman & Beck, 1978). The alpha coefficient for the expanded DAS was .90 in our Phase 1 screening sample, and retest reliability over 1 year in the final sample was .78.

Sociotropy-Autonomy Scales. The SAS (Beck et al., 1983) contains two 30-item questionnaires designed to assess the constructs of sociotropy and autonomy. Participants rate the degree to which hypothetical statements apply to them on 5-point scales. C. J. Robins (1985) reported alpha coefficients of .90 for sociotropy and .80 for autonomy and test-retest

⁵ Our diagnostic experts were Jean Endicott's group at the New York State Psychiatric Institute and Alan Gruenberg, professor of psychiatry at Jefferson University School of Medicine.

correlations over 4–6 weeks of .75 for sociotropy and .69 for autonomy. In the present study, we used the total Sociotropy and Autonomy scale scores in our exploratory analyses (see Table 2 for correlations between sociotropy and autonomy).

Self-Consciousness Scale. The SCS (Fenigstein et al., 1975) is a 23-item self-report questionnaire that measures the dispositional tendency to engage in self-focused attention. We used the Private subscale (10 items rated on 4-point scales), assessing the tendency to attend to covert, internal aspects of the self (e.g., one's thoughts, feelings, and attitudes), in our exploratory analyses. The reliability and validity of the SCS have been well documented (e.g., Carver & Glass, 1976; Fenigstein et al., 1975).

Stress-Reactive Rumination Scale. The SRRS (Robinson, 1997) was adapted from the Response Styles Questionnaire (RSQ; Nolen-Hoeksema et al., 1993). The original RSQ was designed to measure the degree to which people ruminate or distract themselves from depressive symptoms and has shown adequate reliability and validity (Just & Alloy, 1997; Nolen-Hoeksema & Morrow, 1991; Nolen-Hoeksema et al., 1993, 1994). The SRRS consists of 25 items and contains a 9-item subscale, used in the present analyses, that assesses individuals' degree of rumination about negative inferences in response to stressors. The participant is asked to rate how often he or she thinks or does each of the items (e.g., "Think about what the occurrence of the stressor means about you") after experiencing a major negative life event on a 0–100 scale. Internal consistency ($\alpha = .89$) and test–retest reliability of the SRRS subscale over a 1-month interval ($r = .71$) were good.

Beck Depression Inventory. 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).

Results

Overview of Data Analysis Approach

Our data-analytic strategy involved four parts. The main hypotheses were that the HR group would exhibit a higher lifetime prevalence of depressive disorders, including HD, than would the LR group (Hypothesis 1), but the groups would not differ on lifetime prevalence of other disorders (Hypothesis 2). First, we tested Hypotheses 1 and 2 by examining whether the critical risk group differences in lifetime prevalence were significant. Because of unequal cell sizes, we used hierarchical regression analyses to accomplish what is sometimes called hierarchical analysis of variance. Specifically, we conducted Cognitive Risk (high vs. low) \times Sex (male vs. female) \times Site (TU vs. UW) hierarchical analyses of covariance (ANCOVAs), with participants' age, screening BDI scores, and SADS-L current depressive symptom scores as covariates,⁶ on the occurrence (yes or no) of at least one past episode of each disorder. The use of the BDI and SADS-L current depressive symptom scores as covariates was designed to control for residual differences in current depressive symptom levels between the HR and LR groups that might account for any obtained risk group effects in lifetime prevalence of depressive or other disorders. We do not report main effects or interactions that were not theoretically relevant or did not compromise the interpretation of predicted results (e.g., a Sex \times Site interaction). We do, however, report any interactions between a predicted effect and site (e.g., a Cognitive Risk \times Site interaction) that would indicate that the predicted result may not have generalized across both sites. Our tests of the hypotheses were very conservative (see the Discussion section)

in that we used two current depression covariates and two-tailed tests of significance, even though we had clear-cut directional predictions.

Second, as part of our exploratory questions, we conducted analyses with the same design on the severity, duration, and age of onset of lifetime episodic depressive disorders among those participants who had experienced at least one episode of past depression. Degrees of freedom differed slightly across analyses as a result of missing data on some measures. In addition, degrees of freedom were smaller for all analyses involving clinical parameters (e.g., duration) of depressive episodes because these analyses were conducted only with participants who had experienced at least one episode of the disorder.

Parts 3 and 4 of our analytic strategy involved exploratory analyses of the additional cognitive styles. We reconducted our Risk \times Sex \times Site hierarchical ANCOVAs with each of the other cognitive styles (inferential style for positive events, sociotropy, autonomy, private self-consciousness, and stress-reactive rumination) as covariates to determine whether any obtained risk group differences in lifetime prevalence of depressive disorders remained significant with these other cognitive styles controlled. Finally, we added each of these other cognitive styles as main effects and as two-way interactions with the other variables in the hierarchical ANCOVAs to explore whether any of these other styles predicted additional variance in lifetime prevalence of depressive disorders as a main effect or in interaction with cognitive risk. In hierarchical ANCOVAs involving more than three variables, we did not test for four-way or higher interactions owing to missing cell problems and consequent uninterpretability of results.

Hypothesis 1: Lifetime Prevalence of Depressive Disorders

On the basis of the cognitive vulnerability hypotheses of hopelessness (Abramson et al., 1989) and Beck's (1967) theories of depression, we predicted that cognitively high-risk participants would exhibit greater lifetime prevalence of depressive disorders, including the subtype of HD, than would cognitively low-risk participants. Table 3 shows F and R^2 change values and odds ratios for all risk group differences. As shown in Table 3, consistent with this hypothesis, the Cognitive Risk \times Sex \times Site hierarchical ANCOVAs indicated that, after age and the two measures of current depressive symptoms had been controlled, participants in the HR group had significantly greater lifetime prevalence rates than participants in the LR group of the episodic depressive disorders, major depressive disorder (*DSM-III-R* and RDC combined; 38.7% vs. 17.0%)⁷ and HD (39.9% vs. 11.9%). They also

⁶ In most cases, the three covariates met the assumption of homogeneity of regression. When one or more of the covariates violated this assumption, we included it as a full predictor (including all of its main effects and interactions) in the model.

⁷ We present analyses for *DSM-III-R* and RDC major depression combined for the purpose of ease and brevity of presentation. However, the cognitive risk effect was significant for major depression in each diagnostic system separately, $F(1, 338) = 9.48, p < .01$, for *DSM-III-R* major depressive disorder (38.2% HR group vs. 16.5% LR group) and $F(1, 338) = 9.84, p < .01$, for RDC major depressive disorder (36.4% HR group vs. 15.3% LR group), after control for the three covariates.

Table 3

Lifetime Prevalence of Depressive and Other Disorders as a Function of Cognitive Risk, Controlling for Age, Phase 2 Screening BDI Scores, and SADS-L Current Depressive Symptom Scores

| Disorder | Low risk % (n = 176) | High risk % (n = 173) | Risk F | ΔR^2 | OR | 95% CI |
|---|-------------------------|--------------------------|-----------|--------------|-------|-------------|
| Major depression (<i>DSM-III-R</i> or RDC) | 17.0 | 38.7 | 9.48*** | .025 | 3.01 | 1.84–4.94 |
| Minor depression (RDC) | 11.9 | 22.0 | 3.03* | .008 | 2.11 | 1.18–3.77 |
| Hopelessness depression (project) | 11.9 | 39.9 | 22.41**** | .059 | 4.87 | 2.81–8.44 |
| Dysthymic disorder (<i>DSM-III-R</i>) | 2.3 | 3.5 | 0.37 | .001 | 1.56 | 0.43–5.64 |
| Intermittent depressive disorder (RDC) | 2.3 | 4.0 | 0.37 | .001 | 1.84 | 0.53–6.39 |
| Depression not otherwise specified (<i>DSM-III-R</i>) | 3.4 | 6.4 | 1.19 | .003 | 1.76 | 0.63–4.95 |
| Labile personality (RDC) | 1.1 | 8.1 | 0.75 | .002 | 7.76 | 1.74–34.67 |
| Subaffective dysthymic disorder (RDC) | 0.0 | 3.5 | 1.55 | .004 | 13.86 | 0.77–248.04 |
| Any anxiety disorder (<i>DSM-III-R</i> or RDC) | 7.4 | 12.1 | 0.07 | .000 | 1.76 | 0.85–3.63 |
| Any substance use disorder (<i>DSM-III-R</i> or RDC) | 8.5 | 8.7 | 0.00 | .000 | 1.03 | 0.49–2.18 |
| Other psychiatric disorder (RDC) | 4.0 | 2.3 | 0.73 | .002 | 0.58 | 0.17–2.01 |

Note. Degrees of freedom were 1,338 for major depression, hopelessness depression, labile personality, subaffective dysthymic disorder, any anxiety disorder, and other psychiatric disorder. Degrees of freedom were 1,332 for all other disorders. BDI = Beck Depression Inventory; SADS-L = Schedule for Affective Disorders and Schizophrenia–Lifetime interview; OR = odds ratio; CI = confidence interval; *DSM-III-R* = *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.); RDC = research diagnostic criteria.

* $p < .08$. *** $p < .01$. **** $p < .001$.

had marginally greater lifetime prevalence rates of RDC minor depressive disorder (22.0% vs. 11.9%). Indeed, based on the odds ratios (Table 3), the risk of lifetime major depression in the HR group, relative to the LR group, was threefold; the risk of minor depression was twofold; and the risk of HD was almost fivefold.⁸ Moreover, neither of the two current depressive symptom indexes (BDI or SADS-L) themselves predicted lifetime prevalence of major or minor depressive disorder or HD. To examine further whether the cognitive vulnerability hypothesis was supported for clinically significant depressive episodes, defined very strictly, we examined definite major depressive episodes separately from probable episodes. In comparison with the LR group, the HR group had significantly higher prevalences of both definite major depressive disorder (19.1% vs. 7.9%), $F(1, 338) = 4.36, p < .04$, and probable major depressive disorder (26.6% vs. 10.2%), $F(1, 338) = 6.91, p < .01$. However, the risk groups did not differ significantly on lifetime rates of the chronic depressive disorders: *DSM-III-R* dysthymia (3.5% vs. 2.3%), RDC intermittent depressive disorder (4.0% vs. 2.3%), *DSM-III-R* depression not otherwise specified (6.4% vs. 3.4%), RDC labile personality (8.1% vs. 1.1%), and RDC subaffective dysthymia (3.5% vs. 0%). There were no sex differences or Risk \times Site interactions on lifetime prevalence of any depressive disorder. The latter finding indicates that the risk group differences in episodic depressive disorders and HD generalized across the sites.

Hypothesis 2: Lifetime Prevalence of Other Disorders

As can be seen in Table 3, there were no risk group differences in lifetime prevalences of anxiety disorders (*DSM-III-R* or RDC generalized anxiety disorder, panic disorder, simple and social phobias, obsessive–compulsive disorder, or posttraumatic stress disorder combined; 12.1% vs. 7.4%), substance use disorders (*DSM-III-R* or RDC alcohol abuse, dependence, or drug use disorder combined; 8.7% vs. 8.5%), or other disorders (RDC; 2.3% vs. 4.0%).

Exploratory Analyses: Clinical Parameters of Depressive Disorders

Next, we conducted Risk \times Sex \times Site hierarchical ANCOVAs, with age, screening BDI score, and SADS-L current depressive symptom scores as the covariates (see Footnote 6), on the severity (number of criterial and total symptoms), duration, and age at onset of major or minor depressive disorder and HD (see Table 4 for F statistics, R^2 change values, and means for the risk effects). As can be seen in Table 4, the HR group experienced more total symptoms of major depressive disorder and more criterial and total symptoms of minor depressive disorder than the LR group. Also, the LR group had longer HD episodes than the HR group, but this was entirely attributable to 3 men in the LR group who were outliers (durations of 132, 60, and 60 weeks, respectively).⁹ There

⁸ To obtain the odds ratios shown in Table 3, we conducted Risk \times Sex \times Site hierarchical logistic regression analyses with the three covariates. These logistic regression analyses yielded the same results as the main analyses presented in the article.

⁹ The risk group differences in the number of total symptoms of major depressive disorder, $F(1, 52) = 8.58, p < .01$; the number of criterial, $F(1, 39) = 9.70, p < .01$, and total symptoms of minor depressive disorder, $F(1, 39) = 10.24, p < .01$; and the duration of HD, $F(1, 72) = 5.44, p < .03$, all continued to be significant when number of past episodes of depression was also controlled in addition to age, screening BDI scores, and SADS-L current depressive symptom scores.

¹⁰ We did obtain a Risk \times Site interaction, $F(1, 86) = 5.39, p < .03$, on the average duration (in weeks) of major depressive episodes; however, this interaction was entirely attributable to 3 participants (with durations of 166, 120, and 88 weeks, respectively) who were outliers and experienced chronic major depressive episodes. We also obtained a Risk \times Site interaction, $F(1, 70) = 11.32, p < .001$, on the age of onset of major depressive disorder; however, this interaction was entirely attributable as well to 3 participants (with onset ages of 9, 9, and 10 years, respectively) who were outliers.

Table 4
Severity, Average Duration, and Age of Onset of Major, Minor, and Hopelessness Depression as a Function of Cognitive Risk

| Disorder | Variable | LR | | HR | | Risk F | df | ΔR^2 |
|--|--------------------|--------|-------|-------|-------|---------|-------|--------------|
| | | M | SD | M | SD | | | |
| Major depression (ns = 67 HR, 30 LR) | Criterion symptoms | 5.34 | 1.41 | 5.77 | 1.30 | 1.17 | 1, 78 | .009 |
| | Total symptoms | 10.83 | 3.43 | 13.08 | 2.62 | 9.67*** | 1, 53 | .083 |
| | Duration (weeks) | 13.57 | 24.45 | 11.89 | 23.91 | 0.72 | 1, 86 | .007 |
| | Onset age (years) | 16.10 | 2.40 | 15.78 | 2.32 | 0.32 | 1, 70 | .002 |
| Minor depression (ns = 38 HR, 21 LR) | Criterion symptoms | 6.67 | 2.54 | 7.83 | 3.05 | 8.86*** | 1, 40 | .133 |
| | Total symptoms | 8.26 | 3.31 | 10.47 | 3.58 | 9.69*** | 1, 40 | .128 |
| | Duration (weeks) | 6.14 | 9.05 | 4.75 | 5.70 | 0.18 | 1, 42 | .003 |
| | Onset age (years) | 15.90 | 3.38 | 15.00 | 3.39 | 0.82 | 1, 42 | .013 |
| Hopelessness depression (ns = 69 HR, 21 LR) | Criterion symptoms | 7.33 | 1.85 | 7.92 | 1.71 | 2.23 | 1, 79 | .022 |
| | Total symptoms | 13.38 | 3.73 | 13.84 | 3.02 | 0.09 | 1, 73 | .001 |
| | Duration (weeks) | 18.88* | 31.86 | 9.40 | 18.65 | 6.56** | 1, 73 | .058 |
| | Onset age (years) | 15.52 | 3.72 | 15.20 | 2.88 | 0.01 | 1, 63 | .000 |

Note. HR = high risk; LR = low risk.

* This mean is elevated owing to 3 LR men who were outliers (durations of 132, 60, and 60 weeks, respectively).

** $p < .05$. *** $p < .01$.

were no risk group differences in the average duration or onset age of major or minor depressive episodes.¹⁰

Exploratory Analyses: Additional Cognitive Styles

Does cognitive risk predict lifetime prevalence of depressive disorders beyond other cognitive styles? Given that cognitive risk status based on dysfunctional attitudes and inferential style for negative events was related to most of the other cognitive styles (inferential style for positive events, sociotropy, autonomy, self-consciousness, and stress-reactive rumination), we examined whether cognitive risk continued to be associated with lifetime prevalence of major depressive disorder and HD significantly when these other cognitive styles were included as covariates. Table 5 shows that cognitive risk continued to be related significantly to major depressive disorder (*DSM-III-R* and RDC combined) and HD when each of these other cognitive styles was controlled.

Do other cognitive styles predict lifetime prevalence of depressive disorders beyond cognitive risk? Finally, we explored whether each of the five other cognitive styles predicted additional variance in lifetime prevalence of major depressive disorder and HD after controlling for the effects of cognitive risk status. Thus, we conducted a series of four-way hierarchical ANCOVAs with cognitive risk, sex, site, and each of the other cognitive styles (separately) as independent variables; age as a covariate; and major depressive disorder (*DSM-III-R* and RDC combined) and HD as dependent variables. Inferential style for positive events and private self-consciousness were not associated with lifetime prevalence of major depressive disorder or HD either as main effects, when cognitive risk was controlled, or as moderators of cognitive risk. Autonomy was related to major depressive disorder (*DSM-III-R* and RDC combined) significantly even after control for the effects of cognitive risk, $F(1, 333) = 4.36, p < .04, R^2$ change = .01, with higher levels of autonomy associated with a greater lifetime prevalence of major depressive disorder. Sociotropy moderated the effect of cognitive risk in relation to lifetime prevalence

of HD, $F(1, 333) = 4.25, p < .04, R^2$ change = .01, over and beyond the main effects of cognitive risk and sociotropy. To illustrate the pattern of the interaction, following Cohen and Cohen (1983), we graphed lifetime prevalence rates for HR and LR group participants who scored 1 standard deviation above and below the mean on sociotropy. Figure 1 shows that members of the HR group who were also high in sociotropy exhibited higher lifetime rates of HD than HR group members who were low in sociotropy or LR group members who were either high or low in sociotropy. In addition, stress-reactive rumination moderated the effect of cognitive risk in relation to lifetime prevalence of major depressive disorder (*DSM-III-R* and RDC combined), $F(1, 159) = 5.69, p < .03, R^2$ change = .02, and HD, $F(1, 158) = 9.88, p < .002, R^2$ change = .04. Figure 2 (left) shows that HR group members who were also high (1 SD above the mean) in stress-reactive rumination exhibited higher lifetime prevalence rates of major depressive disorder than members of the HR group low in stress-reactive rumination (1 SD below the mean) or members of the LR group either high or low in stress-reactive rumination. Figure 2 (right) shows the same interaction pattern for lifetime prevalence of HD.

Discussion

Cognitive Vulnerability and Lifetime Prevalence of Depressive and Other Disorders

Consistent with the cognitive vulnerability hypotheses of hopelessness (Abramson et al., 1989; Alloy et al., 1988) and Beck's (1967, 1987) theories of depression, we found that individuals who exhibited negative inferential styles and dysfunctional attitudes, but were not currently in a depressive episode, had higher lifetime prevalences of major depressive disorder and the hypothesized subtype of HD, along with a marginally higher prevalence of RDC minor depressive disorder, than did nondepressed individuals who did not exhibit these negative cognitive styles. These risk group differences held for definite major depressive episodes as well as probable episodes. Indeed, the lifetime risk of major depressive

Table 5
Lifetime Prevalence of Episodic Depressive Disorders
as a Function of Cognitive Risk With Other
Cognitive Styles as Covariates

| Disorder | Covariate | Cognitive risk F | ΔR^2 |
|--|----------------|--------------------|--------------|
| Major depression (<i>DSM-III-R</i> or RDC) | CSQ Pos Comp | 24.54**** | .066 |
| | SAS Sociotropy | 14.03**** | .038 |
| | SAS Autonomy | 24.27**** | .065 |
| | SCS Private | 18.51**** | .050 |
| | SRRS Neg Inf | 5.20** | .030 |
| Hopelessness depression | CSQ Pos Comp | 31.95**** | .084 |
| | SAS Sociotropy | 13.24**** | .035 |
| | SAS Autonomy | 39.04**** | .103 |
| | SCS Private | 32.82**** | .086 |
| | SRRS Neg Inf | 4.85** | .024 |

Note. Degrees of freedom were 1, 339 for all cognitive risk effects except those with the SRRS Neg Inf as a covariate, for which the degrees of freedom were 1, 153. *DSM-III-R* = Diagnostic and Statistical Manual of Mental Disorders (3rd ed., rev.); RDC = research diagnostic criteria; CSQ Pos Comp = Cognitive Style Questionnaire positive events composite; SAS = Sociotropy Autonomy Scales; SCS = Self-Consciousness Scale; SRRS Neg Inf = Stress-Reactive Rumination Scale Negative Inferences subscale.

** $p < .05$. **** $p < .001$.

disorder was triple, and the lifetime risk of HD was almost quintuple, in the HR group relative to the LR group. As such, these findings replicate and extend the findings of Alloy et al. (1992), in which nondepressed individuals with a depressive attributional style exhibited higher rates of major depressive disorder and HD during the previous 2 years than did nondepressed individuals with a nondepressive attributional style. Thus, taken together with the Alloy et al. (1992) results, the present findings indicate that, as predicted by the cognitive theories of depression, negative cognitive styles may confer risk for full-blown, clinically significant depressive disorders. Although high-risk participants had higher lifetime rates of episodic depressive disorders than low-risk par-

ticipants, also replicating Alloy et al. (1992), the risk groups did not differ in prevalence of the chronic depressive disorders, dysthymia and intermittent depressive disorder. Given that our participants were only 18–19 years old and that dysthymia and intermittent depressive disorder require a minimum duration of 2 years, it is possible that risk group differences in rates of these disorders might emerge as participants grow older. Forthcoming prospective data from the CVD Project will allow us to examine this possibility.

It is important to note that we obtained support for the cognitive vulnerability hypothesis with respect to major depressive disorder and HD despite statistically controlling for current depressive symptoms with both questionnaire self-report (BDI) and interview-based, clinician-rated (SADS-L) measures of current depressive symptoms. Thus, the presence of greater current depressive symptoms is unlikely to be a plausible explanation for high-risk participants' greater lifetime prevalence of major depressive disorder and HD than low-risk participants. Indeed, the two measures of current depressive symptoms did not by themselves predict lifetime prevalence of major or minor depressive disorder or HD. The use of the BDI and SADS-L current depressive symptom index as covariates in these analyses provides a very (probably overly) conservative test of the cognitive vulnerability hypotheses, because any of the variance in depressive diagnoses that is shared between cognitive styles and current depressive symptom scores is allocated to current depressive symptoms, even though the cognitive theories predict that such shared variance should exist (Alloy, Abramson, Ranieri, & Dyller, 1999). Therefore, the magnitudes of the effects associated with cognitive risk may be underestimates of the true effect sizes in nature.

An important feature of the present study relative to many prior studies (see Abramson, Alloy, & Metalsky, 1995, for a review) designed to test the cognitive vulnerability hypotheses is that depression was assessed with a structured diagnostic interview (SADS-L) and the application of standardized diagnostic criteria (*DSM-III-R* and RDC) rather than with self-report questionnaire measures of depressive symptoms or mood (Kendall, Hollon,

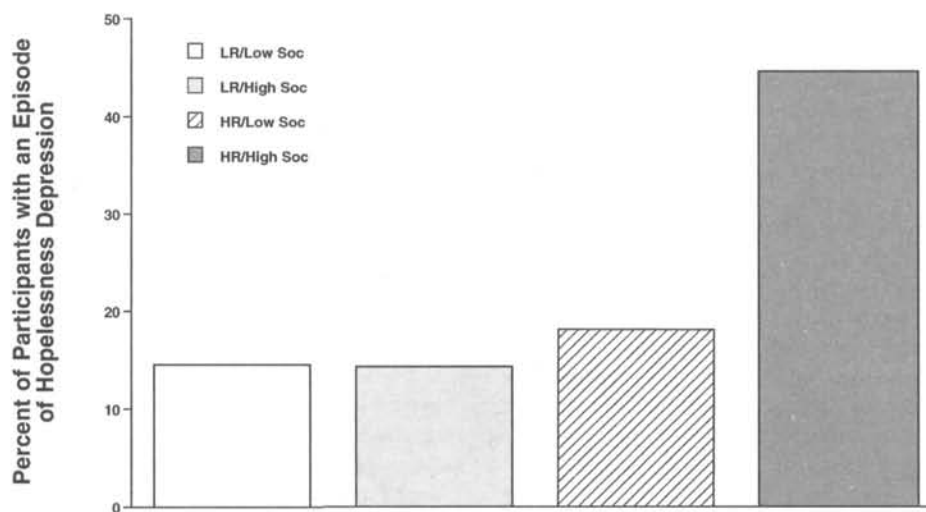


Figure 1. Lifetime prevalence rate of hopelessness depression as a function of cognitive risk group status and Time 1 sociotropy (Soc). LR = low risk; HR = high risk.

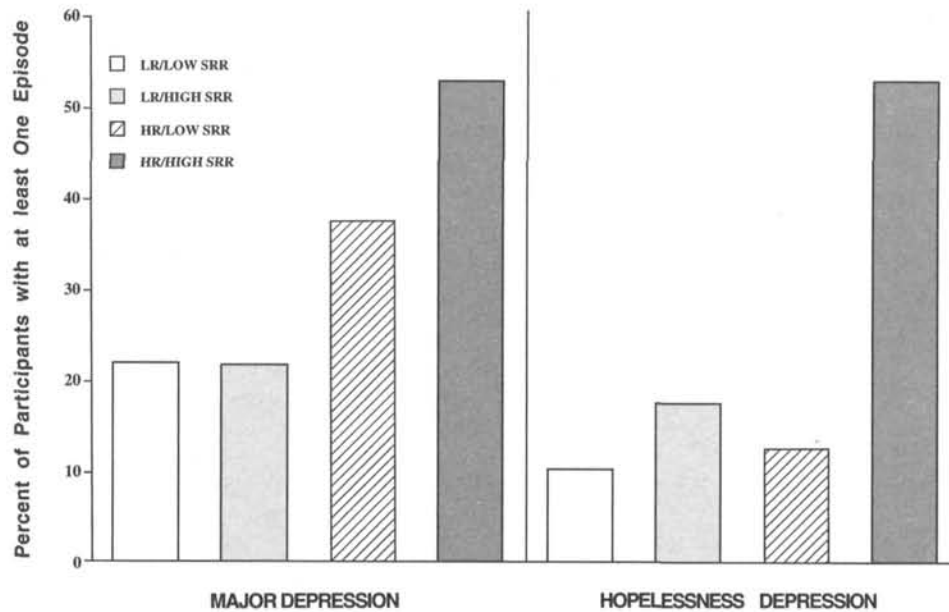


Figure 2. Lifetime prevalence rates of major depressive disorder (left) and hopelessness depression (right) as a function of cognitive risk group status and Time 1 stress-reactive rumination (SRR) at the Temple University site (see Footnote 3). LR = low risk; HR = high risk.

Beck, Hammen, & Ingram, 1987). This is noteworthy because a criticism sometimes leveled at the cognitive theories of depression is that they apply only to mild depressive symptoms (Coyne & Gotlib, 1983). The present findings suggest that this criticism may be unwarranted and that these theories are relevant to explaining more severe, clinically significant forms of depression. It is also of interest that, like Alloy et al. (1992), we obtained similar lifetime rates of comparable depressive disorders (e.g., major depressive disorder) when applying *DSM-III-R* or RDC criteria. Such generality across criterial sets combined with our replication and extension of Alloy et al.'s (1992) findings suggests that the cognitive risk group differences in lifetime prevalence of episodic depressive disorders are robust. Finally, the fact that the risk group differences in lifetime rates of episodic depressive disorders replicated across both sites of our project, despite substantial differences between the sites in ethnic composition and SES, provides further evidence for the robustness and generalizability of our findings.

Whereas the HR group was more likely than the LR group to have experienced episodic depressive disorders, as predicted, the risk groups did not differ in lifetime prevalences of anxiety, substance use, or other psychiatric disorders. This finding suggests that the negative cognitive styles featured as vulnerabilities in the cognitive theories of depression may be associated specifically with increased prevalence of depression, but not other disorders. This specificity is impressive given that anxiety and substance use disorders in particular are frequently comorbid with depression (e.g., Alloy et al., 1990).

The major conceptual limitation of these findings is that, as a result of the retrospective nature of the design, the direction of the association between negative cognitive styles and increased lifetime rates of depressive disorders is unclear. Did the negative cognitive styles exhibited by the HR group temporally precede and

contribute vulnerability to the onset of the lifetime episodes of depression, or did these styles develop as a consequence or "scar" of the past depression (Lewinsohn et al., 1981; Rohde et al., 1990; Zeiss & Lewinsohn, 1988)? An additional ambiguity that actually works against the cognitive vulnerability hypotheses is that LR group members with a history of major depressive disorder or HD may have been high in cognitive vulnerability when those depressive episodes occurred. Those factors that helped LR group members to recover from their past depression (e.g., therapy, medication, and self-help) may also have ameliorated their cognitive vulnerability (Just, Abramson, & Alloy, in press). Given the interpretational ambiguities of the retrospective high-risk design, a more definitive test of the cognitive vulnerability hypotheses requires a prospective design. Preliminary findings from the CVD Project (Abramson, Alloy, Hogan, et al., 1999; Alloy, Abramson, Whitehouse, et al., 1999) indicate that negative cognitive styles do, in fact, predict first onsets and recurrences of major and minor depressive disorder and HD prospectively as well.

Cognitive Vulnerability and Clinical Parameters of Depressive Disorders

Although the hopelessness and Beck theories offer no explicit predictions about the role of inferential styles and dysfunctional attitudes, respectively, in the clinical parameters of depressive episodes, we found that after control for current depressive symptoms, high-risk participants experienced more severe past episodes of major and minor depressive disorder than low-risk participants, again replicating and extending Alloy et al.'s (1992) earlier findings. In contrast, the average duration and age of onset of the two groups' past major and minor depressive episodes did not differ significantly (durations did differ for the HD, but this was attributable to 3 LR group outliers). These findings suggest that negative

cognitive styles may be associated not only with the increased occurrence of depression but with more severe episodes of depression as well. It is intriguing that risk group differences were obtained in the likelihood (prevalence) and severity, but not the duration, of depressive episodes. Several theorists (e.g., Barnett & Gotlib, 1988; Ingram, Miranda, & Segal, 1998) have suggested that different causal factors may be involved in the onset versus maintenance of depression. Perhaps negative cognitive styles contribute vulnerability to the onset and intensity of depressive episodes, but other cognitive or noncognitive (e.g., biological or environmental) processes play a larger role in the maintenance of a depressive episode once it has begun. It will be of interest to examine whether this same distinction between onset and severity versus duration of depressive episodes is produced in future analyses of prospective episodes of depression from the CVD Project.

Other Cognitive Styles as Predictors of Lifetime Prevalence of Depressive Disorders

We also examined the role of five other cognitive styles (inferential style for positive events, sociotropy, autonomy, private self-consciousness, and stress-reactive rumination) as potential mediators or moderators of the cognitive risk effects on lifetime prevalence of major depressive disorder and HD. We found that the HR-LR group differences in lifetime rates of major depressive disorder and HD were maintained when each of these other cognitive styles was controlled, thus supporting the importance of the negative inferential styles and dysfunctional attitudes featured as vulnerabilities in the hopelessness and Beck theories, respectively. That the theoretically predicted relationship between cognitive risk status and past history of depression did not vanish when these other cognitive styles were controlled suggests that these other styles were not mediating the effects of cognitive vulnerability.

Sociotropy and stress-reactive rumination moderated the association between cognitive vulnerability and increased lifetime prevalence of major depressive disorder and HD. We found that HR group members who also tended to ruminate about negative cognitions in response to stressors had higher lifetime rates of both major depressive disorder and HD than HR group members with low rumination and LR group members with either high or low rumination. Robinson (1997) reported that this same Cognitive Risk \times Stress-Reactive Rumination interaction predicted prospective onsets of major depressive disorder and HD episodes. This finding supports the theoretically derived hypothesis (Robinson, 1997; Zullo & Seligman, 1990) that individuals who tend to both make negative inferences after negative life events and then ruminate about them are at especially high risk for depression and indicates that an integration of Nolen-Hoeksema's (1991) response styles theory with the cognitive vulnerability-stress models (Abramson et al., 1989; Beck, 1967) of depression may be very fruitful. Interestingly, private self-consciousness, a construct similar to rumination in some respects (Nolen-Hoeksema, 1991), did not moderate the effects of cognitive risk on lifetime prevalence of major depressive disorder and HD, suggesting that it may be the repetitive, recycling quality of rumination that is crucial to exacerbating the effects of negative cognitive styles.

Sociotropy also moderated the association between cognitive risk and lifetime prevalence of HD. HR group members who were also high in sociotropy, the desire to be intimate with and accepted

by others, had higher rates of HD than HR group members who were low in sociotropy or LR group members who were either high or low in sociotropy. If replicated for prospective onsets of HD, this finding suggests that future elaborations of the hopelessness theory might include greater integration of interpersonal concerns as an additional vulnerability factor for HD.

It should be noted that our findings regarding the associations between the five other cognitive styles and past depression are conditional on the prior presence of a depressotypic inferential style and dysfunctional attitudes. Participants were selected for the CVD Project in such a way as to maximize the potential effects of inferential styles and dysfunctional attitudes. Thus, whereas our study examined the utility of other cognitive styles over and beyond inferential styles and dysfunctional attitudes, it did not pit these five other cognitive styles against CSQ and DAS scores, nor did it illuminate the converse comparison in which the added utility of inferential styles and dysfunctional attitudes is examined in individuals selected on the basis of one or more of these other cognitive styles.

Lifetime Prevalence Rates of Depressive Disorders

Although it may appear that we obtained much higher lifetime prevalence rates for major depressive disorder than did the Epidemiological Catchment Area (ECA; Regier et al., 1988) study of adults, a close examination of the methods used in the two studies suggests that the rates may not be that dissimilar. For example, although our lifetime prevalence of 17% for definite and probable major depressive disorder combined in the LR group was higher than the almost 6% lifetime rate in the ECA study, our rate of 7.9% for definite major depressive disorder in the LR group was not substantially higher than the ECA rate, which also was based on definite diagnoses only. It has been recognized that the ECA rates of depression based on the Diagnostic Interview Schedule (J. N. Robins, Helzer, Crougham, Williams, & Spitzer, 1981) are likely to represent serious underestimations of the lifetime prevalence of depression (Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993; Parker, 1987; Roberts, 1988). However, our rate of definite and probable major depressive disorder combined in the LR group was somewhat high even when compared with the lifetime rate (about 17%) in the more recent National Comorbidity Survey (NCS; Blazer, Kessler, McGonagle, & Swartz, 1994). The somewhat higher prevalence of depression in our study than in these epidemiological samples could indicate either that our rates are too high or that the ECA and NCS rates are too low.

It is possible that the interviewers "overdiagnosed" past depression in the present study as a result of general knowledge that the project was a study of depression. On the other hand, project diagnoses strictly followed *DSM-III-R* and RDC criteria, exhibited high interrater reliability, and were calibrated against those of recognized diagnostic experts (see Footnote 5). In addition, interviewers were unlikely to be biased to overdiagnose depression because they were as likely to interview a low-risk as a high-risk participant and because they were naive to the present retrospective hypotheses. Even if depression was overdiagnosed in the present study, this would not account for the main study finding of significantly higher lifetime prevalence rates of major depressive disorder and HD in the HR group than the LR group because interviewers were unaware of participants' risk group status. Al-

ternatively, following Lewinsohn et al.'s (1993) suggestion, it is possible that our freshman participants reported relatively short-lived episodes that technically met *DSM-III-R* or RDC criteria but would not be recalled if the participants were interviewed later in life. However, the fact that the average duration of diagnosed major depressive disorder and HD episodes in this sample was 3–4 months tends to work against this hypothesis.

A final possibility is that our lifetime rates more accurately reflect the high prevalence of depression in a young, late adolescent sample than do the ECA and NCS rates. Evidence indicates that lifetime prevalence of depression has increased in each successive generation born since World War II (see Klerman, 1988, and Seligman, 1990, for reviews), and the rates of depression in our freshman sample may reflect a continuation of this trend. Indeed, our rates of depression in the LR group are not out of line with those obtained by Lewinsohn et al. (1993), who reported a lifetime rate of about 24% for major depressive disorder at Time 2, when their sample was an average of 17.6 years old (close in age to the present sample). At this point, the data from the present study do not allow us to distinguish with certainty among these three hypotheses. However, we emphasize that any differences in overall rates of depression between our study and other studies do not compromise our critical findings of relative risk group differences in lifetime rates of episodic depression.

Absence of Sex Differences in Lifetime Prevalence of Depressive Disorders

Epidemiological findings indicate a 2:1 ratio of unipolar depression in women versus men from adolescence through adulthood (e.g., Nolen-Hoeksema, 1987). However, an exception to this finding is that many college student samples do not show this usual sex difference in prevalence of depression (Nolen-Hoeksema, 1987, 1990). Consistent with this exception, in the present study of college freshmen, we also did not obtain sex differences in lifetime prevalence of any of the depressive disorders. A second factor that may have contributed to the absence of sex differences in lifetime depression rates in the present sample is that participants were selected on the basis of extreme scores on measures of negative cognitive styles. That is, within the HR and LR groups, men and women had to have extremely negative or positive attitudes and inferential styles, respectively, to be selected for the CVD Project. Given that cognitive style was strongly associated with lifetime prevalence of episodic depressive disorders, and that men and women were equivalent on cognitive styles, there was little chance of sex differences in depression prevalence in this sample.

Conclusion

In conclusion, the present findings provide important new evidence that the dysfunctional attitudes and negative inferential styles of individuals without diagnosable current psychopathology are associated with their lifetime history of clinically significant depressive disorders. As such, they suggest that negative cognitive styles may prove to be a promising vulnerability factor for depression, as hypothesized by the Beck (1967) and hopelessness (Abramson et al., 1989) theories of depression. More definitive conclusions regarding the vulnerability status of negative cognitive styles await the prospective findings of the CVD Project.

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(Appendix follows)

Appendix

Diagnostic Criteria for Depressive Disorders

DSM-III-R MD: (a) Depressed or loss of interest for 2 weeks or more (for definite) or 1 week or more (for subthreshold) for 6 of 7 days of each week; (b) depressed or loss of interest 90% or more (definite) or 75% or more (subthreshold) of each depressed day; and (c) four or more (definite) or three or more (subthreshold) criterial symptoms present, overlapping 6 of 7 days of each week for 2 weeks or more (definite) or 1 week or more (subthreshold).

RDC MD: Same as *DSM-III-R MD* except that one additional criterial symptom is required for definite and probable diagnoses and impairment in functioning is required.

RDC MiD: (a) Depressed 2 weeks or more (definite) or 1 week or more (probable) for 6 of 7 days of each week; (b) depressed 90% or more (definite) or 50% or more (probable) of each depressed day; (c) two or more criterial symptoms present (definite and probable), overlapping 6 of 7 days of each week for 2 weeks or more (definite) or 1 week or more (probable); and (d) impairment in functioning.

DSM-III-R DYS: (a) Depressed 50% or more of waking time for 2 or more years; (b) two or more criterial symptoms present during the depressed days; and (c) no break from depressed mood for 2 or more months.

RDC IDD: Same as *DSM-III-R DYS* with the addition that impairment in functioning is also required. (Note that the 6 criterial symptoms for *DSM-III-R DYS* also are criterial for RDC IDD. However, 10 additional symptoms also are criterial for RDC IDD.)

DSM-III-R DNOS: (a) Depressed 25% or more of waking time for 2 or more years; (b) two or more symptoms from entire list of symptoms that are criterial for MD, MiD, DYS, or IDD; and (c) no break from depressed mood symptoms for 6 or more months.

HD: (a) Hopelessness for 2 weeks or more (definite) or 1 week or more (probable) for 6 of 7 days of each week and (b) five or more (definite) or four or more (probable) criterial symptoms present, overlapping 6 of 7 days of each week for 2 weeks or more (definite) or 1 week or more (probable). The criterial symptoms of HD (Abramson et al., 1989) 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.

Note. *DSM-III-R* = *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.); MD = major depression; RDC = research diagnostic criteria; MiD = minor depression; DYS = dysthymia; IDD = intermittent depressive disorder; DNOS = depression not otherwise specified; HD = hopelessness depression.

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