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Invited Essay

Depressogenic cognitive styles: predictive validity, information processing and personality characteristics, and developmental origins

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Abstract

Two of the major cognitive theories of depression, the theory of Beck [Beck, A. T. (1967). *Depression: clinical, experimental and theoretical aspects*. New York: Harper & Row. and Beck, A. T. (1987) Cognitive models of depression. *Journal of Cognitive Psychotherapy: an International Quarterly*, 1, 5-37] and the hopelessness theory [Abramson, Metalsky, & Alloy, (1989) Hopelessness depression: a theory-based subtype of depression. *Psychological Review*, 96, 358-372], include the hypothesis that particular negative cognitive styles increase individuals' likelihood of developing episodes of depression, in particular, a cognitively mediated subtype of depression, when they encounter negative life events. The Temple-Wisconsin Cognitive Vulnerability to Depression (CVD) project is a two-site, prospective longitudinal study designed to test this cognitive vulnerability hypothesis, as well as the other etiological hypotheses of Beck's and the hopelessness theories of depression. In this article, based on CVD project findings to date, we review evidence that the hypothesized depressogenic cognitive styles do indeed confer vulnerability for clinically significant depressive disorders and suicidality. In addition, we present evidence regarding moderators of these depressogenic cognitive styles, the information processing and personality correlates of these styles and the possible developmental antecedents of these styles. We end with a consideration of future research directions and the clinical implications of cognitive vulnerability to depression. © 1999 Elsevier Science Ltd. All rights reserved.

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1. Cognitive vulnerability hypotheses of depression onset

Individuals vary in their responses to stressful life events. Some may develop severe or long-lasting depression, whereas others don't become depressed at all or only suffer mild, short-lived dysphoria. Investigators have attempted to understand such individual differences in the response to stress in terms of both biological and psychological processes. From the cognitive perspective, the meaning or interpretation people give to their experiences influences whether or not they will become depressed and whether they will be vulnerable to recurrent, severe or long-duration episodes of depression. Two of the major cognitive theories of depression, the hopelessness theory (Alloy, Abramson, Metalsky, & Hartlage, 1988; Abramson et al., 1989) and Beck's theory (Beck, 1967, 1987), are vulnerability–stress theories that attempt to understand individual variability in the response to stress in terms of a set of maladaptive cognitive patterns. According to both theories, particular negative cognitive styles increase individuals' likelihood of developing episodes of depression, in particular, a cognitively mediated subtype of depression (Abramson et al., 1989; Abramson & Alloy, 1990), when they encounter negative life events. According to these cognitive theories, people who possess such depressogenic cognitive styles are vulnerable to depression because they tend to generate interpretations of their experiences that have negative implications for themselves and their futures.

1.1. *Hopelessness theory of depression*

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

1.2. *Beck's theory of depression*

In the theory of Beck (1967, 1987) and Beck, Rush, Shaw, & Emery, 1979), negative self-schemata revolving around themes of inadequacy, failure, loss and worthlessness are hypothesized to provide cognitive vulnerability to depressive symptoms. Such negative self-schemata often are represented as a set of dysfunctional attitudes or self-worth contingencies such as 'If I fail partly, it is as bad as being a complete failure' or 'I am nothing if a person I love doesn't love me'. When they encounter negative life events that impinge on their cognitive vulnerability, individuals exhibiting such dysfunctional attitudes are hypothesized to develop negatively biased construals of the self (low self-esteem), world and future (hopelessness) and,

in turn, depressive symptoms. Thus, although differing in some of their specifics, in both hopelessness and Beck's theories, the hypothesized cognitive vulnerability operates to increase risk for depression through its effects on processing or appraisals of personally relevant life experiences.

2. Testing cognitive vulnerability hypotheses: the behavioral high-risk design

A powerful strategy for testing the cognitive vulnerability hypotheses of hopelessness and Beck's theories is the 'behavioral high-risk design' (e.g. Depue et al., 1981; Alloy, Lipman, & Abramson, 1992). Similar to the genetic high-risk paradigm, the behavioral high-risk design involves studying individuals who do not currently have the disorder of interest but who are hypothesized to be at high or low risk for developing the disorder. However, in the behavioral high-risk design, individuals are selected on the basis of hypothesized psychological, rather than genetic, vulnerability or invulnerability to the disorder. Thus, to test the cognitive vulnerability hypotheses of depression, one would want to select nondepressed individuals who were at high versus low risk for depression based on the presence versus absence of the hypothesized depressogenic cognitive styles. One would then compare these cognitively high and low risk groups on their likelihood of exhibiting depression both in the past, in a retrospective version of the design, and in the future, in a prospective version of the design.

Several reviews of the empirical validity of the cognitive vulnerability hypotheses of depression (e.g. Barnett & Gotlib, 1988; Persons & Miranda, 1992; Segal & Ingram, 1994), based largely on studies using 'remitted depression' designs, generally have concluded that there is little support for these hypotheses (see Alloy, Abramson and Just (1995) and Just, Abramson and Alloy (in press) for the problems with remitted depression designs as tests of the cognitive vulnerability hypotheses). In contrast to these negative conclusions, recent studies using or approximating a behavioral high-risk design have obtained substantial support for the cognitive vulnerability hypotheses of depression. For example, utilizing a retrospective behavioral high-risk design, Alloy et al. (1992) tested the attributional vulnerability hypothesis of the hopelessness theory for clinically significant depression. Alloy et al. compared nondepressed undergraduates who either did or did not exhibit attributional vulnerability for depression accompanied by low self-esteem (an internal, stable, global attributional style for negative events) on their history of major depressive disorder and the hypothesized subtype of HD over the previous two years. Consistent with the hopelessness theory, they found that attributionally vulnerable students were more likely to exhibit past major depressive disorder and HD and experienced more episodes and more severe episodes of these disorders than attributionally invulnerable students. Moreover, in recent studies approximating a prospective behavioral high-risk design, individuals who exhibited negative cognitive styles were more likely to develop depressive moods or symptoms when they experienced negative life events than were individuals who did not exhibit such negative styles (e.g. Nolen-Hoeksema, Gingsus & Seligman, 1986, 1992; Metalsky, Halberstadt, & Abramson, 1987; Metalsky & Joiner,

1992; Metalsky, Joiner, Hardin, & Abramson, 1992; Metalsky et al., 1993; Alloy, Just & Panzarella, 1997b; Alloy & Clements, 1998).

3. The Temple-Wisconsin Cognitive Vulnerability to Depression (CVD) project

The ongoing Temple-Wisconsin Cognitive Vulnerability to Depression (CVD) project is a collaborative, 2-site study that uses a prospective behavioral high-risk design to test the cognitive vulnerability and other etiological hypotheses of hopelessness and Beck's theories of depression for both depressive symptoms and clinically significant depressive episodes. In this article, we review some of the initial findings from the CVD project¹ and several related studies. In the CVD project, university freshmen who were nondepressed and had no other current Axis I psychopathology were followed every 6 weeks for 2 years and then every 4 months for an additional 3 years with self-report and structured interview assessments of stressful life events, cognitions, and symptoms and diagnosable episodes of psychopathology.

3.1. Participant selection and characteristics

Participants were selected for the CVD project based on a two-phase screening procedure. In phase I, 5378 freshmen (2438 at Temple University (TU); 2940 at the University of Wisconsin (UW)) completed the Cognitive Style Questionnaire (CSQ; Abramson, Metalsky, & Alloy, 1998), an expansion of the Attributional Style Questionnaire (Seligman, Abramson, Sanimal, & von Baeyer, 1979) that assesses styles for inferring causes, consequences and characteristics about the self for positive and negative life events and an expanded Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978). The CSQ and DAS assess the cognitive vulnerabilities featured in hopelessness theory and Beck's theory, respectively, and were used to identify freshmen at 'generic' high or low cognitive risk for depression based on both of these theories. Freshmen who scored in the highest quartile (most negative) of the phase I screening sample on both the DAS and the CSQ composite (stability, globality, consequences and self dimensions) for negative events formed a pool of potential high-risk (HR) participants, whereas those who scored in the lowest quartile (most positive) on both instruments formed a pool of potential low-risk (LR) participants.

In phase II, a random subset of freshmen who met the phase I criteria for the HR or LR groups and were <30 years old were administered an expanded Schedule for Affective Disorders and Schizophrenia, Lifetime (SADS-L) interview (Endicott & Spitzer, 1978). The interviews were conducted by extensively trained research assistants who were blind to participants' risk group status and the interviews yielded high interrater reliability (kappas for all diagnoses ≥ 0.90). Based on the SADS-L interview and the application of Diagnostic and statistical manual of mental disorders, 3rd edition revised (DSM-III-R; American Psychiatric Association, 1987) and Research diagnostic criteria

¹ Details of the CVD project findings reviewed here may be found in the individual articles cited.

(RDC; Spitzer, Endicott, & Robins, 1978), participants were excluded from the study if they exhibited any current episodic or chronic mood disorder², any other current Axis I disorder, current psychotic symptoms, a past history of any bipolar spectrum disorder, or any serious medical illness that would preclude participation in a longitudinal study. The final sample included 173 HR (83 at TU; 90 at UW) and 176 LR (87 at TU; 89 at UW) participants. Table 1 displays demographic and cognitive style characteristics of the final project sample. The final sample was representative of the phase I screening samples and the freshmen classes at TU and UW on all demographic variables, with the exception that the final sample had a higher proportion of women than did the phase I sample and the entire freshmen classes (see Alloy and Abramson (1998) for more details about the sample).

An important strength of the CVD project is that the issue of replication of results across the two sites can be addressed. Insofar as the UW sample had a high representation of Caucasian individuals from rural, farming, small town and suburban backgrounds, whereas the TU sample was very urban with a high representation of minority (mostly African-American) and lower socioeconomic status participants (see Table 1), replication of major findings across the two sites would provide strong evidence of generalizability of results. All results from the CVD project reviewed below did, in fact, replicate across both sites.

3.2. *Project assessments*

Within one month of the SADS-L interview, the final sample completed a comprehensive time 1 assessment that included measures of Axis II personality disorders and dimensions (with the Personality Disorder Examination (PDE); Loranger (1988), a semistructured diagnostic interview based on DSM-III-R), self-referent information processing (SRIP Task Battery; Alloy, Abramson, Murray, Whitehouse, & Hogan, 1997a), cognitive styles (CSQ, DAS, sociotropy-autonomy), additional coping styles³ (rumination vs. distraction, self-consciousness), social support and the initial assessments of stress (with a combination questionnaire and semistructured interview patterned after Brown and Harris (1978)) and hypothesized mediating cognitions (inferences for actual events, hopelessness, negative cognitive triad). Participants were then assessed prospectively every 6 weeks for 2 years and, then, every 16 weeks for an

² Participants who met DSM-III-R or RDC criteria for a past mood disorder but who had remitted from this disorder for a minimum of 2 months were retained in the final sample. The minimum 2-month remission from a past depression was designed to insure that any depression onsets during the prospective follow-up were new episodes and not relapses of prior depression. We included participants who were nondepressed currently but had a past depression so as not to be left with an unrepresentative HR group consisting of individuals who, despite possessing very negative cognitive styles, do not readily become depressed (perhaps because they have other protective factors). That is, if the cognitive theories of depression are correct, the 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). Thus, in order not to bias the CVD project against the cognitive theories by possibly excluding the very participants who are at risk for depression according to these theories, we included nondepressed participants who had remitted from a past depression.

³ The coping style and social support measures were administered only to the TU cohort at time 1 and throughout the first 2 years of prospective follow-up; thereafter, these measures were administered at both sites.

Table 1
Final CVD project sample: demographic and cognitive style characteristics

	High-risk	Low-risk
<i>Temple site</i>		
<i>N</i>	83	87
DAS mean item score	4.39 (0.55)	2.17 (0.29)
CSQ-NEG. Comp. mean item score	5.05 (0.47)	2.71 (0.43)
Age (years)	18.45 (1.40)	19.57 (2.98)
Average parental education (years)	13.76 (2.47)	13.45 (2.26)
Combined parental income (US\$)	48,061 (36,013)	39,882 (25,906)
Median parental income (US\$)	35,000	37,650
Sex	67.5% women	66.7% women
Ethnic group	68.3% Caucasian	57.7% Caucasian
<i>Wisconsin site</i>		
<i>N</i>	90	89
DAS mean item score	4.50 (0.44)	2.23 (0.33)
CSQ-NEG Comp. mean item score	5.15 (0.40)	2.78 (0.37)
Age (years)	18.67 (0.37)	18.77 (1.14)
Average parental education (years)	15.20 (2.17)	15.03 (2.27)
Combined parental income (US\$)	82,911 (100,473)	71,782 (53,219)
Median parental income (US\$)	67,500	55,000
Sex	68.9% women	67.4% women
Ethnic group	95.6% Caucasian	92.1% Caucasian

Note. DAS means the Dysfunctional Attitudes Scale, CSQ-NEG. Comp. the Cognitive Style Questionnaire Composite for Negative Outcomes. Standard deviations are in parentheses.

additional 3 years, with a combination of self-report and structured interview procedures on the occurrence of negative life events, inferences for these events, hopelessness and the negative cognitive triad, coping styles and social support and the onset and offset of symptoms and DSM-III-R and RDC episodes of depression, HD and other psychopathology. Table 2 presents the project criteria for diagnosing the hypothesized subtype of HD. The occurrence of life events, cognitions and symptom and episode onsets and offsets during each 6-week or 16-week interval were dated to the day. All assessments were conducted by interviewers who were blind to participants' risk group status.

In addition, at the end of each year of follow-up, participants were reassessed on the cognitive styles featured as vulnerabilities in hopelessness and Beck's theories, the additional coping styles and their self-referent information processing. Participants also completed a second PDE interview for Axis II personality disorders and dimensions at the end of year 5. Finally, at the end of year 2 of follow-up, potential developmental predictors of cognitive vulnerability and depression were assessed. Specifically, participants completed measures of their lifetime history of neglect and abuse experiences and of their parents' feedback styles and parenting behaviors and the participants' parents completed measures of their own cognitive styles (CSQ, DAS), symptoms, parenting, feedback styles and were directly interviewed with

Table 2
CVD project criteria for the diagnosis of hopelessness depression

A	Hopelessness must be present for at least 2 weeks for a definite diagnosis or at least 1 week for a probable diagnosis
B	At least five of the following symptoms must be present for at least 2 weeks, overlapping with each other on at least 12 out of 14 days for a definite diagnosis or at least 4 of the following symptoms must be present for at least 1 week, overlapping with each other on at least 6 out of 7 days for a probable diagnosis criterial symptoms: sadness, retarded initiation of voluntary responses, suicidal ideation/acts, sleep disturbance—initial insomnia, lack of energy, self-blame, difficulty in concentration, psychomotor retardation, brooding/worrying, lowered self-esteem, dependency
C	The onset of hopelessness must precede the onset of the criterial symptoms by at least 1 day and no more than 1 week

the SADS-L and PDE. Further details of the CVD project design, assessments and rationale may be found in Alloy and Abramson (1998).

4. Do depressogenic cognitive styles predict depression and suicidality?

The behavioral high-risk design of the CVD project allowed us to conduct both retrospective and prospective tests of the cognitive vulnerability hypotheses. In this section, we review some of the project's initial findings on the predictive validity of the hypothesized negative cognitive styles for depressive disorders and suicidality.

4.1. Retrospective lifetime history of depression

Based on the cognitive vulnerability hypotheses and evidence that attributional styles exhibit some stability over the lifespan (Burns & Seligman, 1989), Alloy et al. (1998a) examined the lifetime prevalence rates of DSM-III-R and RDC depressive disorders, the subtype of HD, and other Axis I disorders in HR versus LR participants. Consistent with the cognitive vulnerability hypotheses and the earlier findings of Alloy et al. (1992), Alloy et al. (1998a) found that the HR group showed greater lifetime prevalence than the LR group of major depressive disorder (DSM and RDC; 39 vs. 17%), RDC minor depressive disorder (22 vs. 12%), HD (40 vs. 12%) and depressive spectrum disorders (RDC labile personality (8 vs. 1%) and subaffective dysthymia (4 vs. 0%)). Indeed, the HR group showed double the rate of lifetime major depression than the LR group, and triple the rate of HD. Moreover, these HR–LR differences were specific to depressive disorders; there were no statistically significant differences in the rates of anxiety (panic disorder, generalized anxiety disorder, specific and social phobias, obsessive–compulsive disorder, posttraumatic stress disorder), addictive (alcohol and drug abuse), or other Axis I disorders. These findings suggest that depressogenic cognitive styles may indeed confer risk for full-blown, clinically significant depressive disorders and for HD, and that the risk may be specific to depression.

One possible confound in interpreting these lifetime history findings is that the risk group differences may be due to differential residual depressive symptoms associated with HR status (even though no one with current episodes of psychopathology was allowed in the final sample). To rule out this possibility, Alloy et al. (1998a) examined Beck Depression Inventory (BDI; Beck et al., 1979) scores at the time of the cognitive vulnerability assessment as a covariate. With the exceptions of RDC minor depression and the depressive spectrum disorders, the significant risk group differences on lifetime history of major depression and HD were maintained, even with BDI scores controlled. Note that the use of the BDI as a covariate in these analyses provides a very conservative test of the cognitive vulnerability hypotheses because any of the variance in depressive diagnoses that is shared between cognitive risk status and current BDI is allocated to the BDI, even though the cognitive theories predict that such shared variance should exist (Alloy, Abramson, Raniere, & Dyller, in press a).

Alloy et al. (1998a) also examined several other hypothesized cognitive vulnerability factors for depression, including self-consciousness (Ingram, 1990), sociotropy and autonomy (Beck, 1987) and inferential style for positive events (Alloy, Reilly-Harrington, Fresco, Whitehouse, & Zechmeister, in press b). These other variables did not predict lifetime history of depressive disorders (major, minor and HD) and moreover, the HR–LR differences in lifetime prevalence of major, minor and HD remained when these other hypothesized vulnerability factors were used as covariates⁴, thus supporting the importance of the negative inferential styles and dysfunctional attitudes featured as cognitive vulnerabilities in hopelessness and Beck's theories, respectively.

These retrospective lifetime prevalence findings provide the first demonstration that as predicted by the cognitive theories of depression, negative cognitive styles may confer risk for full-blown, clinically significant depressive disorders and for the hypothesized subtype of HD. This is noteworthy because a criticism of the cognitive theories of depression is that they only apply to mild depression. The results of Alloy et al. (1998a) suggest that this criticism is inappropriate and that these theories are relevant to explaining more severe, clinically significant forms of depression. Second, the results also provide support for the hypothesis that the specific subtype of HD exists in nature and conforms to theoretical description. Of course, the major conceptual limitation of the retrospective findings of Alloy et al. (1998a) is that the causal direction of the association between cognitive vulnerability and increased lifetime rates of depressive disorders is unclear. Did the negative cognitive styles temporally precede and contribute to the onset of the past episodes of depression or did these styles develop as a result of the past depression? To more clearly test whether negative cognitive styles actually increase risk for depression, a prospective test of the cognitive vulnerability hypotheses is needed.

4.2. Prospective incidence of first onsets and recurrences of depression

Alloy et al. (1998d) examined CVD project findings on the prospective incidence of first onsets and recurrences of depressive and anxiety disorders for the first 2½ years of follow-up at

⁴ The only exception to this pattern is that when sociotropy was used as a covariate, HR–LR differences in lifetime prevalence of minor depression were no longer significant.

both sites. All information regarding prospective rates of Axis I disorders was obtained from expanded SADS, Change (SADS-C; Spitzer & Endicott, 1978) interviews administered every 6 weeks during this initial follow-up period. More than half of the CVD project sample (75 HR, 113 LR) entered college with no prior history of clinically significant depression. Among these individuals, Alloy et al. (1998d) reported that consistent with the cognitive vulnerability hypotheses, HR participants showed a greater likelihood than LR participants of a first onset of DSM-III-R or RDC major depressive disorder (17 vs. 1%), RDC minor depressive disorder (39 vs. 6%) and the subtype of HD (41 vs. 5%). In contrast, there were no risk group differences in first onsets of anxiety disorders (7 vs. 3%). Moreover, in order to control for any residual depressive symptoms associated with HR status, Alloy et al. (1998d) used initial BDI scores as a covariate. Even with initial BDI scores controlled, the impressive risk group differences in rates of first onsets of depressive episodes were maintained. These findings provide especially important support for the cognitive vulnerability hypotheses because they are based on a truly prospective test, uncontaminated by prior history of depression. Moreover, they suggest that depressogenic cognitive styles confer specific risk for first onsets of depression, and in particular, the subtype of HD, but not for anxiety disorders.

What about those participants who, though nondepressed at the outset of the CVD project, did have a prior history of clinically significant depression? Given that depression is a recurrent disorder (Belsher & Costello, 1988; Judd, 1997), it is important to know whether depressogenic cognitive styles confer vulnerability to recurrences as well as first onsets of depression. Alloy et al. (1998d) reported that HR participants with a past history of depression were more likely than LR participants with prior depression to develop recurrences of DSM-III-R and RDC major depression (27 vs. 6%), RDC minor depression (50 vs. 26.5%) and HD (52 vs. 22%) and these differences were maintained even when initial BDI scores were controlled. HR participants with a prior history of depression were also more likely than previously depressed LR participants to experience onsets of an anxiety disorder (11 vs. 2%). Thus, the cognitive vulnerability hypotheses were upheld for both recurrences as well as first onsets, suggesting similar processes may, at least in part, underlie the first as well as subsequent episodes of depression. The prospective CVD project results are exciting because they provide the first demonstration that negative cognitive styles, or for that matter, any hypothesized psychological vulnerability factor, indeed appear to confer vulnerability to clinically significant depressive episodes.

In addition to cognitive vulnerability, Steinberg, Alloy and Abramson (1998a) examined other predictors of relapse and recurrence of depression during the first 2½ years of prospective follow-up in the CVD project. From a clinical and demographic perspective, Steinberg et al. found that a lower age of onset of the first lifetime episode of depression, a lifetime history of dysthymia, and the severity of the first episode all predicted relapse/recurrence of clinically significant depression, even when cognitive risk status was controlled statistically. Moreover, cognitive risk continued to predict relapse/recurrence of depressive disorders when these three clinical predictors were controlled statistically. It was also of interest that cognitive vulnerability did not worsen as a function of intervening episodes of depression. That is, participants' inferences for stressful events did not increase in negativity from before to after each recurrence of major or minor depression (Steinberg et al., 1998a), supporting the stability of depressogenic cognitive styles.

Finally, in support of Lewinsohn and colleagues' (e.g. Lewinsohn, Steinmetz, Larson, & Franklin, 1981; Rohde, Lewinsohn, & Seeley, 1990, 1994) 'scar hypothesis', Steinberg et al. (1998a) reported that participants who suffered a first depressive episode during the prospective follow-up period experienced decreases in social support from others and interpersonal dependency on others which persisted after recovery from the episode. The decrease in social support from before to after the depressive episode marginally predicted future recurrence of depression, whereas the decrease in interpersonal dependency did not predict further recurrence. Steinberg et al.'s findings suggest that some of the deleterious effects of a depressive episode, particularly the first episode, persist after the episode remits and may contribute to vulnerability to additional episodes in the future.

4.3. Prospective incidence of suicidality

The cognitive theories of depression may also be especially useful for understanding the processes giving rise to suicidality. Drawing on prior research demonstrating a powerful link between hopelessness and suicide (e.g. Minkoff, Bergman, Beck, & Beck, 1973; Kazdin, French, Unis, Esveldt-Dawson, & Sherick, 1983; Petrie & Chamberlain, 1983; Beck, Steer, Kovacs, & Garrison, 1985; Beck, Brown, & Steer, 1989; Beck, Brown, Berchick, Stewart, & Steer, 1990), Abramson et al. (1989) speculated that suicidality, on a continuum from suicidal ideation to completed suicide, may be a core symptom of HD. Indeed, according to both hopelessness and Beck's theories, individuals exhibiting negative inferential styles and dysfunctional attitudes, respectively, should be at risk for suicidality mediated by hopelessness. In a test of this hypothesis, Joiner and Rudd (1995) found that college students with a stable, global attributional style for negative interpersonal events showed increases in suicidality when they experienced interpersonal stressors, consistent with prediction. However, contrary to prediction, hopelessness did not mediate the relation between the depressogenic attributional style and increases in suicidality.

Using CVD project data from the first 2½ years of prospective follow-up, Abramson et al. (1998a) also tested the cognitive vulnerability hypotheses of suicidality, as assessed by both questionnaire self-report and structured diagnostic interviews (SADS-C). The questionnaire assessment of suicidality consisted of a composite of the suicidality items from the BDI (item #9) and the Symptom Check List-90 (Derogatis, 1977; items #15 and 59) for each 2-week period of the follow-up period and the interview assessment consisted of the suicide items from the SADS-C given every 6 weeks during the follow-up.

Abramson et al. (1998a) reported that the HR group was more likely than the LR group to exhibit suicidality as assessed on the SADS-C (28 vs. 12.6%; $p < 0.001$), and they exhibited higher levels of suicidality than LR participants on the questionnaire composite measure (0.053 vs. 0.021; $p < 0.001$). As can be seen in Table 3, HR participants were also more likely than LR participants to exhibit other hypothesized risk factors for suicidality not explicitly specified in hopelessness or Beck's theories, including prior history of suicidality (e.g. Hawton, 1987; Beck, Steer, & Brown, 1993), personal history of depressive disorders (e.g. Hawton, 1987; Lewinsohn, Rohde, & Seeley, 1993), borderline personality dysfunction (Isometsa et al., 1996) and parental history of depression (e.g. Wagner, 1997). Given that cognitive vulnerability was related to these other hypothesized risk factors for suicidality, Abramson et al. (1998a)

Table 3
Cognitive risk group differences on other hypothesized risk factors for suicidality

Risk factor	Low risk	High risk
Prior history of suicidality (%)	7.4	23.8***
Prior history of DSM-III-R and/or RDC major depression (%)	17.1	38.7***
Prior history of RDC minor depression (%)	12.0	22.0*
Borderline personality dysfunction	0.44 (0.88)	0.88 (1.49)**
Antisocial personality dysfunction	1.28 (2.27)	1.40 (2.01)ns
Parental history of depression (%)	24.6	40.5**

Note. For prior history of suicidality, major depression, minor depression and parental history of depression, the numbers in the table represent the percent of participants exhibiting the variable. For borderline and antisocial personality dysfunctions, the numbers in the table represent participants' scores on the personality dimension; standard deviations are in parentheses. * $p < 0.02$; ** $p < 0.002$; *** $p < 0.001$; ns = not significant. (Adapted from Abramson et al., 1998a).

controlled statistically for these other risk factors and found that cognitive risk status continued to predict prospective suicidality. Finally, Abramson et al. (1998a) found that hopelessness as measured by the Hopelessness Scale (Beck, Weissman, Lester, & Trexler, 1974) across the 2½ year follow-up completely mediated the relationship between cognitive vulnerability and prospective suicidality as measured by both questionnaire and interview, even when controlling for prior history of suicidality. It should be noted that controlling for prior history of suicidality in tests of the cognitive vulnerability hypotheses may be overly conservative because prior history of suicidality may, itself, be a result of cognitive vulnerability in part.

4.4. Rumination as a moderator of cognitive vulnerability to depression

According to the response styles theory of depression of Nolen-Hoeksema (1991), individuals who tend to ruminate in response to dysphoria will experience more severe and prolonged depressions, whereas those who tend to distract themselves from their dysphoria will experience milder and shorter depressions. Rumination refers to “behaviors and thoughts that focus one’s attention on one’s depressive symptoms and on the implications of these symptoms” (Nolen-Hoeksema, 1991, p. 569), whereas distraction refers to active attempts to ignore depressive symptoms and instead, to focus on pleasant or neutral activities. Consistent with the response styles theory, several cross-sectional, longitudinal and experimental studies have found that rumination is associated with longer and more severe episodes of depression (e.g. Morrow & Nolen-Hoeksema, 1990; Nolen-Hoeksema & Morrow, 1991; Nolen-Hoeksema, Morrow, & Fredrickson, 1993; Nolen-Hoeksema, Parker, & Larson, 1994; Just & Alloy, 1997). Lyubomirsky and Nolen-Hoeksema (1995) suggested that rumination may prolong and intensify depression because it increases the continued accessibility of negative cognitions and enhances pessimistic thinking.

In an extension of the logic of the response styles theory, Robinson and Alloy (1995, 1998) 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 may be more likely to

develop an episode of depression in the first place. That is, negative cognitive styles provide negative thoughts and inferences but such cognitions will be more likely to be depressogenic when they are 'on a person's mind'. Thus, Robinson and Alloy predicted that stress-reactive rumination would exacerbate the association between negative cognitive styles and onset of depression. To test this prediction, Robinson and Alloy (1995, 1998) used CVD project data to examine lifetime prevalence and prospective incidence of depressive episodes as a function of both cognitive risk status and the tendency to ruminate in response to stressors. Stress-reactive rumination was assessed with a modified version of the Response Styles Questionnaire (Nolen-Hoeksema & Morrow, 1991) developed by Robinson (1997) that was administered at the start of the prospective follow-up phase of the CVD project (at time 1). Consistent with the hypothesis, Robinson and Alloy found that stress-reactive rumination interacted with cognitive risk to predict both lifetime history and prospective onsets of major depressive and HD episodes. While there was no difference in the likelihood of past or future onset of major depression and HD among LR participants with high versus low stress-reactive rumination, among HR participants, the lifetime prevalence and prospective incidence of major depression and HD were significantly greater for those who were also high in stress-reactive rumination than for those who did not tend to ruminate in response to stressors.

4.5. Cognitive vulnerability as a specific risk factor for hopelessness depression (HD)

As indicated by our review of CVD project findings to date, depressogenic cognitive styles have considerable predictive validity for clinically significant episodes of depression, HD and suicidality. But do these maladaptive cognitive styles confer vulnerability to the hypothesized subtype of HD specifically? According to Abramson et al. (1989) (see also Abramson, Alloy, & Hogan, 1997), a negative inferential style may provide risk for HD in particular, but not for other hypothesized subtypes of depression. In a cross-sectional study of out-patients with RDC major depression, endogenous type, Spangler, Simons, Monroe, and Thase (1993) evaluated whether a subset of these patients who had a match between the content domains of their negative attributional style and a recently occurring stressful event exhibited the predicted HD symptom profile (see Table 2) to a greater extent than did those who did not have an attributional style-stressor content match. Spangler et al. (1993) obtained partial support for the hopelessness theory's (Abramson et al., 1989) subtype prediction. Consistent with the theory, depressed patients with an attributional style-stressor match exhibited higher levels of hopelessness and a unique constellation of symptoms as compared to depressed patients without this match. However, the 'matched' group did not show higher levels of all of the six HD symptoms assessed in the study. Whereas the 'matched' group had higher levels of negative cognitions, psychomotor retardation and a trend toward more sadness than the 'unmatched' groups, they did not differ on decreased motivation, suicidality and energy loss. Two difficulties with the interpretation of the Spangler et al. (1993) study, however, are the cross-sectional design and the fact that their patient sample all met criteria for RDC endogenous depression, which may have constrained the type or range of symptoms exhibited by their participants. Indeed, Abramson et al. (1989) speculated that endogenous depression may be fundamentally distinct from the concept of HD inasmuch as the hypothesized core

psychological process in endogenous-type or melancholic depressions is anhedonia rather than hopelessness.

Three other studies that have used or approximated the prospective behavioral high-risk design have obtained more support for the role of a negative inferential style as a specific vulnerability factor for HD. Alloy et al. (1997b) used a short-term, prospective behavioral high-risk design with a daily diary methodology for assessing daily life events and symptoms of an undergraduate sample at high versus low attributional risk for depression. In accord with hopelessness theory, attributionally high-risk participants exhibited higher levels and greater within-day and across-days variability of HD symptoms, but not other depression symptoms. In addition, across-days variability of HD symptoms, but not non-HD symptoms, was further predicted by the interaction of attributional style and daily events, whereas within-day variability of HD symptoms was a function of the main effects of attributional style and daily events. In a prospective design with an unselected undergraduate sample, Alloy and Clements (1998) reported that attributional style for negative events at time 1 interacted with subsequently occurring negative life events to predict time 2 hopelessness and the hypothesized symptoms of HD (after controlling for the time 1 levels of hopelessness and HD symptoms), but did not predict other non-HD symptoms or symptoms of other psychopathology. Similarly, Metalsky and Joiner (1997) also found that the attributional diathesis–stress interaction prospectively predicted HD symptoms, but not depressive symptoms in general, in an unselected undergraduate sample. Thus, there is preliminary support for the specificity of the depressogenic cognitive styles as a vulnerability factor for the subtype of HD, in particular. Future analyses from the CVD project will examine the specificity issue further by testing whether cognitive risk status, despite predicting prospective episodes of HD, fails to predict prospective episodes of DSM-III-R melancholic depression and RDC endogenous depression.

5. Characteristics of cognitively vulnerable individuals

Given that depressogenic cognitive styles predict lifetime history and prospective incidence of depressive disorders and suicidality, it is important to more fully characterize the personalities of cognitively vulnerable individuals. As part of the CVD project, during the time 1 assessment at the beginning of the prospective follow-up phase, we examined the information processing and personality correlates of cognitive vulnerability and invulnerability.

5.1. Cognitive vulnerability and self-referent information processing

According to the cognitive theories of depression, people with negative cognitive styles are vulnerable to depression in part, because they tend to engage in negatively toned information processing about themselves when they encounter stressful events. In the theory of Beck (1967, 1987), for example, depression-prone people are hypothesized to possess negative self-schemata that bias their perception, interpretation and memory of personally-relevant experiences. In the hopelessness theory (Abramson et al., 1989), individuals with depressogenic cognitive styles are hypothesized to draw negative inferences about their self-characteristics in response to stressful circumstances. Thus, Alloy et al. (1997a) examined whether nondepressed individuals with

negative cognitive styles do, in fact, process information about themselves more negatively than do those with positive styles, based on a Self-Referent Information Processing (SRIP) Task Battery, administered to the HR and LR participants in the CVD project at time 1. Alloy et al. assessed self-referent processing both in depression-relevant and depression-irrelevant content domains because Beck's theory suggests that depression-prone individuals have specific negative self-schemata related to incompetence, worthlessness and low motivation, but do not have negative schemata in all content domains (Dykman, Abramson, Alloy, & Hartlage, 1989; Greenberg & Alloy, 1989; McClain & Abramson, 1995). Thus, Beck's content specificity hypothesis would suggest that information processing biases should be limited to stimulus material congruent with the content embodied in the self-schemata.

Table 4
Words judged 'me' and 'not me' and response times for these judgments

	Low risk		High risk	
	mean	S.D.	mean	S.D.
<i>Self-descriptiveness judgments</i>				
<i>Me response</i>				
Positive DR	0.92	0.02 ^{***}	0.78	0.02
Negative DR	0.09	0.01 ^{***}	0.17	0.01
Positive DI	0.86	0.01 ^{**}	0.80	0.01
Negative DI	0.19	0.02 [*]	0.23	0.02
<i>Not me response</i>				
Positive DR	0.08	0.01 ^{***}	0.22	0.01
Negative DR	0.91	0.02 ^{***}	0.83	0.02
Positive DI	0.14	0.01 ^{**}	0.20	0.01
Negative DI	0.81	0.02 [*]	0.77	0.02
<i>Response times for judgments</i>				
<i>Me response</i>				
Positive DR	1902.45	121.55 ^{***}	2581.40	123.47
Negative DR	4261.83	249.34 ⁺	3633.00	253.29
Positive DI	2250.89	178.67	2536.16	181.50
Negative DI	3563.76	267.69	3841.50	271.93
<i>Not me response</i>				
Positive DR	4574.49	253.94 ⁺	3957.64	257.96
Negative DR	2205.97	108.69 ^{**}	2656.48	110.41
Positive DI	3323.84	141.52	3881.27	143.76
Negative DI	2492.17	98.29	2898.43	99.85

Note. DR means depression-relevant, DI depression-irrelevant. Response times are given in ms. Proportion of 'not me' judgments are the inverse of proportion of 'me' judgments. (Adapted from Alloy et al., 1997a). ⁺ $p < 0.10$; ^{*} $p < 0.05$; ^{**} $p < 0.01$; ^{***} $p < 0.001$ for HR-LR differences.

The depression-relevant domains used in the SRIP Task Battery were selected on the basis of clinical descriptions of the depressive self-concept, whereas the irrelevant domains were never mentioned in clinical descriptions of the depressive self-concept (see Alloy et al., 1997a). In addition, Greenberg and Alloy (1989) showed that the relevant domains were empirically correlated with depressive symptoms, whereas the irrelevant domains were uncorrelated with depression. Adjectives were generated that corresponded to the positive and negative endpoints of the depression-relevant and irrelevant domains. The final pool of adjectives contained those that were rated by a separate sample as most highly related to one domain and least highly related to the other domains. The positive adjectives in the depression-relevant and irrelevant domains were equated for likableness as were the negative adjectives in the relevant and irrelevant domains. Finally, the adjectives in each of the four content by valence sets were equated on word length and word frequency.

The SRIP Task Battery was based on earlier work on self-referent processing in depressed versus nondepressed persons (see Alloy et al., 1997a for a review) and consisted of the following four tasks that yielded five dependent measures. First, the HR and LR participants

Table 5
Behavior descriptions and behavior predictions

	Low risk		High risk	
	mean	S.D.	mean	S.D.
<i>Behavior descriptions</i>				
<i>Behavior examples</i>				
Positive DR	2.39	0.09 ⁺	2.14	0.10
Negative DR	0.56	0.04 ⁺	0.67	0.04
Positive DI	1.63	0.07	1.78	0.08
Negative DI	0.15	0.04	0.26	0.04
<i>Words judged self-descriptive</i>				
Positive DR	2.80	0.46 ^{***}	2.44	0.86
Negative DR	0.73	0.55 [*]	0.87	0.69
Positive DI	2.47	0.72 [*]	2.30	0.79
Negative DI	0.22	0.58 ^{**}	0.45	0.77
<i>Behavior predictions</i>				
Positive DR	78.21	0.97 ^{***}	71.66	1.00
Negative DR	20.44	1.09 ^{***}	38.12	1.13
Positive DI	79.60	1.14 ^{**}	75.10	1.18
Negative DI	23.45	1.55 ^{***}	33.56	1.61

Note. DR means depression-relevant, DI depression-irrelevant. For the behavior descriptions task—behavior examples, the numbers in the table represent the number of behavioral examples provided per self-descriptive word for each type of content. For the behavior descriptions task—words judged self-descriptive, the numbers in the table represent the number of words out of three possible for each type of content that were judged as self-descriptive. For behavior predictions, the numbers in the table represent the predicted probability (0–100%) of future behavior for each type of content. ⁺ $p < 0.06$; ^{*} $p < 0.05$; ^{**} $p < 0.01$; ^{***} $p < 0.001$ for HR-LR differences. (Adapted from Alloy et al., 1997a).

were presented with the adjectives in random order on a computer monitor and were asked to judge the self-descriptiveness of each adjective by pressing a ‘me’ or ‘not me’ button on the keyboard. Response times for participants’ decisions were also measured without their awareness. In task 2, participants received a booklet containing adjectives of each of the four content by valence types. For each adjective they judged to be self-descriptive, they were asked to provide specific evidence of their past behaviors that indicated why the adjective described them. For example, if a participant believed she was incompetent, she had to provide specific examples of past incompetent behaviors in her life. In task 3, participants read statements describing hypothetical behaviors in each of the four types of domains. They were asked to judge on a 0–100% scale the probability that they would behave or react in the way described if they were in the situation in the future. In task 4, after a 2-h delay, participants received an incidental free recall test for the adjectives they earlier had judged ‘me’ or ‘not me’.

Consistent with the cognitive theories of depression, Alloy et al. (1997a) hypothesized that relative to LR participants, HR participants would show greater endorsement, faster endorsement, more behavioral descriptions, higher behavioral predictions and greater recall of negative adjectives, whereas LR participants would show preferential processing of positive adjectives. Moreover, based on the content specificity hypothesis of Beck (1967), Alloy et al. predicted that these risk group differences would be more pronounced for the depression-relevant than irrelevant content domains. Thus, Alloy et al. predicted a risk \times content \times valence three-way interaction for each of the self-referent processing measures.

As predicted, Alloy et al. (1997a) found that the risk \times content \times valence interaction was significant for each of the five dependent measures of the SRIP Task Battery. Tables 4–6 display the findings for each dependent measure. As can be seen in Table 4, HR participants

Table 6
Correct recall

	Low risk		High risk	
	mean	S.D.	mean	S.D.
<i>Proportion of words recalled</i>				
<i>Me response</i>				
Positive DR	0.30	0.01**	0.26	0.01
Negative DR	0.05	0.01	0.06	0.01
Positive DI	0.28	0.01	0.29	0.01
Negative DI	0.05	0.01	0.07	0.01
<i>Not me response</i>				
Positive DR	0.02	0.01***	0.06	0.01
Negative DR	0.17	0.01	0.17	0.01
Positive DI	0.04	0.01	0.04	0.01
Negative DI	0.14	0.01	0.14	0.01

Note. DR means depression-relevant, DI depression-irrelevant. Analyses were performed only on the proportion of words recalled because the number of words recalled for each type of content is confounded by different numbers of words seen in the relevant vs. irrelevant categories. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$ for HR-LR differences. (Adapted from Alloy et al., 1997a).

said 'me' to fewer positive and more negative adjectives than LR participants, and these risk group differences were larger for depression-relevant than irrelevant content. HR participants were also slower in responding 'me' to positive depression-relevant content and showed a trend to be faster in saying 'me' to negative depression-relevant content, with smaller group differences for irrelevant content (see Table 4). Relative to the LR group, the HR group provided fewer behavioral examples for positive depression-relevant content and more examples for negative depression-relevant content and they predicted they would be less likely to behave in positive depression-relevant ways and more likely to behave in negative depression-relevant ways and this difference was smaller for the irrelevant content (see Table 5). Finally, HR participants correctly recalled fewer positive depression-relevant adjectives than LR participants (see Table 6). It is also noteworthy that the risk group differences in self-referent processing remained, even when current levels of depressive symptoms were controlled and that participants' depressive symptom levels did not interact with the content and valence of the stimuli in the same manner as did their cognitive styles. These findings suggest that the self-referent processing differences associated with cognitive vulnerability status are not attributable to any residual differences in the risk groups' depressive symptoms.

To summarize, relative to individuals at low cognitive risk for depression, those at high cognitive risk showed preferential self-referent processing of negative depression-relevant material as evidenced by relatively greater endorsement, faster processing, greater accessibility and better recall of content involving themes of incompetence, worthlessness and low motivation. Even more consistent across the SRIP measures, HR participants were also less likely to process positive depression-relevant stimuli than were LR participants. These findings are significant for two reasons. First, they indicate that negatively toned self-referent processing, previously demonstrated to be characteristic of depressed individuals (e.g. Segal, 1988; Greenberg & Beck, 1989), also occurs among nondepressed individuals who are vulnerable to depression by virtue of possessing negative cognitive styles. The relatively negative information processing biases observed in HR participants serve to elaborate more fully the concept of negative cognitive styles. Second, from a methodological perspective, the findings are important because they provide converging evidence for information processing effects of cognitive styles on laboratory tasks adapted from cognitive science paradigms (e.g. Segal, 1988; McCabe & Gotlib, 1993), and thereby, further support the construct validity of the cognitive style questionnaire measures.

In order to determine whether the negative self-referent processing shown by HR participants mediated their greater vulnerability to onsets of depressive episodes, we created a composite of the five dependent measures from the SRIP Task Battery and tested this composite as a mediator of cognitive risk effects for prospective onsets of depressive episodes of Alloy et al. (1998d). The negative self-referent processing composite did not mediate the cognitive risk effects; however, it did moderate the risk group differences. We obtained a significant risk \times SRIP interaction for prospective incidence of major, minor and HD episodes. While there was no difference in the likelihood of onset of major, minor and HD episodes among LR participants with high versus low negative self-referent processing, among HR participants, the prospective incidence of depression was significantly greater for those who also showed negative self-referent processing than for those who were low in negative self-referent processing.

5.2. Cognitive vulnerability and personality disorder diagnoses and dimensions

Aside from the tendency to engage in negative information processing about the self, what other personality characteristics correlate with cognitive vulnerability to depression? Abramson et al. (1998b) hypothesized that individuals with depressogenic cognitive styles should be especially likely to exhibit Axis II personality dysfunction. First, the comorbidity between depression and personality disorders is high, ranging from 30 to 70% (Farmer & Nelson-Gray, 1990). Second, among depressed inpatients, those with personality disorders, particularly borderline personality disorder, are more likely to exhibit cognitive vulnerability than other depressives (Rose, Abramson, Hodulik, Halberstadt, & Leff, 1994). Finally, many currently diagnosed personality disorders are associated with the cognitive profiles defining cognitive risk for depression (e.g. Beck et al., 1990), suggesting a link between cognitive vulnerability to depression and personality disorders. For example, cluster C, the ‘anxious/fearful cluster’, is associated with feelings of incompetence, helplessness and weakness. Moreover, many of the personality disorder categories are defined, in part, by the behavioral and interpersonal characteristics that would be likely to cohere with cognitive vulnerability. For example, cognitive vulnerability easily could lead to the avoidance of work and social activities associated with avoidant personality disorders.

Whereas Alloy et al. (1998a,d) found relative specificity of Axis I psychopathology among HR individuals, Abramson et al. (1998b) expected relative nonspecificity of personality dysfunction for three reasons. First, prior research has documented relative nonspecificity of personality disorders in general with many people exhibiting more than one personality disorder (e.g. Farmer & Nelson-Gray, 1990). Second, all three of the personality disorder clusters have been associated with depression, with cluster A, the ‘odd/eccentric’ cluster and cluster C, the ‘anxious/fearful’ cluster, disorders occurring more frequently among depressed outpatients and cluster B, the ‘dramatic/erratic’ cluster, disorders among depressed inpatients (Farmer & Nelson-Gray, 1990). Finally, many different personality disorders are associated with cognitive, behavioral and interpersonal characteristics likely to characterize individuals exhibiting cognitive vulnerability for depression. As discussed above, this is perhaps most obvious for cluster C. However, Abramson et al. (1998b) also expected an association between cognitive vulnerability and cluster B, given the fragile self-esteem and preoccupation with feedback from others of the narcissist, the feelings of helplessness and dependence of the histrionic and the fear of abandonment of the borderline. With respect to cluster A, cognitively HR persons even may show personality dysfunction on the schizotypal and paranoid dimensions given their expected social anxiety and, perhaps, suspiciousness of others.

To assess personality dysfunction, Abramson et al. (1998b) examined the data from the Personality Disorder Examination (PDE), administered at time 1 of the CVD project. The PDE provides for DSM-III-R categorical personality disorder diagnoses as well as dimensional scores, which is important given that participants in the CVD project were young (mean age = 18) and, therefore, relatively unlikely to have developed full-blown diagnosable personality disorder by the time they entered the study. Despite their young age, Abramson et al. reported that the HR group showed a significantly higher rate than the LR group of actual diagnosable personality disorders (5.4 vs. 1.7%). There were not enough participants with actual personality disorders to examine each personality disorder category separately; however,

all three clusters were represented among the personality disorders exhibited by participants. In addition, as expected, the HR group was rated higher than the LR group on personality dimensions from all three clusters, including the paranoid and schizotypal dimensions from cluster A, the borderline, histrionic and narcissistic dimensions from cluster B and the avoidant, dependent, obsessive–compulsive, and passive–aggressive dimensions from cluster C. HR participants were also rated higher on the self-defeating dimension, but the risk groups did not differ on the schizoid, antisocial and sadistic dimensions. With the exception of the narcissistic, passive–aggressive, and self-defeating dimensions, these HR–LR differences in personality dysfunction didn't disappear when participants' depressive symptom levels (BDI scores) were controlled. Abramson et al. (1998b) additionally reported that the risk group differences in lifetime prevalence of episodic unipolar depressive disorders (major, minor and HD) reviewed above were maintained, even when the personality dysfunction associated with HR status was controlled, suggesting that personality dysfunction is not the sole mediator of the effect of cognitive vulnerability on development of depression.

The finding of an association between cognitive vulnerability for depression and personality disturbance is intriguing given the current theoretical controversy about the relationship between mood and personality disorders and the demonstrated comorbidity between the two (Farmer & Nelson-Gray, 1990). This finding points to the importance of interpersonal dysfunction among individuals exhibiting depressogenic cognitive styles. A shortcoming of the cognitive theories of depression is that cognitive vulnerability for depression often has been described as if it occurs in a behavioral and interpersonal vacuum (but see Gotlib and Hammen (1992), Joiner, Alfano, and Metalsky (1992) and Panzarella and Alloy (1998) for recent interpersonal–cognitive integrations). The results of Abramson et al. (1998b) begin to reveal the behavioral and interpersonal styles associated with cognitive risk for depression. In this regard, researchers have just begun to examine the cognitive–behavioral–interpersonal configurations that confer risk for depression (e.g. Gotlib & Hammen, 1992; Joiner et al., 1992; Segrin & Abramson, 1994; Alloy, Fedderly, Kennedy-Moore, & Cohan, 1998).

6. Developmental origins of cognitive vulnerability to depression

If depressogenic cognitive styles do confer vulnerability for depression and suicidality, as indicated by the CVD project findings, then it becomes important to understand the origins of these cognitive styles. What are the developmental antecedents of cognitive vulnerability versus invulnerability to depression? As part of the CVD project, we have directly studied 320 of the parents of the cognitively HR and LR participants with respect to the parents' cognitive styles, parenting behaviors, psychopathology and personality, as well as the HR and LR participants' early childhood life events and neglect and abuse experiences. In this section, we briefly review some of the preliminary findings from the CVD project and several related studies on possible developmental precursors of negative versus positive cognitive styles. It's important to note that the CVD project findings presented in this section are indeed preliminary inasmuch as analyses are still in progress. Moreover, our initial explorations of potential precursors of cognitive styles have relied on retrospective designs and, thus, should be interpreted as generating hypotheses for more definitive testing in future prospective designs.

6.1. *Parental psychopathology and children's cognitive vulnerability to depression*

Prior research has demonstrated that children of depressed parents are at increased risk for depression themselves (e.g. Downey & Coyne, 1990). In particular, parental depression may contribute to the development of depressogenic cognitive styles and, thus, cognitive vulnerability to depression, in their offspring through a variety of mechanisms including genetic transmission, modeling and negative parenting practices, among others. Along these lines, Hammen (1992) reported that children of women with mood disorders evidenced more negative attributional styles and cognitions about themselves. In order to explore the possible familial origins of negative cognitive styles, Abramson, Alloy, Tashman, Whitehouse and Hogan (1998d) examined the association between CVD project participants' cognitive risk status and their parents' depression based on the participants' reports of their parents' psychiatric history using the family history RDC method (Andreason, Endicott, Spitzer, & Winokur, 1977), as well as direct interview of the parents with the SADS-L. Child and parent reports of parental psychopathology showed significant, but modest, agreement, with ϕ -coefficients ranging from 0.18–0.43 for various disorders.

Based on the child reports of parental psychopathology, Abramson et al. (1998d) found that HR participants' mothers were significantly more likely and their fathers showed a trend to be more likely to have a history of depressive disorder than were LR participants' mothers and fathers, respectively (35 vs. 18% for mothers and 18 vs. 12% for fathers). There were no HR–LR differences in the parents' rates of other disorders. The direct interview of parents yielded an interaction between participants' cognitive risk status and parent sex on lifetime parental depression. Mothers of HR participants had greater lifetime histories of depression than mothers of LR participants, whereas fathers of HR and LR participants didn't differ. In sum, both child and parent reports about parents' depression were consistent in showing greater lifetime depression in the mothers of HR than LR individuals. However, the findings for fathers were weak and, thus, risk group differences in fathers' depression need greater study. This study is the first to examine rates of depression in the parents of nondepressed persons who are at high or low cognitive risk for depression. The findings of Abramson et al. (1998d) are consistent with those of Hammen (1992) and with the hypothesis that mothers' depression may contribute to the development of cognitive vulnerability to depression in their offspring. These results provide the background for further exploration of possible mediators of the association between parental depression and children's cognitive risk for depression.

6.2. *Modeling, parental inferential feedback and cognitive vulnerability to depression*

Children may learn their cognitive styles in part from significant others, in particular, their parents. One possible mechanism is that children model the cognitive styles of their parents. If modeling of parents' styles is a contributor to the development of cognitive vulnerability versus invulnerability to depression, then children's cognitive styles should correlate with those of their mothers or fathers. In support of the modeling hypothesis, Seligman et al. (1984) reported that 8–13-year old children's attributional styles for negative events correlated with those of their mothers but not their fathers. However, other studies have failed to replicate parent–child

correlations in attributional styles (Kaslow, Rehm, Pollack, & Siegel, 1988; Oliver & Berger, 1992; Turk & Bry, 1992).

In addition to modeling, the feedback parents provide to their children about causes and consequences of negative events in the child's life may contribute to the child's cognitive risk for depression. Children may be taught, implicitly or explicitly, to make the same inferences about events in their lives as those made by parents for the children's events. If parental feedback contributes to children's cognitive risk, then parents' typical inferential communications to their children should be correlated with the children's cognitive styles. Consistent with the feedback hypothesis, Turk and Bry (1992) reported that fathers' attributions for events in their adolescent child's life were positively correlated with these adolescents' attributional styles. Similarly, Fincham and Cain (1986) found that children who attributed academic failures to external causes had parents who attributed the child's failures to their own (the parents') lack of effort, something external to the child.

Alloy et al. (1998b) examined the modeling and feedback hypotheses with data from the CVD project. To test the modeling hypothesis, the same cognitive style measures (CSQ and DAS) were administered to the parents of the HR and LR participants as the participants completed themselves. To test the feedback hypothesis, parent and child report versions of the Parent Attributions for Children's Events Scale (PACE; Steinberg, Tashman, Alloy, & Abramson, 1998b), a questionnaire that assessed parents' typical feedback to their child regarding the causes and consequences of negative events in the child's life, were administered. Consistent with the modeling hypothesis, Alloy et al. (1998b) found that parents of HR participants had more dysfunctional attitudes than parents of LR participants. Alloy et al. also obtained support for the feedback hypothesis. According to the child reports of their parents' behavior, both mothers and fathers of HR participants provided more depressogenic feedback about causes and consequences of stressful events that happened to their child than did mothers and fathers of LR participants. Thus, not only may children model the cognitive styles of their parents, but they may also be directly taught habitual ways of interpreting events by the inferential feedback they receive from their parents.

6.3. Parenting, developmental maltreatment and cognitive vulnerability to depression

In addition to modeling of parents' cognitive styles and parental inferential feedback, negative parenting practices and a developmental history of maltreatment and neglect may also contribute to individuals' formation of cognitive risk for depression. Recent reviews of the association between parent and child depression (e.g. Chiariello & Orvaschel, 1995; Tashman, 1997) have suggested that parental depression may impact on children's risk for depression by interfering with the parent's capacity to relate to the child. The two aspects of parenting most often implicated in the association between parent-child relations and children's risk for depression are parental overprotection and lack of emotional warmth, a pattern of parenting called 'affectionless control' by Parker (1983). A key question is whether affectionless control increases risk for children's depression by inculcating a negative cognitive style. Consistent with this hypothesis, Jaenicke et al. (1987) found that depressed mothers' critical, threatening and commanding interactional style toward her child predicted the development of a negative cognitive style in the child.

Alloy et al. (1998c) also addressed this hypothesis by examining the parenting practices of the parents of the CVD project participants as reported by both the participants and their parents (parent report version) on the Children's Report of Parental Behavior Inventory (CRPBI; Schaeffer, 1965). The CRPBI yields scores on three dimensions of parenting: acceptance vs. rejection (positive involvement), psychological autonomy vs. psychological control (negative control) and firm control vs. lax control (lax control). With the exception of lax control for fathers, there was significant but modest agreement between parent and child reports for mothers and fathers on all other CRPBI subscales (r 's ranged from 0.23 to 0.39). Parents of HR participants were less accepting of their child than were parents of LR participants (positive involvement dimension), based on both parent and child reports. On the negative control dimension, Alloy et al. (1998c) obtained a risk \times child sex interaction, in which parents of HR sons reported more negative control than parents of LR sons, whereas the parents of HR and LR daughters didn't differ. For the child reports, HR participants of both sexes tended to report more negative control by their parents than did LR participants. Thus, considering the positive involvement and negative control dimensions of parenting in particular, Alloy et al. (1998c) obtained some support for the hypothesis that a parenting style of affectionless control was associated with depressogenic cognitive styles in offspring.

Rose and Abramson (1998) recently argued that a developmental history of maltreatment and neglect may also contribute to the formation of cognitive risk for depression based on several lines of reasoning. Rose and Abramson suggested that the paradigm used in the original learned helplessness experiments, in which animals were exposed to uncontrollable electric shocks (Seligman, 1975), is analogous to a laboratory model of abuse. They also noted that research demonstrating 'depressive realism' (e.g. Alloy & Abramson, 1988) suggests that depressives may not be as irrational as originally portrayed in Beck's cognitive distortion theory of depression (e.g. Beck, 1967). Rose and Abramson suggested that the negative cognitive styles that confer risk for depression might be the internal representations of maltreatment or adverse environments depressives actually experienced, rather than cognitive distortions. This position is consistent with models formulated by several theorists (e.g. Joiner et al., 1992; Gotlib & Beach, 1995; Panzarella & Alloy, 1998) that emphasize the nature of the individual's interpersonal environment in contributing to vulnerability to depression.

Consistent with their formulation, Rose and Abramson (1998) found that adults who exhibited negative cognitive styles reported experiencing emotional, sexual and physical abuse and neglect while growing up. Four findings from Rose and Abramson's study are particularly relevant to the issue of the origins of a depressogenic cognitive style. First, depressed participants reported significantly higher rates of neglect and emotional, physical and sexual abuse during their childhoods than did nondepressed controls. Second, reported severity of childhood abuse predicted lifetime history of clinical depression. Third, participants with childhood onset of clinical depression reported more severe overall abuse than did participants with later-onset depression, who, in turn, reported more severe abuse than never depressed participants. Indeed, in 92% of the cases, the age of first abuse preceded the onset of the first depression, consistent with the hypothesis that developmental maltreatment contributes to risk for depression. Finally, greater severity of childhood maltreatment was associated with a more negative cognitive style (cognitive vulnerability) in adulthood.

Rose and Abramson (1998) hypothesized that emotional abuse may be a particularly virulent contributor to cognitive vulnerability to depression, because, unlike physical or sexual abuse, in emotional abuse, the abuser by definition supplies negative cognitions to the victim. For example, the individual may be told why negative events happen (e.g. “Of course you didn’t get invited to the prom. You’re ugly.”) and internalize these attributions. Anecdotally, Rose and Abramson noted that participants spontaneously voiced the same view themselves while being administered the maltreatment interview. Participants who had experienced multiple forms of abuse made comments such as, “Bruises heal. Unless you end up needing reconstructive surgery, getting beaten isn’t the worst thing. But I couldn’t forget those terrible things my mother said to me. I can’t get the names she called me out of my head”. Consistent with the hypothesis that emotional abuse may be particularly relevant to the development of cognitive vulnerability to depression, using data from the CVD project, Abramson et al. (1998c) found that the cognitively HR participants reported a greater history of emotional abuse than the LR participants, but the risk groups did not differ on reported physical and sexual abuse. Our finding reported above (Alloy et al., 1998b) that HR participants’ parents provided more negative inferential feedback to them than did LR participants’ parents is also supportive of the specific importance of emotional abuse in the maltreatment–cognitive vulnerability to depression association.

These results demonstrate important associations among childhood maltreatment (particularly emotional abuse), depressogenic cognitive styles and lifetime history of depression. The correlational data obtained in the studies of Rose and Abramson (1998) and Abramson et al. (1998c) cannot establish that the association between early maltreatment and risk for depression is causal, nor can they demonstrate that this association is mediated by increased cognitive vulnerability; however, they are consistent with the hypothesis that developmental maltreatment predisposes cognitive vulnerability to depression and provide fodder for future prospective tests of the hypothesis (see Randolph and Dykman (1998) for the finding that one mediator of the link between critical parenting and depression is dysfunctional attitudes).

7. Directions for future research and clinical implications of the CVD project findings

In this article, we have reviewed promising evidence from the CVD project and related studies indicating that the negative inferential styles and dysfunctional attitudes featured in hopelessness and Beck’s theories of depression, alone and in combination with negative self-referent information processing and rumination, confer vulnerability to suicidality and clinically significant depressive disorders, particularly, the subtype of hopelessness depression. We have also presented preliminary findings on the information processing and personality correlates and developmental origins of depressogenic cognitive styles.

Many important theoretical issues remain to be addressed with the CVD project data. Inasmuch as the cognitive theories of depression are vulnerability–stress models, in which depressogenic cognitive styles confer vulnerability to depression when individuals confront negative life events, future analyses of CVD project data must test whether nondepressed HR participants are more likely than nondepressed LR participants to develop depressive episodes only when they experience stressful life events. Given that negative life events were assessed

repeatedly and dated to the day they occurred in the CVD project, prospective tests of the cognitive diathesis–stress hypothesis will be possible. Along these lines, it will also be important to test whether any predictive effect of the cognitive risk \times stress interaction for future depressive episodes is mediated by the occurrence of hopelessness, as required by theory, and whether it is specific to hopelessness depression as opposed to other possible subtypes of depression. Finally, additional environmental and individual difference variables that may serve as protective factors against the development of hopelessness and depression remain to be explored. Much evidence indicates that social support buffers against depression when people experience stressful events (e.g. Cohen & Wills, 1985). Panzarella and Alloy (1998) theorized and provided support for the idea that material, emotional and informational support from others may buffer against depression by providing ‘adaptive inferential feedback’ that promotes benign inferences about the causes, consequences and meaning of stressful events rather than depressogenic ones.

What are the clinical implications of the CVD project findings to date? Insofar as our findings suggest that negative cognitive styles confer risk for depression and suicidality, modifying these vulnerabilities is an important goal for prevention of initial onsets as well as recurrences of depression. Little is known about how to modify depressogenic cognitive styles once they’ve formed; however, recent therapeutic studies (e.g. Hollon, Skelton, & Loosen, 1991; Hollon & Beck, 1994) suggest that cognitive–behavioral therapy (CBT) aimed at ameliorating negative cognition may reduce the risk for relapse of depression and thus, may have promise for preventing initial onsets of depression as well. Modifications of CBT specifically designed to prevent depression in college students are currently being tested (DeRubeis, Seligman, Schulman, Reivich, & Hallon, 1998) with initial success. Cognitively vulnerable individuals who have experienced developmental maltreatment may profit from more focus on the past than is typical in CBT (Rose & Abramson, 1998). Specifically, it may be beneficial to teach such individuals to reinterpret their abusive histories in terms of being raised by parents or others who maltreated them, not because they were inherently bad or defective, but because their caretakers, for whatever reason, did not have the psychological competence to raise a child in a less traumatic way. Given that negative cognitive styles may be especially likely to contribute vulnerability to depression when exacerbated by rumination, the depressogenic capacity of such styles might also be lessened by training individuals in more effective methods for coping with stressful events. Also, because of its acknowledgment of ‘hopelessness-inducing’ environments, the hopelessness theory (Abramson et al., 1989) suggests a greater focus on environmental modifications than is currently practiced in cognitive therapy. That is, prevention efforts might also be directed at lessening the stressfulness of the environments of cognitively vulnerable people.

If confirmed by future prospective studies, our findings on the developmental antecedents of depressogenic cognitive styles suggest that primary prevention efforts could be aimed at building positive cognitive styles in children by educating parents to model and provide feedback about more benign inferences for stressful events, as well as by direct training in generating positive interpretations of stressful events in schools. Shatte, Gillham, Jaycox, Reivich, and Seligman (in press) have developed such a training program for school children that has shown initial signs of success. Finally, parenting classes that teach parents less abusive

ways of raising their children may also aid in the prevention of cognitive vulnerability to depression, as well as depression itself (Rose & Abramson, 1998).

In conclusion, the findings to date from the CVD project provide important new evidence that individuals' habitual manner of construing experiences in their lives, their cognitive styles, influences their vulnerability to clinically significant depression. Given that depression may lead to considerable suffering and impairment, is often a recurrent disorder and can be lethal when accompanied by suicidality, the documentation of a significant cognitive risk factor for depression has considerable potential for the development of interventions that could prevent this serious disorder.

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