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Cognitive Vulnerability-Stress Theories of Depression: Examining Affective Specificity in the Prediction of Depression Versus Anxiety in Three Prospective Studies

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Anxiety and depression overlap extensively at the level of symptoms and disorder. We tested the etiological factors from two cognitive vulnerability-stress models of depression (Hopelessness theory and Beck's theory) for specificity in predicting depression compared with anxiety. Multiple symptom measures of anxiety and depression with good discriminant validity, diagnoses of anxiety and depression, cognitive vulnerability (negative cognitive style and dysfunctional attitudes), and negative events were assessed in three prospective studies: one with a short-term (5-weeks) follow-up, the second with a long-term (2-years) follow-up, and the third with an academic midterm design. Results show that negative events were a general risk factor for anxiety and depression. Cognitive vulnerability for depression interacted with negative events to predict future depression specifically but not anxiety. Comparison of the two theories suggests that their cognitive vulnerability-stress components overlap largely in the prediction of depression. Implications for the co-occurrence of anxiety and depression as well as for the cognitive vulnerability-stress theories of depression are discussed.

KEY WORDS: cognitive vulnerability; stress; depression; anxiety.

INTRODUCTION

Anxiety and depression commonly co-occur. This overlap can be seen at the level of anxious and depressive mood, symptoms, and disorder from samples of children through adults (see Brady & Kendall, 1992; Clark & Watson, 1991; Maser & Cloninger, 1990; Mineka, Watson, & Clark, 1998, for reviews). For example, it is common to observe concurrent correlations of .70 between some commonly used

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measures of anxious and depressive symptoms (Clark & Watson, 1991) and to see approximately half of individuals receiving a clinically significant diagnosis of one categorical disorder (e.g., depression) earn a co-occurring diagnosis of the other type of disorder (e.g., anxiety; Mineka et al., 1998).

The fact that emotional symptoms and disorders overlap leads to difficulties in testing causal models for either depression or anxiety. Given the pattern of high overlap between anxiety and depression, a researcher cannot be certain whether a putative causal factor or mechanism for specific symptoms (e.g., depression) is, in fact, contributing to those particular symptoms unless both anxiety and depression are assessed properly. Few studies in the literature have tested causal models of anxiety or depression while measuring both anxiety and depressive symptoms to ensure that the proposed etiological factors are affectively specific as hypothesized. The primary aim of this study is to test whether cognitive vulnerability for depression interacts with negative life events to predict depression more specifically compared with anxiety.

Cognitive Vulnerability-Stress Models of Depression

The two central cognitive theories to be tested, hopelessness theory (HT; Abramson, Metalsky, & Alloy, 1989) and Beck's cognitive theory (BT; Beck, 1987) have garnered considerable empirical support (see Abramson et al., 2002; Ingram, Miranda, & Segal, 1998, for general reviews). According to the cognitive vulnerability-stress component of HT, a depressogenic cognitive style is hypothesized to interact with negative life events to contribute to increases in depressive symptoms. In HT cognitive vulnerability is conceptualized as a tendency to make negative inferences about the cause (i.e., global and stable attributions), consequences, and meaning for one's self-concept, of a negative life event. Similarly, BT posits a vulnerability-stress component in which dysfunctional attitudes are hypothesized to interact with negative events to contribute to elevations of depressive symptoms. In BT, cognitive vulnerability is conceptualized as depressive self-schemas containing dysfunctional attitudes, such as one's worth derived from being perfect or needing approval from others.

These cognitive models were proposed originally as etiological theories of depression, so they may be relatively specific to depression compared to anxiety. The interaction of cognitive vulnerability with negative events has been proposed to be an etiologically specific risk factor for depression (Hankin & Abramson, 2001). Prospective research has found that cognitive vulnerability interacting with stressors is associated with future increases in depressive symptoms and disorder (e.g., Hankin, Abramson, & Siler, 2001; Joiner, Metalsky, Lew, & Klocek, 1999; Lewinsohn, Joiner, & Rohde, 2001; Metalsky & Joiner, 1992). In contrast, negative events have been hypothesized to contribute nonspecifically to elevated levels of negative affect (i.e., both anxiety and depressive symptoms) more generally (Hankin & Abramson, 2001). Negative events have been found to be broadly associated with both anxiety and depression (e.g., Lewinsohn et al., 2001; Luten, Ralph, & Mineka, 1997; Metalsky & Joiner, 1992, see McMahon, Grant, Compas, Thurm, & Ey, 2003, for a review of stressors and symptom specificity).

These studies clearly show that initial levels of cognitive vulnerability interact with ongoing negative life events to predict depression, but is cognitive vulnerability a specific risk factor for depression? The majority of studies have found that cognitive vulnerability as a main effect risk factor, without negative events, is associated with depression compared with anxiety (see Mineka, Pury, & Luten, 1995, for a review; Alloy et al., 2000; Gladstone, Kaslow, Seeley, & Lewinsohn, 1997; Lewinsohn, Seeley, & Gotlib, 1997; Weiss, Susser, & Catron, 1998), although this is not always found (Haeffel et al., 2003; Luten et al., 1997). Some prospective vulnerability-stress studies have found that HT's cognitive vulnerability-stress component predicts depressive symptoms more specifically than anxious symptoms (Hankin, Abramson, & Angelli, 1999; Metalsky & Joiner, 1992), whereas others have not found such specificity (Luten et al., 1997; Ralph & Mineka, 1998). However, many of these studies are limited by use of symptom measures that are saturated with high levels of negative affect and lack affective specificity and discriminant validity (see Ralph & Mineka, 1998, for an exception). We located no studies that tested BT's cognitive vulnerability-stress component for affective symptom specificity.

Structural models of anxiety and depression (e.g., Barlow, Chorpita, & Turkovsky, 1996; Clark & Watson, 1991; Mineka et al., 1998) emphasize the need to assess affectively specific symptoms to differentiate general negative affect from relatively specific depression and anxiety. Such structural models recognize and explicitly model the natural co-occurrence of anxiety and depression while simultaneously trying to maximize discriminant validity. For example, one influential model, the tripartite model of anxiety and depression (Clark & Watson, 1991), states that the strong covariation between anxiety and depression is due to a shared negative affect factor (also called general distress), whereas the unique aspects of depression can be captured by a low positive affect (anhedonia) factor and the unique aspects of anxiety can be distinguished by an anxious arousal factor. The general distress factor consists of symptoms common to both anxiety and depression, such as difficulty sleeping and poor concentration. The relatively depression specific factor of anhedonia is characterized by symptoms such as loss of interest and lack of enjoyment in pleasurable activities, whereas the relatively specific anxiety factor of anxious arousal features symptoms such as shortness of breath and dizziness. Various factor analytic studies support the notion that depression is characterized by general distress and anhedonia, whereas anxiety is represented by general distress and anxious arousal (e.g., Brown, Chorpita, & Barlow, 1998; Chorpita, Albano, & Barlow, 1998; Clark, Beck, & Stewart, 1990; Joiner, 1996; Nitschke, Heller, Imig, McDonald, & Miller, 2001; Watson, Clark, et al., 1995; Watson, Weber, et al., 1995). Although there is debate over the precise number of factors needed to best represent the covariance of anxiety and depression (see Burns & Eidelson, 1998), the research clearly indicates the importance of separating anhedonic depression and anxious arousal from general distress to differentiate anxiety and depression.

An additional limitation of past research is the lack of studies prospectively testing the specificity of the cognitive vulnerability-stress factor at the level of clinically significant disorder. Lewinsohn et al. (2001) found that cognitive vulnerability interacting with stressors predicted depressive disorder, but not nonmood disorders, among adolescents. However, the specificity for depressive compared with anxious

disorders is not clear in this study because all nondepressive disorders were grouped together, and there were few cases of anxiety disorder to examine separately. Thus, it is unclear whether cognitive vulnerability interacts with negative life events to predict depression versus anxiety, particularly using more precise affective symptom measures.

Finally, few studies have explicitly compared the etiological components from BT and HT (see Abramson et al., 2002). Lewinsohn and colleagues (2001) investigated the cognitive vulnerability-stress component from BT and HT in a sample of adolescents. They found a different pattern for HT and BT in that HT's Attributional style × stress interaction was inversely related to depression and BT's Dysfunctional attitudes × Stress interaction was positively associated with depression. However, as acknowledged by Lewinsohn and colleagues, this study was limited by poor measurement (low reliability) of cognitive vulnerability (see Hankin & Abramson, 2002). Haeffel et al. (2003) directly compared BT's dysfunctional attitudes and HT's negative cognitive style using more reliable measures in a sample of adults, but they did not examine the vulnerability-stress component of the cognitive theories. Thus, there remains a need to compare the vulnerability-stress components from HT and BT using reliable and valid measures of cognitive vulnerability and stressors while assessing affective symptom specificity with more precise measures.

The Current Investigation

In the current investigation we seek to advance knowledge in this area in the following ways. First, we test the affective specificity of the vulnerability-stress components from HT and BT. Most past studies testing vulnerability-stress hypotheses in depression have ignored the well-known association between depression and anxiety; the current series of studies was conducted to examine etiological specificity of the cognitive vulnerability-stress component predicting depression versus anxiety. Second, we report on results from three independent, prospective vulnerability-stress studies. The first two studies use a 2-time point, panel design (e.g., Joiner et al., 1999; Metalsky & Joiner, 1992, 1997) with different follow-up intervals. Most studies have used only short-term follow-ups (typically 5–10 weeks) to predict elevations of depressive symptoms; we use a short-term (5-weeks) and long-term (2-years) follow-up interval in two independent studies. The third study employs an academic midterm design (e.g., Metalsky, Halberstadt, & Abramson, 1987; Metalsky Joiner, Hardin, & Abramson, 1993; Ralph & Mineka, 1998). We used the same measures of cognitive vulnerability and affective symptoms across all three studies so that results can be compared across the different designs and time intervals; this procedure provides a more rigorous test of the affective specificity of the cognitive vulnerability-stress component with multiple replications.

In sum in this investigation, we use various affective measures with improved discriminant validity, based on a structural model of anxiety and depression (tripartite theory of anxiety and depression), to test whether the etiological factors from cognitive vulnerability-stress models of depression predict prospective increases in depression more specifically than anxiety. It is hypothesized that cognitive vulnerability for depression will interact with negative life events to prospectively predict

depression specifically, but not anxiety. In contrast, negative life events are hypothesized to operate as a nonspecific etiological risk factor for anxiety and depression. To test these hypotheses, data from three different prospective studies were examined. We assessed initial levels of cognitive vulnerability along with prospectively measured negative life events, multiple measures of depressive and anxious symptoms, and the occurrence of clinically significant depressive and anxiety disorders.

STUDY 1

Method

Participants and Procedures

Unselected undergraduate students served as participants. Participants completed a packet of questionnaires for the initial assessment (T1). In Study 1, the undergraduate participants completed a follow-up assessment (T2) 5-weeks after T1 as part of a psychology study. A total of 216 (61 male) participants completed the follow-up assessment out of an original 240 who took part in the initial assessment. There were no significant differences on initial measures of cognitive vulnerability, negative life events, or depressive and anxious symptoms between participants who completed the T2 assessments and those who did not.

Measures

*Cognitive Style Questionnaire (*CSQ; Alloy et al., 2000*).* The CSQ assesses the cognitive vulnerability, including negative inferences for cause, consequence, and self, featured in HT. The CSQ consists of 12 hypothetical scenarios (six interpersonal and six achievement) relevant to young adults, each of which presents the participant with a hypothetical negative event and allows the participant to write down one cause for the event. Respondents then rate the degree to which the cause of the hypothetical negative event is stable, and global (negative inferences for causal attributions; 24 items). In addition, they rate the likelihood that further negative consequences will result from the occurrence of the negative event (negative inferences for consequences; 12 items) and the degree to which the occurrence of the event signifies that the person's self is flawed (negative inference for self; 12 items). The CSQ was scored by summing participants' responses for the negative inferences for cause (stable and global attributions), consequence, and self, and then dividing by the total items. This results in average item-scores on the CSQ ranging from 1 to 7 with higher scores indicating a more negative cognitive style. Coefficient alpha was .92 (48 items). CSQ validity is provided by research showing that the CSQ, alone or in interaction with negative events, predicts depressive symptoms and episodes (Alloy et al., 2000; Metalsky & Joiner, 1992). The CSQ was given at T1.

Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978*).* The DAS (Form A) is a 40-item questionnaire designed to measure the cognitive vulnerability featured in BT. Average item-scores on the DAS range from 1 to 7, with higher scores

reflecting more dysfuctional attitudes. The DAS's validity has been supported by studies finding that the DAS, as main effect or in interaction with negative events, predicts depression (e.g., Hamilton & Abramson, 1983; Ilardi & Craighead, 1999; Joiner et al., 1999). Overall coefficient alpha was .89 (40 items). The DAS was given at T1.

*Negative Life Events Questionnaire (*NLEQ; Metalsky & Joiner, 1992*).* The NLEQ includes negative life events typically experienced by college students. It assesses a broad range of life events from school/achievement to interpersonal/romantic difficulties. The NLEQ consists of 67 different negative life events. Scores on the NLEQ are counts of stressors and range from 0 to 67. Higher scores reflect the occurrence of more negative events. The majority of the stressors (approximately 90%) from the NLEQ could be considered as dependent negative events (occur partly as result of participant's behavior or personality), whereas a minority (10%) were independent, fateful events (outside participant's control; see Hammen, 1991). The NLEQ was given at T1 and T2. At Time 2, participants were instructed to indicate which of these 67 events had occurred to them over the 5-week interval between T1 and T2. At T1, the time frame for NLEQ was the 5-weeks preceding the initial assessment. The NLEQ's validity has been demonstrated in past vulnerability-stress studies (Metalsky & Joiner, 1992).

*Beck Depression Inventory (*BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961*).* The BDI assesses levels of depressive symptoms with 21 items that are rated on a scale from 0 to 3 with scores ranging from 0 to 63 and with higher scores reflecting more depressive symptoms. The BDI is a reliable and well-validated measure of depressive symptomatology (see Beck, Steer, & Garbin, 1988), although it does not enable clinical diagnoses of depression. Higher scores indicate greater severity of depression. The BDI was given at T1 and T2. At T2 participants were instructed to rate the BDI items for the entire 5-week interval from T1 to T2. At T1 participants rated the BDI items for the past 5-weeks before the initial assessment. Coefficient alpha for the BDI was .88.

*Mood and Anxiety Symptom Questionnaire (*MASQ; Watson, Weber, et al., 1995*).* This questionnaire contains 90 items to assess the general distress and specific anxiety and depressive symptoms based on the tripartite theory of anxiety and depression (Clark & Watson, 1991). The MASQ subscales, General Distress: Depression (GDDEP), General Distress: Anxiety (GDANX), Anhedonic Depression (DEP), and Anxious Arousal (ANXAR) were used in this study. Examples of GDDEP include "felt sad," DEP include "felt cheerful" (reverse scored), GDANX include "felt afraid," and ANXAR include "felt faint." The MASQ scales were used to provide multiple, theoretically based, measures of emotional distress symptoms to cover the general and specific affective aspects of anxiety and depression. Higher scores on each of the subscales reflect greater levels of depressive or anxious symptomatology. Reliability and validity of the MASQ has been demonstrated in previous studies (e.g., Ralph & Mineka, 1998; Watson, Clark, et al., 1995; Watson, Weber, et al., 1995). The MASQ was given at T1 and T2. The instructions for the MASQ were the same as for the BDI. The participants at T2 were asked to rate the items for the whole 5-weeks covering the interval from T1 to T2. At Time 1, participants responded to the items for the past 5-weeks prior to T1. Coefficient alpha for GDDEP (12 items)

Table I. Descriptive Statistics and Correlations Among Main Measures—Study 1

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1 CSQ														
2 DAS	.45													
3 BDI1	.38	.31												
4 GDANX1	.42	.28	.55											
5 GDDEP1	.47	.42	.62	.68										
6 DEP1	.39	.42	.57	.51	.73									
7 ANXAR1	.32	.27	.55	.73	.57	.47								
8 NLEQ1	.20	.30	.24	.26	.32	.26	.29							
9 BDI2	.33	.35	.53	.45	.45	.44	.45	.28						
10 GDANX2	.30	.21	.27	.57	.39	.27	.46	.21	.60					
11 GDDEP2	.40	.34	.40	.49	.58	.41	.41	.25	.67	.75				
12 DEP2	.40	.44	.41	.46	.60	.66	.40	.34	.58	.44	.68			
13 ANXAR2	.27	.25	.28	.48	.36	.27	.56	.12	.56	.77	.63	.42		
14 NLEQ2	.18	.33	.29	.32	.36	.40	.35	.58	.49	.38	.43	.41	.34	
M	3.90	3.08	14.19	27.08	22.89	53.12	27.05	27.97	12.42	24.59	19.81	53.54	23.81	24.83
SD	.71	.66	8.3	8.91	6.53	13.26	8.51	10.67	9.51	8.41	6.40	13.15	7.83	11.19

Note. $N = 216$ for all variables. CSQ = Cognitive Style Questionnaire; DAS = Dysfunctional Attitudes Scale; BDI = Beck Depression Inventory; GDDEP = general distress—depression; GDANX = general distress—anxiety; DEP = Anhedonic depressive symptoms; ANXAR = anxious arousal; NLEQ = Negative Life Events Questionnaire. All correlations above .16 are significant at $p < .05$ and correlations above .23 are significant at $p < .01$.

was .92, for GDANX (10 items) was .81, for ANXAR was .86 (16 items), and DEP (22 items) was .92.

Results

Preliminary Analyses

Descriptive statistics and correlations for the main variables for Study 1 are presented in Table I. The measures of cognitive vulnerability (CSQ and DAS) were moderately correlated with negative life events as well as with the different depressive and anxious symptom questionnaires. Also, negative events were moderately associated with both depressive and anxious symptoms. As expected, the general distress measures of depressive and anxious symptoms were, for the most part, correlated highly (i.e., above .65), whereas the discriminant correlations of the affectively specific measures from the tripartite model, anhedonic depressive and anxious arousal symptoms, were relatively less correlated (i.e., around .40–.50).

Data Analytic Plan

There are two ways to address the study's main question: Does the cognitive vulnerability-stress component predict depression more specifically than anxiety? First, composite symptoms of anxiety and depression were examined. For the purpose of these analyses, two composite symptom variables were created: a composite depressive symptoms variable (DEPRESS) and a composite anxiety symptoms variable (ANX). To form these variables, we first standardized each of the observed depressive symptom measures (BDI, GDDEP, and DEP) and the observed anxiety

symptom measures (GDANX, and ANXAR), respectively. We then summed the standardized depression measures to create the composite depression variable, and we summed the standardized anxiety measures to create the composite anxiety variable. This procedure creates highly reliable depression and anxiety variables (see Tram & Cole, 2000 for an analogous data reduction procedure). Second, the specific symptom factors from the tripartite model factors (anxious arousal and anhedonic depression) were examined in both Studies 1 and 2. The anxious arousal factor consisted of the ANXAR measure, and the anhedonic depression factor consisted of the DEP measure.

We used setwise hierarchical multiple regression (see Cohen & Cohen, 1983, pp. 402–422; Metalsky & Joiner, 1992) to test hypotheses with the continuous depression and anxiety symptom data. In this analysis, a set of covariates is entered first into the regression equation, followed by the entry of a set (or sets) of independent variables. This procedure is especially useful in predicting residual change scores when the dependent variable is a postscore measure (e.g., T2 depressive symptoms) and the covariate is a prescore measure (T1 depressive symptoms). In the first step of the regression, the T1 symptoms and T1 negative life events were entered to control for overlapping variance with the predictor variables and T2 symptoms. The second step involves entering the main effects of T1 Cognitive vulnerability (CSQ or DAS) and negative events (NLEQ) at T2. The third step enters the T1 cognitive vulnerability $\times$ T2 NLEQ interaction.[5] For the third step, the slope of the lines for all of the significant interactions were tested and found to differ significantly from 0 (Aiken & West, 1991).

Test of the Cognitive Vulnerability-Stress Component for HT and BT: Composite Depressive and Anxious Symptoms

To test the first hypothesis that HT's cognitive style and BT's dysfunctional attitudes, respectively, would interact with negative life events to predict prospective changes in depressive symptoms, a series of hierarchical multiple regressions was conducted controlling for T1 depressive symptoms and T1 stressors.

For the test of HT, as seen in Table II, the main effect of T2 NLEQ, independent of CSQ, predicted T2 composite depressive symptoms. Importantly, the critical test of the vulnerability-stress component for HT, the CSQ $\times$ NLEQ2 interaction, predicted T2 depressive symptoms. For the test of BT, as seen in Table II, the main effect of T2 NLEQ, independent of DAS, predicted T2 composite depressive symptoms. The test of BT's vulnerability-stress component, the DAS $\times$ NLEQ2 interaction, predicted T2 depressive symptoms.

These analyses show that the Cognitive vulnerability $\times$ Stress interaction from HT and BT predicts T2 depressive symptoms. To illustrate these interactions, we

[5]Some investigators (e.g., Monroe & Simons, 1991) have suggested that including stressors of varying severity (e.g., hassles and major negative life events) in the same vulnerability-stress analysis may be misleading. Given this caution, we repeated all of our vulnerability-stress analyses using only hassles interacting with cognitive vulnerability and only major negative life events interacting with cognitive vulnerability. The same pattern of results was obtained as reported in the main analyses in the text with hassles and major negative life events combined together into a count of general negative stressors.

Table II. Cognitive Vulnerability, Negative Life Events, and Interaction Predicting T2 Composite and Triparite Model's Depressive and Anxious Symptoms Controlling for T1 Symptoms and Stressors in Study 1

Predictor	β	*pr* (Partial correlation)	*t*	Step R^2 change
Composite Symptom Analyses				
HT; composite depressive symptoms				
Step 1				.45
T1 DEPRESS covariate	.67	.67	11.63***	
T1 NLEQ covariate	.14	.14	2.28*	
Step 2				.10
CSQ	.16	.18	2.71**	
T2 NLEQ	.34	.42	6.13***	
Step 3				.03
CSQ × T2 NLEQ	.19	.28	3.77***	
	Model $R^2 = .59$, $F(4, 216) = 61.21$, $p < .001$			
HT; composite anxious symptoms				
Step 1				.39
T1 ANX covariate	.61	.59	9.58***	
T1 NLEQ covariate	.05	.05	0.71	
Step 2				.06
CSQ	.06	.07	0.83	
T2 NLEQ	.21	.24	3.12**	
Step 3				.01
CSQ × T2 NLEQ	.10	.12	1.65	
	Model $R^2 = .45$, $F(4, 216) = 26.21$, $p < .001$			
BT; composite depressive symptoms				
Step 1				.45
T1 DEPRESS covariate	.67	.67	11.63***	
T1 NLEQ covariate	.14	.14	2.28*	
Step 2				.10
DAS	.10	.19	1.67	
T2 NLEQ	.32	.42	5.55***	
Step 3				.02
DAS × T2 NLEQ	.13	.18	2.39**	
	Model $R^2 = .57$, $F(4, 216) = 51.77$, $p < .001$			
BT; composite anxious symptoms				
Step 1				.39
T1 ANX covariate	.61	.59	9.58***	
T1 NLEQ covariate	.05	.05	0.71	
Step 2				.05
DAS	.02	.08	0.23	
T2 NLEQ	.21	.24	3.03**	
Step 3	.00			
DAS × T2 NLEQ	.00	.00	0.01	
	Model $R^2 = .45$, $F(4, 216) = 21.31$, $p < .001$			
HT and BT combined composite depressive symptoms				
Step 1				.46
T1 DEPRESS covariate	.67	.67	11.63***	
T1 NLEQ covariate	.14	.14	2.28*	
Step 2				.10
CSQ	.14	.17	2.26*	
DAS	.06	.07	0.89	
T2 NLEQ	.36	.40	5.48***	
Step 3				.03
CSQ × T2 NLEQ	.16	.04	0.48	
DAS × T2 NLEQ	.20	.04	0.56	
	Model $R^2 = .59$, $F(7, 216) = 32.32$, $p < .001$			

Table II. Continued

Predictor	β	*pr* (Partial correlation)	*t*	Step R^2 change
Tripartite Model Analyses				
HT; Anhedonic depression				
Step 1				.46
T1 DEP covariate	.66	.66	11.54***	
T1 NLEQ covariate	.18	.23	2.98**	
Step 2				.04
CSQ	.16	.21	2.73*	
T2 NLEQ	.23	.29	3.89***	
Step 3				.01
CSQ × T2 NLEQ	.21	.15	1.99*	
	Model $R^2 = .51$, $F(4, 216) = 44.7$, $p < .001$			
HT; Anxious arousal				
Step 1				.31
T1 ANXAR covariate	.60	.60	8.76***	
T1 NLEQ covariate	.06	.06	0.98	
Step 2				.03
CSQ	.10	.12	1.50	
T2 NLEQ	.15	.17	2.17*	
Step 3				.01
CSQ × T2 NLEQ	.25	.15	1.92	
	Model $R^2 = .36$, $F(4, 216) = 22.99$, $p < .001$			
BT; Anhedonic depression				
Step 1				.46
T1 DEP covariate	.66	.66	11.31***	
T1 NLEQ covariate	.18	.23	2.98**	
Step 2				.04
DAS	.15	.18	2.32*	
T2 NLEQ	.21	.25	3.37***	
Step 3				.01
DAS × T2 NLEQ	.23	.14	1.78 ($p = .07$)	
	Model $R^2 = .51$, $F(4, 216) = 26.4$, $p < .001$			
BT; Anxious arousal				
Step 1				.31
T1 ANXAR covariate	.56	.56	8.62***	
T1 NLEQ covariate	.06	.06	0.98	
Step 2				.02
DAS	.05	.06	0.76	
T2 NLEQ	.14	.15	2.01*	
Step 3				.007
DAS × T2 NLEQ	.19	.10	1.3	
	Model $R^2 = .33$, $F(4, 216) = 2039$, $p < .001$			
HT and BT combined anhedonic depression				
Step 1				.46
T1 DEP covariate	.66	.66	11.31***	
T1 NLEQ covariate	.18	.23	2.98**	
Step 2				.07
CSQ	.12	.15	1.99*	
DAS	.09	.11	1.45	
T2 NLEQ	.21	.26	3.47***	
Step 3				.01
CSQ × T2 NLEQ	.28	.06	0.79	
DAS × T2 NLEQ	−.09	−.02	−0.23	
	Model $R^2 = .54$, $F(6, 216) = 28.86$, $p < .001$			

Note. CSQ = negative cognitive style; DAS = dysfunctional attitudes scale; DEPRESS = composite measures of depressive symptoms; ANX = composite measure of anxious symptoms; DEP = anhedonic depressive symptoms; ANXAR = anxious arousal symptoms; NLEQ = Negative Life Events Questionnaire.

*$p < .05$. **$p < .01$. ***$p < .001$.

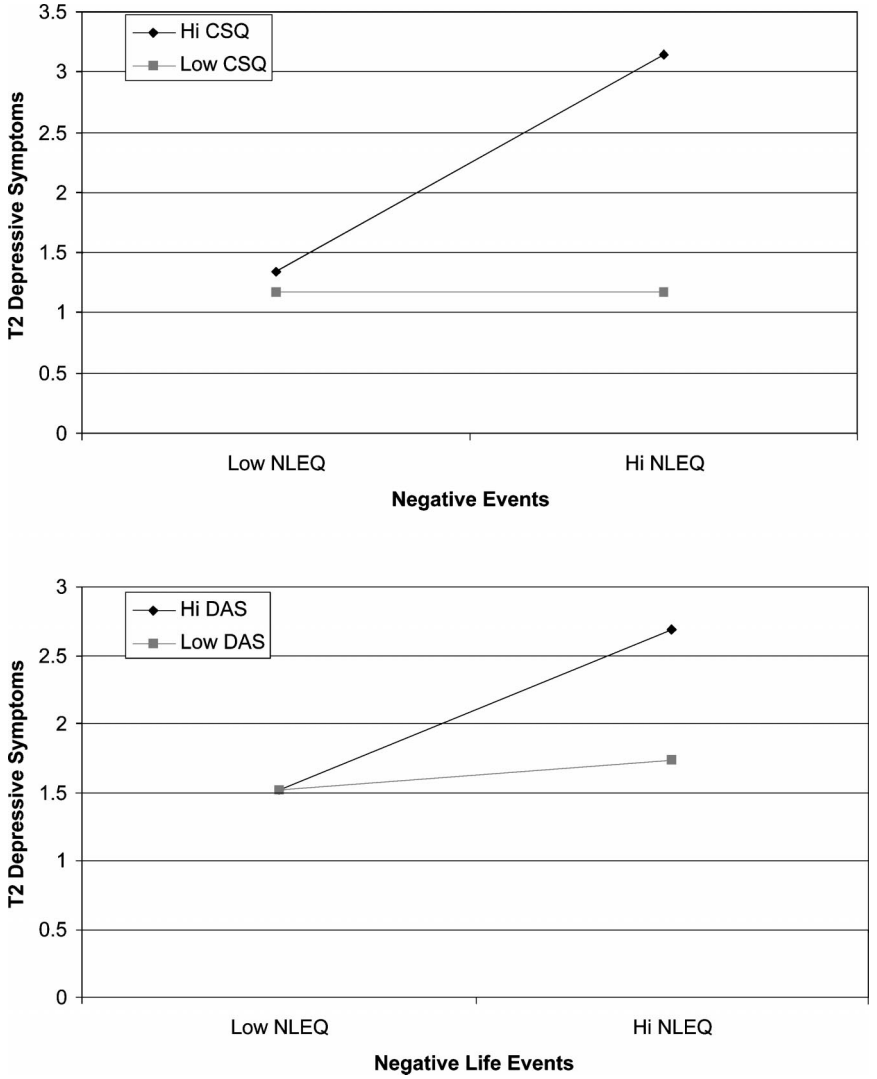

Fig. 1. Regression equation plotting composite depressive symptoms in Study 1 at Time 2, adjusting for Time 1 depression and stressors, as a function of the cognitive vulnerability and negative events interaction for HT (top half) and BT (bottom half). CSQ = HT's negative cognitive style and DAS = BT's dysfunctional attitudes.

graphed the cognitive vulnerability-stress interaction (see Cohen & Cohen, 1983, pp. 323, 419) using specific values (i.e., 1 *SD* above and below the mean) for the predictor variables in the regression equations. Figure 1 shows these results graphed for predicting T2 composite depressive symptoms for HT's and BT's vulnerability-stress component. Higher levels of cognitive vulnerability (from either HT or BT) combined with greater number of stressors was associated with the highest elevations in T2 depressive symptoms over the follow-up.

To test HT's and BT's cognitive vulnerability-stress components predicting T2 composite anxiety symptoms, hierarchical multiple regressions were conducted controlling for T1 anxiety symptoms and T1 NLEQ. These results are shown in Table II. Neither of the main effects of HT's negative cognitive style nor BT's dysfunctional predicted T2 anxiety symptoms. Further, neither cognitive vulnerability-stress interaction predicted T2 anxiety symptoms. However, T2 negative life events predicted T2 anxiety symptoms, even after controlling for initial levels of negative events and anxiety symptoms.

The results above show that the cognitive vulnerability-stress interaction from both HT and BT predicted T2 depressive, but not anxious symptoms. To provide a more stringent test of the affective specificity of the cognitive vulnerability-stress interaction predicting depressive symptoms, we conducted similar hierarchical regression analyses as done above and in Table II, except that initial levels of *both* depression and anxiety composite symptoms were controlled (e.g., see Hankin, Roberts, & Gotlib, 1997; Luten et al., 1997), to predict T2 depressive symptoms. Results showed that BT's Dysfunctional attitudes × Stress interaction ($\beta = .35$, $t = 2.85$, $p < .001$) and HT's Negative cognitive style × Stress interaction ($\beta = .32$, $t = 2.98$, $p < .001$) predicted T2 depressive symptoms.[6] Thus, even when initial levels of anxiety, depression, and stressors are controlled, the Cognitive vulnerability × Stress interaction from HT and BT predicted T2 depressive symptoms.

Test of Vulnerability-Stress Component for HT and BT: Tripartite Model's Specific Depressive and Anxious Symptoms

For the test of HT, as seen in Table II, the main effect of T2 NLEQ, independent of CSQ, predicted T2 anhedonic depressive symptoms. Importantly, the critical test of the vulnerability-stress component for HT, the CSQ × NLEQ2 interaction, predicted T2 anhedonic depressive symptoms. For the test of BT, as seen in Table II, the main effect of T2 NLEQ, independent of DAS, predicted T2 anhedonic depressive symptoms. The test of BT's vulnerability-stress component, the DAS × NLEQ2 interaction, predicted T2 anhedonic depressive symptoms marginally. We graphed the significant HT and BT cognitive vulnerability-stress interactions predicting T2 anhedonic depressive symptoms; the form of these graphs was the same as shown in Fig. 1 for composite depression and is not shown for space considerations.

Table II shows the results of the regression analyses testing HT's and BT's cognitive vulnerability-stress components predicting T2 anxious arousal symptoms. Neither of the main effects of HT's negative cognitive style nor BT's dysfunctional predicted T2 anxious arousal symptoms. Further, neither cognitive vulnerability-stress interaction predicted T2 anxious arousal symptoms. However, T2 negative life events predicted T2 anxious arousal symptoms, even after controlling for initial levels of negative events and anxious arousal symptoms.

[6] An additional stringent test for examining affective symptom specificity is to control for initial levels of depressive symptoms and current levels of anxiety symptoms (i.e., at T2). These analyses also were consistent with HT's and BT's cognitive vulnerability × stress interaction being associated particularly with depressive symptoms. DAS × NLEQ2 predicted T2 depressive symptoms, and CSQ × NLEQ2 predicted T2 depressive symptoms, even after controlling for T1 depressive and T2 anxiety symptoms. Contact the first author for details on these analyses.

Analyses Comparing the Cognitive Theories of Depression

To investigate whether HT's and BT's cognitive vulnerability-stress components largely overlap or distinctly predict the prospective development of depression, regression equations were fit in which T1 depressive symptoms and T1 negative events were entered first, followed by both cognitive factors (CSQ and DAS) and T2 negative life events entered second, and last by both of the Cognitive vulnerability × Stress (CSQ × NLEQ2; DAS × NLEQ2) interactions entered third. These analyses test the hypothesis that one of the cognitive vulnerability-stress components from either HT or BT predicts prospective depression above and beyond controlling for the vulnerability-stress component from the other theory. Table II shows these results for composite depressive symptoms and for anhedonic depression. In general, these analyses suggested considerable overlap of the cognitive vulnerability-stress components in HT and BT. Neither cognitive vulnerability-stress component uniquely predicted T2 composite depressive symptoms or anhedonic depressive symptoms. Across all of these analyses, the magnitude of the effect sizes (e.g., partial correlations) dropped considerably compared with the effect sizes seen when the cognitive vulnerability-stress interactions from HT and BT were analyzed independently, further suggesting considerable overlap of the cognitive vulnerability-stress components in HT and BT.

Discussion

Results from Study 1 were consistent with hypotheses. Cognitive vulnerability for depression interacted with negative life events encountered over the 5-weeks follow-up interval to predict prospective elevations of depressive symptoms specifically, but not anxiety symptoms, at T2. This etiological affective specificity was found regardless of whether composite depression and anxiety were used or whether the specific tripartite theory measures of anhedonic depression and anxious arousal were examined. Negative life events operated as a nonspecific risk factor for both depression and anxiety as prospective changes in stressors over time were associated with elevations in affective symptoms. The cognitive vulnerability-stress interaction was found to predict depression for both HT's Negative cognitive style × Stress and BT's Dysfunctional attitudes × Stress when examined separately. When analyses were conducted to compare directly the uniqueness of HT or BT, neither cognitive vulnerability-stress interaction remained significant after controlling for the other model's vulnerability-stress component, suggesting that both HT and BT's vulnerability-stress interaction are generally effective by themselves, but overlap in their prediction of depressive symptoms. These findings were consistent with the study's hypotheses and previous short-term prospective vulnerability-stress studies of depression (e.g., Joiner et al. 1999; Metalsky & Joiner, 1992). Importantly, the current findings expand on these past findings by examining both HT and BT together simultaneously and by testing these models for affective symptom specificity.

A possible limitation of this study is its relatively short prospective follow-up. To introduce a stronger test of the affective specificity of HT' and BT's cognitive vulnerability-stress components, we conducted a second study. This second study was similar in most respects to Study 1 except that (1) a longer time interval between T1

and T2 was used (2-years instead of 5-weeks) and (2) diagnostic interviews were used to assess for clinically significant levels of depression and anxiety over the 2-years follow-up in addition to affective symptom levels.

STUDY 2

Method

Participants and Procedures

Unselected undergraduate students served as participants. Participants completed a packet of questionnaires for the initial assessment (T1). In Study 2, participants completed a follow-up assessment (T2) that occurred 2-years after the initial assessment. The participants who completed the follow-up packet of questionnaires were paid for their time. A total of 233 (70 male) participants completed the follow-up assessment out of 258.

Measures

Cognitive Vulnerability. The same CSQ and DAS from Study 1 were used in Study 2 to measure HT's negative cognitive style and BT's dysfunctional attitudes respectively. They were administered at T1.

*Negative Life Events Questionnaire (*NLEQ; Metalsky & Joiner, 1992*).* The same NLEQ was given at T1 and T2. At Time 2, participants were instructed to indicate which of the 67 events had occurred to them over the 2-years period. The time frame for NLEQ given at T1 was the 10 weeks preceding the initial assessment.

Diagnostic Interview for Depressive and Anxiety Disorders. This diagnostic interview was used only in Study 2. From the 233 participants who completed questionnaire measures in Study 2, a randomly selected 75 (34 male) participants were interviewed with an expanded version of the Schedule for Affective Disorders and Schizophrenia—Lifetime (SADS-L) interview (see Alloy et al., 2000). Diagnoses of depressive disorder included major depressive disorder and dysthymia based on criteria specified in the *Diagnostic and Statistical Manual*—4th edition (*DSM-IV*; APA, 1994). Diagnoses of anxiety disorder included panic disorder, social phobia, generalized anxiety disorder, obsessive–compulsive disorder, and specific phobia, based on *DSM-IV* criteria. The original SADS-L (Endicott & Spitzer, 1978) was expanded to include additional probes for making diagnoses according to *DSM-IV*. The interview was expanded as part of the Temple–Wisconsin Cognitive Vulnerability to Depression (CVD) project, and this expansion has been found to be reliable and valid (Alloy et al., 2000). The lead author (BLH) conducted interviews within 2 months after the participants completed the T2 packet of questionnaires. The first author completed training for diagnostic interviewing based on the CVD project; this training program has achieved good reliability (kappa reliability for all project diagnoses above .90; Alloy et al., 2000). Similar reliability was observed in the present study (100% agreement for approximately 10% of interviews rated by a 2nd interviewer). Participants were interviewed for occurrence of depressive or anxiety disorders that happened

during the 2-years prospective interval. The occurrence of depressive disorder was coded as 1; none as 0. Anxiety disorder occurrence was coded as 1; none as 0. In addition, lifetime diagnoses of depression and anxiety were obtained, but only the occurrence of disorders during the 2-years prospective interval between T1 and T2 are reported here. Importantly, none of the participants who experienced an episode of depression or anxiety during the prospective follow-up were diagnosed with a current anxiety or depressive disorder at the start of the study (T1).

*Beck Depression Inventory (*BDI; Beck et al., 1961*).* The BDI was given at T1 and T2. At Time 1, participants rated the items for the past 10 weeks prior to the initial assessment. We used a 10-week period, instead of the conventional instructions for the past 1-week, because we wanted to assess participants' stable, trait-like symptoms at baseline to control for these in analyses, and averaging a construct over longer period of times provides a more reliable assessment (Epstein, 1980). At T2 participants were instructed to respond to the BDI items thinking about 1 week in the past 2-years when they felt the most depressed. This methodology of thinking about a one week period in the past when most depressed has been used successfully and validly in previous research (e.g., Roberts & Gotlib, 1997; Zimmerman, Coryell, Corenthal, Corenphal, & Wilson, 1986).

*Mood and Anxiety Symptom Questionnaire (*MASQ; Watson, Weber, et al., 1995*).* The MASQ was given at T1 and T2. The instructions for the MASQ in Study 2 were the same as for the BDI. Participants at T2 were instructed to respond to the items thinking about 1 week in the past 2-years when they felt the most distressed. Although these instructions differ from how the MASQ has been used previously, this change in methodology and instructions is the same as the alterations made for the BDI and has been used validly in past research. At T1, participants answered for the past 10 weeks prior to T1 for the same reasons as stated for the BDI.

Results

Descriptive statistics and correlations for the main variables for Study 2 are presented in Table III. As in Study 1, the measures of cognitive vulnerability (CSQ and DAS) were moderately correlated with negative life events as well as with the different depressive and anxious symptom questionnaires. Also, negative events were moderately associated with both depressive and anxious symptoms as well as with clinically significant anxiety disorders and marginally with depressive disorders. As with Study 1, the measures of general depressive and anxious symptoms were correlated highly (i.e., around .60), although the discriminant correlations of anxious arousal and anhedonic depression were only moderate and relatively lower (i.e., around .30). Also, anxiety disorders were moderately correlated with general distress—anxiety and anxious arousal as well as marginally with some depressive symptoms. Depressive disorder was associated with depressive symptoms, but not anxious arousal or general distress—anxiety. At the diagnostic level, 33.3% individuals with a depressive disorder had an anxiety disorder.

The same data reduction and analytic plan from Study 1 was used in Study 2 to address the central question examining cognitive vulnerability-stress predictions for depression specifically over the 2-years follow-up. Specifically, we computed

Table III. Descriptive Statistics and Correlations Among Main Measures—Study 2

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1 CSQ																
2 DAS	.38															
3 BDI1	.51	.40														
4 GDANX1	.42	.34	.55													
5 GDDEP1	.44	.43	.69	.67												
6 DEP1	.37	.35	.61	.42	.74											
7 ANXAR1	.30	.35	.44	.73	.50	.32										
8 NLEQ1	.29	.27	.34	.32	.43	.38	.26									
9 BDI2	.36	.31	.61	.47	.60	.49	.37	.31								
10 GDANX2	.33	.29	.42	.60	.42	.26	.53	.31	.35							
11 GDDEP2	.28	.26	.49	.37	.55	.45	.30	.23	.49	.35						
12 DEP2	.20	.25	.43	.25	.44	.45	.22	.23	.43	.25	.80					
13 ANXAR2	.30	.29	.38	.60	.42	.26	.61	.26	.33	.74	.29	.23				
14 NLEQ2	.24	.34	.34	.31	.39	.35	.26	.46	.51	.27	.39	.46	.22			
15 DEP DX	.15	.11	.45***	.16	.29*	.10	.19	.10	.52***	.12	.25*	.22	.22	.23		
16 ANX DX	.16	.11	.28*	.25*	.26*	.14	.32**	.17	.26*	.37**	.26*	.28*	.26**	.31**	.11	
M	3.83	3.54	12.94	26.01	22.98	55.29	25.42	25.63	15.96	33.93	24.56	75.09	25.77	31.72	18.5%	25.9%
SD	.71	.49	9.05	8.60	6.28	14.05	7.53	11.38	10.30	11.31	7.69	16.5	9.08	11.84		

Note. $N = 233$ for all variables, except DEP DX and ANX DX, $N = 75$. CSQ = Cognitive Style Questionnaire; DAS = Dysfunctional Attitudes Scale; BDI = Beck Depression Inventory; GDDEP = general distress—depression; GDANX = general distress—Anxiety; DEP = anhedonic depressive symptoms; ANXAR = anxious arousal; NLEQ = Negative Life Events Questionnaire; DEP DX = clinical diagnoses of depressive disorder; ANX DX = clinical diagnoses of anxiety disorder.

For continuous measures, all correlations above are significant at $p < .001$ with $N = 233$. For correlations with DEP DX and ANX DX, $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

the composite depressive symptoms variable (DEPRESS) and a composite anxiety symptoms variable (ANX) in the same manner as Study 1. Further, the specific symptom factors from the tripartite model factors (anxious arousal and anhedonic depression) were examined also as in Study 1. Setwise hierarchical regressions for the continuous symptoms measures, in which T1 affective symptoms and stressors were controlled, were used also in Study 2. Logistic regression was used for the dichotomous outcome data of anxiety and depressive disorder in Study 2.

Test of the Cognitive Vulnerability-Stress Component for HT and BT: Composite Depressive and Anxious Symptoms

The results for HT and BT in Study 2 were the same as in Study 1. As seen in Table IV, the main effect of T2 NLEQ, independent of CSQ, predicted T2 composite depressive symptoms. The vulnerability-stress component for HT, the CSQ × NLEQ2 interaction, predicted T2 depressive symptoms. For the test of BT, as seen in Table IV, the main effect of T2 NLEQ, independent of DAS, predicted T2 composite depressive symptoms. The test of BT's vulnerability-stress component, the DAS × NLEQ2 interaction, predicted T2 depressive symptoms. These analyses show higher levels of cognitive vulnerability (from either HT or BT) combined with greater number of stressors was associated with the highest elevations in T2 depressive symptoms. The form of these interactions was graphed and showed the same pattern as in Study 1 (see Fig. 1), so are not presented for space.

The results of Study 2 for HT's and BT's cognitive vulnerability-stress components predicting T2 composite anxiety symptoms were the same as in Study 1. As seen in Table IV, neither of the main effects of HT's negative cognitive style nor BT's dysfunctional predicted T2 anxiety symptoms. Further, neither cognitive vulnerability-stress interaction predicted T2 anxiety symptoms. However, T2 negative life events predicted T2 anxiety symptoms, even after controlling for initial levels of negative events and anxiety symptoms.

Consistent with Study 1, the results above show that the cognitive vulnerability-stress interaction from both HT and BT predicted T2 depressive, but not anxious symptoms. A more stringent test of the affective specificity of the cognitive vulnerability-stress interaction predicting depressive symptoms was conducted as in Study 1 by controlling for initial levels of *both* depression and anxiety composite symptoms to predict T2 depressive symptoms. Results from Study 2 showed that BT's Dysfunctional attitudes × Stress interaction ($\beta = .55$, $t = 4.41$, $p < .001$) and HT's Negative cognitive style × stress interaction ($\beta = .52$, $t = 4.23$, $p < .001$) predicted T2 depressive symptoms.[6] Thus, even when initial levels of anxiety, depression, and stressors are controlled, the Cognitive vulnerability × Stress interaction from HT and BT predicted T2 depressive symptoms over a 2-years interval.

Test of Vulnerability-Stress Component for HT and BT: Tripartite Model's Specific Depressive and Anxious Symptoms

As seen in Table IV, the main effect of T2 NLEQ, independent of CSQ, predicted T2 anhedonic depressive symptoms. The vulnerability-stress component for HT, the

Table IV. Cognitive Vulnerability, Negative Life Events, and Interaction Predicting T2 Composite Symptoms and Tripartite Model Factor Symptoms Controlling for T1 Symptoms and T1 Stressors in Study 2

Predictor	β	*pr* (Partial correlation)	*t*	Step R^2 change
Composite symptom analyses				
HT; Depressive symptoms				
Step 1				.43
T1 DEPRESS covariate	.63	.63	11.52***	
T1 NLEQ covariate	.05	.05	0.84	
Step 2				.09
CSQ	.01	.01	0.22	
T2 NLEQ	.35	.40	6.40***	
Step 3				.04
CSQ × T2 NLEQ	.52	.28	4.26***	
	Model $R^2 = .56$, $F(4, 233) = 54.24$, $p < .001$			
HT; Anxious symptoms				
Step 1				.25
T1 ANX covariate	.51	.51	8.30***	
T1 NLEQ covariate	.05	.05	0.89	
Step 2				.10
CSQ	.11	.13	1.90	
T2 NLEQ	.34	.35	5.45***	
Step 3				.01
CSQ × T2 NLEQ	.28	.13	1.93	
	Model $R^2 = .36$, $F(4, 233) = 23.85$, $p < .001$			
BT; Depressive symptoms				
Step 1				.43
T1 DEPRESS covariate	.63	.63	11.52***	
T1 NLEQ covariate	.05	.05	0.84	
Step 2				.09
DAS	.08	.06	0.82	
T2 NLEQ	.36	.40	6.41***	
Step 3				.04
DAS × T2 NLEQ	.56	.29	4.44***	
	Model $R^2 = .56$, $F(4, 233) = 54.67$, $p < .001$			
BT; Anxious symptoms				
Step 1				.25
T1 ANX covariate	.51	.51	8.30***	
T1 NLEQ covariate	.05	.05	0.89	
Step 2				.09
DAS	.02	.03	0.40	
T2 NLEQ	.35	.35	5.40***	
Step 3				.01
DAS × T2 NLEQ	.27	.12	1.87	
	Model $R^2 = .35$, $F(4, 233) = 25.25$, $p < .001$			
HT and BT combined depressive symptoms				
Step 1				.43
T1 DEPRESS covariate	.63	.63	11.52***	
T1 NLEQ covariate	.05	.05	0.84	
Step 2				.09
CSQ	.01	.01	0.06	
DAS	.04	.05	0.79	
T2 NLEQ	.35	.40	6.38***	
Step 3				.03
CSQ × T2 NLEQ	.11	.02	0.35	
DAS × T2 NLEQ	.46	.09	1.42	
	Model $R^2 = .56$, $F(7, 233) = 38.74$, $p < .001$			

Table IV. Continued

Predictor	β	*pr* (Partial correlation)	*t*	Step R^2 change
Tripartite Model Analyses				
HT; Anhedonic depression				
Step 1				.21
T1 DEP covariate	.43	.41	6.59***	
T1 NLEQ covariate	.09	.09	1.40	
Step 2				.10
CSQ	.01	.01	0.21	
T2 NLEQ	.37	.35	5.24***	
Step 3				.03
CSQ × T2 NLEQ	.45	.21	3.13***	
	Model $R^2 = .34$, $F(4, 233) = 18.56$, $p < .001$			
HT; Anxious arousal				
Step 1				.39
T1 ANXAR covariate	.62	.62	11.80***	
T1 NLEQ covariate	.02	.02	0.30	
Step 2				.04
CSQ	.06	.07	1.09	
T2 NLEQ	.17	.21	3.15**	
Step 3				.003
CSQ × T2 NLEQ	.11	.07	1.01	
	Model $R^2 = .43$, $F(4, 233) = 40.41$, $p < .001$			
BT; Anhedonic depression				
Step 1				.21
T1 DEP covariate	.43	.41	6.59***	
T1 NLEQ covariate	.09	.09	1.40	
Step 2				.10
DAS	.04	.04	.60	
T2 NLEQ	.35	.33	4.91***	
Step 3				.02
DAS × T2 NLEQ	.38	.17	2.61**	
	Model $R^2 = .33$, $F(4, 233) = 18.38$, $p < .001$			
BT; Anxious arousal				
Step 1				.39
T1 ANXAR covariate	.62	.62	11.82***	
T1 NLEQ covariate	.02	.02	0.30	
Step 2				.04
DAS	.0	.01	0.17	
T2 NLEQ	.19	.23	3.42***	
Step 3				.002
DAS × T2 NLEQ	.09	.06	0.91	
	Model $R^2 = .43$, $F(4, 233) = 39.90$, $p < .001$			
HT and BT combined anhedonic depression				
Step 1				.20
T1 DEP covariate	.41	.41	6.00***	
T1 NLEQ covariate	.09	.09	1.40	
Step 2				.10
CSQ	−.02	−.02	−0.37	
DAS	.04	.04	0.57	
T2 NLEQ	.36	.34	5.42***	
Step 3				.03
CSQ × T2 NLEQ	.67	.12	1.81	
DAS × T2 NLEQ	−.26	−.05	−0.67	
	Model $R^2 = .33$, $F(6, 233) = 16.01$, $p < .001$			

Note. CSQ = negative cognitive style; DAS = dysfunctional attitudes scale; DEPRESS = composite measures of depressive symptoms; ANX = composite measure of anxious symptoms; DEP = anhedonic depressive symptoms; ANXAR = anxious arousal symptoms; NLEQ = Negative Life Events Questionnaire.
*$p < .05$. **$p < .01$. ***$p < .001$.

CSQ × NLEQ2 interaction, predicted T2 anhedonic depressive symptoms. For the test of BT, as seen in Table IV, the main effect of T2 NLEQ, independent of DAS, predicted T2 anhedonic depressive symptoms. The test of BT's vulnerability-stress component, the DAS × NLEQ2 interaction, predicted T2 anhedonic depressive symptoms significantly. The form of these interactions was the same as shown in Fig. 1 for composite depression and is not shown for space considerations.

Table IV shows the results for predicting T2 anxious arousal symptoms. Neither of the main effects of HT's negative cognitive style nor BT's dysfunctional predicted T2 anxious arousal symptoms. Further, neither cognitive vulnerability-stress interaction predicted T2 anxious arousal symptoms. However, T2 negative life events predicted T2 anxious arousal symptoms, even after controlling for initial levels of stressors and anxious arousal symptoms.

Test of Vulnerability-Stress Component Predicting Anxiety and Depressive Disorder

Logistic regression analyses were used to predict the occurrence of depressive disorder from T1 to T2 based on the cognitive vulnerability-stress component of HT and BT. The order of entry for the logistic regressions was the same as for the hierarchical regressions conducted above. Results are shown in Table V. Initial composite depressive symptoms predicted depressive disorder. The second step with the main effects was nonsignificant, and neither individual effect of cognitive vulnerability or T2 negative events was significant. Last, the addition of the vulnerability-stress component was significant for both HT and BT. HT's CSQ × NLEQ2 interaction and BT's DAS × NLEQ2 interaction both predicted occurrence of depressive disorder, even after controlling for initial depression and initial stressors. These analyses show that individuals with high cognitive vulnerability who experienced more negative events over the 2-years follow-up were the most likely to experience the occurrence of a depressive disorder.

Logistic regression analyses were conducted to predict occurrence of anxiety disorders over the 2-years follow-up while controlling for T1 composite anxiety symptoms and T1 negative events. As seen in Table V, only the main effect of initial anxiety symptoms predicted occurrence of anxiety disorders from T1 to T2. Although negative events were significantly associated with the occurrence of anxiety disorders at the zero-order correlational level (see Table IV), they no longer significantly predicted anxiety disorder after controlling for initial anxiety symptoms and initial levels of stressors. Neither vulnerability-stress component, from HT or BT, significantly predicted occurrence of anxiety disorder.

In the analyses above, we found that cognitive vulnerability (both from HT and BT) interacted with negative events to predict prospective occurrence of depressive disorder over the 2-years interval. We sought to examine whether this result for depressive disorder would be maintained after initial T1 composite anxiety symptoms as well as depressive symptoms were controlled statistically. Results from these logistic regressions showed that BT's DAS × NLEQ2 interaction ($\beta = 1.72$, Wald $= 3.41$, $OR = 5.83$, $p < .05$) and HT's CSQ × NLEQ2 interaction ($\beta = 2.28$, $Wald = 4.53$, $OR = 9.98$, $p < .05$) predicted occurrence of depressive disorder. Thus,

Table V. Logistic Regression Analyses of Depressive and Anxiety Disorder Status as Function of Cognitive Vulnerability, Negative Life Events, and Interaction From T1 to T2 in Study 2

Predictor	Step χ^2 change	Wald	β	Odds ratio	Step R^2 change
Depressive disorders—HT					
Step 1	10.16**				.22
T1 DEPRESS covariate		8.4	.44	1.55**	
T1 NLEQ		1.09	.04	1.02	
Step 2	3.50				.07
Cognitive style		.99	.59	1.54	
T2 Negative events		2.33	.72	2.06	
Step 3					
Cognitive style × Stress interaction	5.37*	4.61	2.30	10.03*	.10
Depressive disorders—BT					
Step 1	10.16**				.22
T1 DEPRESS covariate		8.4	.44	1.55**	
T1 NLEQ		1.09	.04	1.02	
Step 2	3.18				.07
Dysfunctional attitudes		.75	.73	1.24	
T2 negative events		2.47	.77	2.16	
Step 3					
Dysfunctional attitudes × Stress interaction	3.77*	3.43	1.76	5.84*	.10
Depressive disorders—HT and BT combined					
Step 1	10.16**				.22
T1 DEPRESS covariate		8.4	.44	1.55**	
T1 NLEQ		1.09	.04	1.02	
Step 2	4.42				.09
Cognitive Style		1.15	.66	1.37	
Dysfunctional attitudes		.94	.70	1.18	
T2 negative events		2.55	.77	2.16	
Step 3	5.49*				.10
Cognitive style × Stress	1.3	1.15	5.36		
Dysfunctional attitudes × Stress	.24	.88	2.25		
Anxiety disorders—HT					
Step 1	6.45*				.13
T1 ANX covariate	4.53	.33	1.4*		
T1 NLEQ covariate		.06	.07	1.08	
Step 2	.29				.01
Cognitive style		.07	.03	.97	
T2 Negative events		.29	.19	1.21	
Step 3	3.53				.04
Cognitive style × Stress interaction		3.05	1.45	3.67	
Anxiety disorders—BT					
Step 1	6.45*				.13
T1 ANX covariate		4.53	.33	1.4*	
T1 NLEQ covariate	.06	.07	1.08		
Step 2	.75				.01
Dysfunctional attitudes		.19	.13	.79	
T2 negative events		.38	.22	1.25	
Step 3	3.54				.03
Dysfunctional attitudes × Stress interaction		3.09	1.52	3.96	

Note. $N = 75$. ANX = Composite of anxiety symptoms; DEPRESS = Composite of depressive symptoms.
*$p < .05$. **$p < .01$. ***$p < .001$.

the cognitive vulnerability-stress interactions (from both HT and BT) were associated with prospective occurrence of depressive disorder even after controlling for initial levels of both anxiety and depressive symptoms, initial stressors, and the main effects of cognitive vulnerability and prospective stressors.

These analyses show that the Cognitive vulnerability × Stress interaction from HT and BT predicts occurrence of depressive disorder from T1 to T2. To illustrate this, we graphed the vulnerability-stress interaction. These graphical results for depressive disorder are represented in Fig. 2 for HT and BT. Consistent with Fig. 1 for depressive symptoms, Fig. 2 shows that individuals with higher levels of cognitive vulnerability, when encountering high levels of stressors, reported the highest occurrence of depressive disorder over the prospective follow-up.

Analyses Comparing the Cognitive Theories of Depression

As in Study 1, similar regression equations were fit in which T1 depressive symptoms and T1 negative events were entered first, followed by both cognitive factors (CSQ and DAS) and T2 negative life events entered second, and last by both of the Cognitive vulnerability × Stress (CSQ × NLEQ2; DAS × NLEQ2) interactions entered third. Table IV shows these results for composite depressive symptoms and for anhedonic depression and Table V for depressive disorder. Consistent with findings from Study 1, these analyses suggested considerable overlap of the cognitive vulnerability-stress components in HT and BT. Neither cognitive vulnerability-stress component uniquely predicted T2 composite depressive symptoms, anhedonic depressive symptoms, or occurrence of depressive disorder. Across all of these analyses, the magnitude of the effect sizes (e.g., partial correlations) dropped considerably compared with the effect sizes seen when the cognitive vulnerability-stress interactions from HT and BT were analyzed independently, further suggesting considerable overlap of the cognitive vulnerability-stress components in HT and BT.

Discussion

Results from Study 2 were consistent with hypotheses and with the findings from Study 1. Cognitive vulnerability for depression interacted with negative life events encountered over the 2 year follow-up interval to predict prospective elevations of depressive symptoms specifically, but not anxiety symptoms, at T2. This pattern was found regardless of whether composite depression and anxiety were used or the specific tripartite theory measures of anhedonic depression and anxious arousal. In addition, the cognitive vulnerability-stress interaction predicted occurrence of clinically significant depressive disorder, but not anxiety disorders, over the 2-years prospective follow-up. As in Study 1, negative life events were a nonspecific risk factor for both depression and anxiety and were associated with elevations in affective symptoms. Both HT's and BT's cognitive vulnerability-stress interaction were found to predict depression when examined separately, but analyses that compared directly the uniqueness of HT or BT showed that neither cognitive vulnerability-stress interaction remained significant after controlling for the other model's vulnerability-stress component.

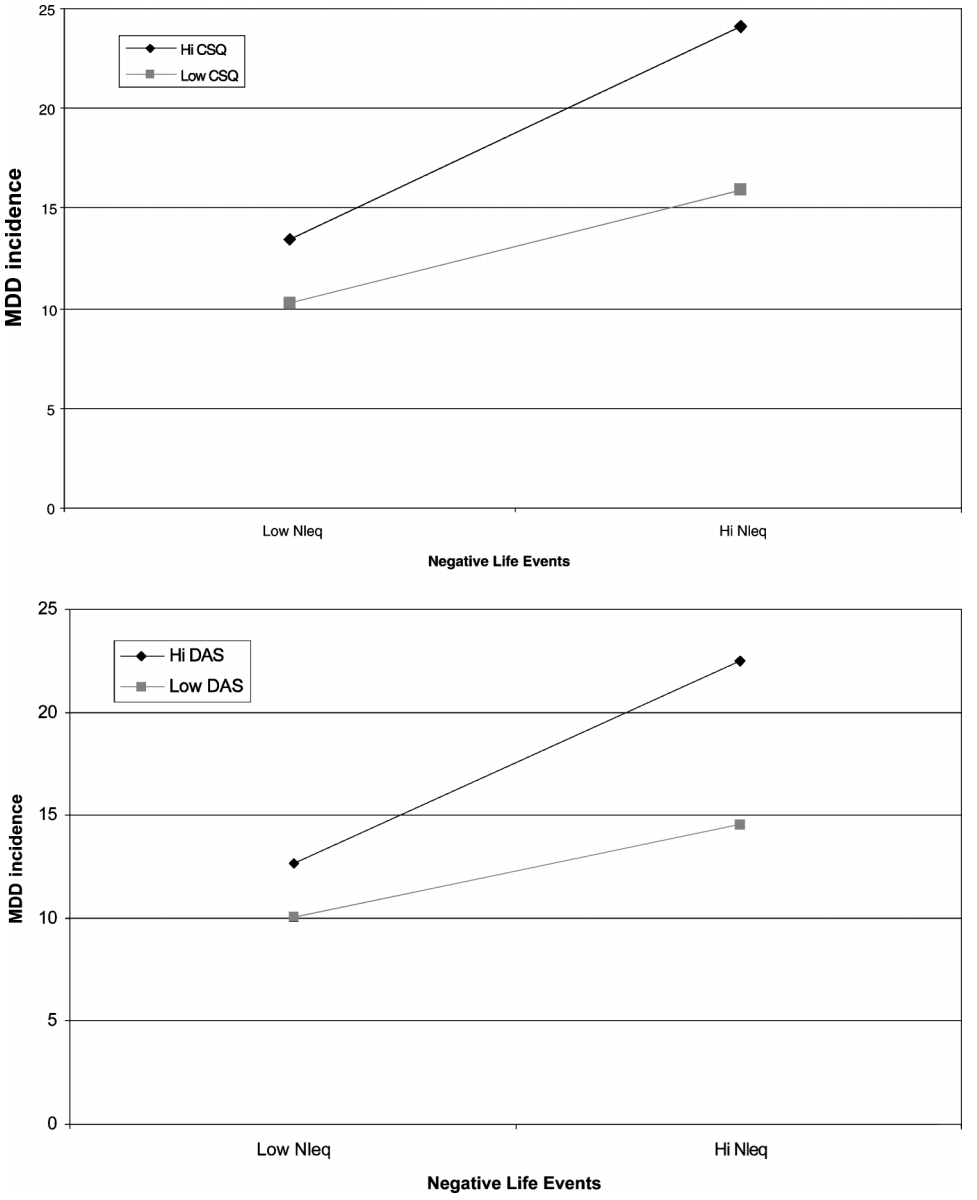

Fig. 2. Interaction between cognitive vulnerability and negative events in the prediction of occurrence of depressive disorder in Study 2 from Time 1 to Time 2 for HT (top half) and BT (bottom half). CSQ = HT's negative cognitive style and DAS = BT's dysfunctional attitudes.

A strength of Study 2 is its longer follow-up period (2 years) compared with the shorter-term prospective interval of Study 1 and most past studies. Across short and long-term prospective follow-ups, the same pattern of findings was revealed. This replication across studies with different time periods enhances confidence in the

cognitive vulnerability-stress hypotheses and their affective specificity. In addition, in Study 2 we examined etiological factors predicting both depressive and anxiety symptoms and disorder, whereas most prior studies have only investigated etiological factors at the symptom level. Importantly, results were the same for self-report questionnaire assessment of symptoms and clinical interview assessment of disorder.

Still, a potential limitation of both Studies 1 and 2 is that it was not possible to date precisely when participants encountered stressors using a negative events checklist given at T2 to assess the number of stressors experienced over the follow-up (from either Study 1 or 2). Depressive or anxious symptoms may have increased prior to encountering stressors. Thus, it is unclear whether negative life events, in fact, preceded elevations in affective symptoms over the follow-up as hypothesized. Indeed, the relationship between stressors and emotional distress is complex and transactional (Hankin & Abramson, 2001). Negative life events have been found to precede depression, and depressed individuals often generate additional stressors (Hammen, 1991; Monroe & Hadjiyannakis, 2002).

Given this known complexity, we controlled for baseline levels of both affective symptoms and stressors in both Studies 1 and 2, before entering our explanatory variables (i.e., cognitive vulnerability and stressors experienced over the follow-up), to reduce the potential interpretative confound that initial levels of emotional distress or stressors (prior to entry into the study) might have caused for testing the cognitive vulnerability-stress interactions as etiological factors predicting prospective elevations in depression. Although controlling for initial levels of both symptoms and stressors cannot completely solve the temporal resolution of future stressors and symptoms, using this conservative covariance data analytic approach with a prospective design establishes the temporal precedence that cognitive vulnerability, assessed at the outset, interacts with prospective changes in negative events to predict elevations in depression. This temporal precedence enhances the confidence that the cognitive vulnerability-stress component is a risk factor for future increases in depression and not simply a correlate of depression or anxiety (Barnett & Gotlib, 1988; Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001).

However, even by controlling for initial levels of affective symptoms and stressors, more precise timing of the relation between stress and symptom elevation is not possible with the 2 time point panel design employed in Studies 1 and 2. Another methodological strategy to examine vulnerability-stress hypotheses is to use a prospective design that involves naturalistically occurring stressors (e.g., Abela & Seligman, 2001; Metalsky et al., 1987, 1993; Ralph & Mineka, 1998), in which the timing of the stressor and the development of emotional distress symptoms are known and precisely assessed. Past research with this design, known as the "academic midterm study," shows that initial levels of cognitive vulnerability interact with the naturalistic stressor (failure on a class exam) to predict enduring elevations in emotional distress and depressive symptoms several days after receipt of the exam grade. Thus, the primary advantage of the academic midterm design is that it allows for a strong and precise test of the temporal precedence and unfolding of depressive and anxious symptoms after experiencing a stressor because the timing of the midterm exam and changes in affective symptoms over time is carefully documented. In Study 3, we used the academic midterm design to test HT's and BT's cognitive

vulnerability-stress components predicting depressive symptoms more specifically than anxiety in a manner that permits precise dating in the assessment of cognitive vulnerability, stressor, and development of affective symptoms over time.

STUDY 3

Method

Participants and Procedures

Participants were 110 (39 men) undergraduates randomly selected from psychology courses at a large midwestern university; they participated in the study for extra credit.

At Time 1 (2 weeks before the midterm exam), students completed measures of cognitive vulnerability (HT's negative cognitive style and BT's dysfunctional attitudes), mood and anxiety symptoms (BDI and MASQ), and their aspirations for performance on the exam. Students then took their first exam and received their grade in class 1 week after the exam. Time 2 occurred in class on the day the students received their exam grade. Time 3 occurred 5 days after receipt of their exam grade. At Times 2 and 3, the students completed mood and anxiety symptom measures (BDI and MASQ). The timing and procedures of the present academic midterm study are very similar to those previously used to examine vulnerability-stress hypotheses (e.g., Metalsky et al., 1993; Ralph & Mineka, 1998).

Measures

Cognitive Vulnerability. The same CSQ and DAS from Study 1 and 2 were used in Study 3 to measure HT's negative cognitive style and BT's dysfunctional attitudes respectively. They were administered at T1 2 weeks before the midterm exam.

Beck Depression Inventory (BDI; Beck et al., 1961). The BDI was given at T1, T2, and T3. At Times 1, 2, and 3 participants rated the depressive symptoms items for the past week.

Mood and Anxiety Symptom Questionnaire (MASQ; Watson, Weber, et al., 1995). The MASQ was given at T1, T2, and T3. The instructions for the MASQ in Study 3 were the same as for the BDI. Participants were instructed to respond to the items thinking about the past week.

Outcome on Midterm Exam. At T1, students were asked about their aspirations for the grade on the first exam. Their exam aspirations were subtracted from the actual grade to yield a failure score (higher score means a more negative outcome on the exam). This same procedure was used in the prior academic midterm studies to obtain a naturalistic, objective stressor.

Results

Descriptive statistics and correlations for the main variables for Study 3 are presented in Table VI. Similar correlations were obtained in Study 3 as in Study 1 and

Table VI. Descriptive Statistics and Correlations Among Main Measures—Study 3

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1 CSQ																		
2 DAS	.49																	
3 BDI1	.57	.35																
4 GDANX1	.48	.33	.68															
5 GDDEP1	.61	.33	.85	.73														
6 DEP1	.57	.25	.74	.53	.75													
7 ANXAR1	.27	.28	.57	.75	.54	.31												
8 EXAM	.06	.06	.03	.08	.02	.00	.06											
9 BDI2	.57	.44	.78	.57	.65	.58	.55	.16										
10 GDANX2	.35	.47	.53	.61	.52	.42	.58	. 06	.56									
11 GDDEP2	.56	.47	.77	.63	.72	.64	.48	. 06	.76	.62								
12 DEP2	.54	.36	.64	.46	.64	.80	.18	. 14	.70	.44	.69							
13 ANXAR2	.22	.29	.49	.52	.47	.35	.65	. 05	.52	.63	.57	.33						
14 BDI3	.55	.41	.77	.56	.63	.56	.53	.15	.77	.55	.50	.42	.34					
15 GDANX3	.33	.43	.52	.61	.50	.40	.55	.01	.56	.58	.51	.44	.71	.65				
16 GDDEP3	.53	.43	.75	.64	.71	.61	.45	.09	.73	.64	.73	.65	.62	.73	.74			
17 DEP3	.51	.33	.61	.45	.64	.80	.15	.02	.65	.28	.63	.77	.41	.66	.39	.65		
18 ANXAR3	.20	.28	.49	.49	.46	.32	.60	.10	.35	.56	.45	.38	.71	.35	.77	.51	.26	
M	3.66	3.45	8.07	19.8	27.5	58.6	25.3	1.4	8.5	18.4	23.1	56.2	25.8	8.8	15.9	21.3	59.9	21.3
SD	0.87	0.45	8.7	6.8	6.07	15.6	9.6	1.04	10.7	6.6	9.3	10.7	9.2	11.05	6.07	10.05	16.6	7.4

Note. $N = 110$ for all variables. CSQ = Cognitive Style Questionnaire; DAS = Dysfunctional Attitudes Scale; BDI = Beck Depression Inventory; GDDEP = general distress—depression; GDANX = general distress—anxiety; DEP = anhedonic depressive symptoms; ANXAR = anxious arousal; EXAM = deviation between aspiration on exam and actual exam grade.
Correlations above .19 are significant at $p < .05$, above .25 at $p < .01$, and above .30 at $p < .001$.

2 and in the previous midterm studies. The measures of cognitive vulnerability (CSQ and DAS) were moderately correlated with the different depressive and anxious symptom questionnaires.

The same data reduction and analytic plan from Study 1 and 2 was used in Study 3 to address the central question examining cognitive vulnerability-stress predictions for depression specifically for T3. Specifically, we computed the composite depressive symptoms variable (DEPRESS) and a composite anxiety symptoms variable (ANX). Further, the specific symptom factors from the tripartite model factors (anxious arousal and anhedonic depression) were examined. Setwise hierarchical regressions were used to test the hypothesis that cognitive vulnerability would interact with the failure stressor to predict enduring elevations in depressive symptoms (at T3) specifically, but not anxiety symptoms.

Test of the Cognitive Vulnerability-Stress Component for HT and BT: Composite Depressive and Anxious Symptoms

The results for HT and BT were consistent generally with these hypotheses. As seen in Table VII, the vulnerability-stress component for HT, the CSQ × Exam failure interaction, and for BT, the DAS × Exam failure, both predicted T3 depressive symptoms. These analyses show higher levels of cognitive vulnerability (from HT and BT) combined with an objective, naturalistic stressor assessed at a specific time in relation to assessments of affective symptoms was associated with the persistent elevations in depressive symptoms at T3.

Consistent with Study 1 and 2, we conducted the more stringent test of the affective specificity of the cognitive vulnerability-stress interaction predicting depressive symptoms by controlling for initial levels of *both* depression and anxiety composite symptoms to predict T3 depressive symptoms. Results showed that BT's Dysfunctional attitudes × Exam failure interaction ($\beta = 1.78, t = 2.98, p < .01$) and HT's Negative cognitive style × Exam failure interaction ($\beta = .76, t = 2.33, p < .01$) predicted T3 composite depressive symptoms. Thus, even when initial levels of anxiety, depression, and stressors are controlled, the Cognitive vulnerability × Failure interaction from HT and BT predicted enduring depressive symptoms at T3.

Test of Vulnerability-Stress Component for HT and BT: Tripartite Model's Specific Depressive and Anxious Symptoms

As seen in Table VII, the vulnerability-stress component for HT, the CSQ × Exam failure interaction, and for BT, the DAS × Exam failure both predicted T3 anhedonic depressive symptoms. Neither BT's nor HT's vulnerability-stress component predicted T3 anxious arousal symptoms significantly.

Analyses Comparing the Cognitive Theories of Depression

As in Study 1 and 2, regression equations were fit in which T1 depressive symptoms was entered first, followed by both cognitive factors (CSQ and DAS) and exam failure entered second, and last by both of the Cognitive vulnerability × Stress

Table VII. Hierarchical Regression Analyses of Composite and Tripartite Model's Depressive and Anxiety Symptoms as Function of Cognitive Vulnerability, Midterm Exam Failure, and Interaction in Study 3

Predictor	β	*pr* (Partial correlation)	*t*	Step R^2 change
Composite symptom analyses				
HT; Composite depressive symptoms T3				
Step 1				.49
T1 DEPRESS covariate	.69	.69	9.99***	
Step 2				.01
CSQ	.12	.13	1.36	
EXAM	.03	.04	0.44	
Step 3				.02
CSQ × EXAM	.75	.22	2.31**	
	Model $R^2 = .52$, $F(4, 110) = 27.82$, $p < .001$			
Composite anxious symptoms T3				
Step 1				.17
T1 ANX covariate	.41	.41	4.57***	
Step 2				.08
CSQ	.30	.32	3.42**	
EXAM	.04	.04	0.45	
Step 3				.0
CSQ × EXAM	.03	.03	0.06	
	Model $R^2 = .25$, $F(4, 110) = 8.66$, $p < .001$			
BT; Composite depressive symptoms T3				
Step 1				.49
T1 DEPRESS covariate	.69	.69	9.99***	
Step 2				.02
DAS	.16	.21	2.22*	
EXAM	.01	.01	0.15	
Step 3				.04
DAS × EXAM	1.85	.29	3.12**	
	Model $R^2 = .55$, $F(4, 110) = 31.64$, $p < .001$			
Composite Anxious Symptoms T3				
Step 1				.17
T1 ANX covariate	.41	.41	4.57***	
Step 2				.05
DAS	.22	.22	2.35*	
EXAM	.04	.05	0.49	
Step 3				.0
DAS × EXAM	.83	.10	1.05	
	Model $R^2 = .22$, $F(4, 110) = 7.16$, $p < .001$			
Composite depressive symptoms T3—HT and BT combined				
Step 1				.49
T1 DEPRESS covariate	.69	.69	9.99***	
Step 2				.02
Cognitive Style	.05	.06	0.57	
Dysfunctional Attitudes	.14	.18	1.83	
EXAM	.03	.05	0.51	
Step 3				.04
Cognitive Style × EXAM	.10	.03	0.26	
Dysfunctional Attitudes × EXAM	1.72	.22	2.32*	
	Model $R^2 = .55$, $F(6, 110) = 20.76$, $p < .001$			
Tripartite Model Analyses				
HT; Anhedonic depressive symptoms T3				
Step 1				.41
T1 DEP covariate	.64	.64	8.62***	
Step 2				.00
CSQ	.05	.05	0.50	
EXAM	.02	.02	0.21	

Table VII. Continued

Predictor	β	*pr* (Partial correlation)	t	Step R^2 change
Step 3				.02
CSQ × EXAM	.81	.22	2.30**	
	Model $R^2 = .44$, $F(4, 110) = 20.43$, $p < .001$			
Anxious arousal symptoms T3				
Step 1				.12
T1 ANXAR covariate	.35	.35	3.88***	
Step 2				.01
CSQ	.06	.06	0.67	
EXAM	.05	.06	0.57	
Step 3				.0
CSQ × EXAM	.30	.07	0.69	
	Model $R^2 = .13$, $F(4, 110) = 4.03$, $p < .001$			
BT; Anhedonic depressive symptoms T3				
Step 1				.41
T1 DEP covariate	.64	.64	8.62***	
Step 2				.01
DAS	.11	.15	1.49	
EXAM	.02	.03	0.27	
Step 3				.03
DAS × EXAM	1.35	.20	2.06*	
	Model $R^2 = .45$, $F(4, 110) = 20.85$, $p < .001$			
Anxious arousal symptoms T3				
Step 1				.12
T1 ANXAR covariate	.35	.35	3.88***	
Step 2				.02
DAS	.14	.14	1.49	
EXAM	.05	.07	0.97	
Step 3				.01
DAS × EXAM	.79	.09	0.97	
	Model $R^2 = .15$, $F(4, 110) = 4.67$, $p < .001$			
Anhedonic depressive symptoms T3—HT and BT combined				
Step 1				.41
T1 DEP covariate	.64	.64	8.62***	
Step 2				.01
Cognitive Style	.02	.02	.16	
Dysfunctional Attitudes	.12	.14	1.41	
EXAM	.02	.02	.26	
Step 3				.03
Cognitive Style × EXAM	.45	.10	1.01	
Dysfunctional Attitudes × EXAM	.88	.11	1.08	
	Model $R^2 = .45$, $F(6, 110) = 13.98$, $p < .001$			

Note. CSQ = negative cognitive style; DAS = Dysfunctional Attitudes Scale; DEPRESS = composite measures of depressive symptoms; ANX = composite measure of anxious symptoms; DEP = anhedonic depressive symptoms; ANXAR = anxious arousal symptoms; NLEQ = Negative Life Events Questionnaire.
*$p < .05$. **$p < .01$, ***$p < .001$.

(CSQ × Exam failure; DAS × Exam failure) interactions entered third. Table VII shows these results for composite depressive symptoms and for anhedonic depression. These analyses showed that BT's Dysfunctional attitudes × Exam failure interaction uniquely predicted T3 composite depressive symptoms, whereas HT's Negative cognitive style × Exam failure did not. In contrast, neither HT's nor BT's cognitive vulnerability-stress component uniquely predicted anhedonic depressive symptoms.

Even though BT's Dysfunctional attitudes × Stress interaction remained a significant predictor of composite depressive symptoms, across all of these analyses the magnitude of the effect sizes (e.g., partial correlations) dropped considerably compared with the effect sizes seen when the cognitive vulnerability-stress interactions from HT and BT were analyzed independently. Taken together with the results from Studies 1 and 2, this suggests considerable overlap of the cognitive vulnerability-stress components in HT and BT.

Discussion

Results from Study 3 were consistent with hypotheses and with the findings from Studies 1 and 2. Cognitive vulnerability for depression interacted with a naturalistic stressor (an exam failure) to predict enduring elevations of depressive symptoms specifically, but not anxiety symptoms, at T3. This pattern was found regardless of whether composite depression and anxiety were used or the specific tripartite theory measures of anhedonic depression and anxious arousal. Both HT's and BT's cognitive vulnerability-stress interaction were found to predict depression when examined separately. Analyses that compared directly the uniqueness of HT or BT showed that neither cognitive vulnerability-stress interaction remained significant for predicting enduring changes in anhedonic depressive symptoms after controlling for the other model's vulnerability-stress component, whereas BT's Dysfunctional attitudes × Exam failure uniquely predicted composite depressive symptoms after controlling for HT's Cognitive vulnerability × Exam failure.

A strength of Study 3 is that the use of an academic midterm design enables precise assessments and dating of cognitive vulnerability, the stressor, and changes in depressive and anxious symptoms over time. In contrast to Studies 1 and 2 as well as most of the prospective vulnerability-stress studies employing 2 time point designs, Study 3 directly addressed the temporal precedence of a stressor, interacting with initial levels of cognitive vulnerability, predicting enduring elevations of depressive symptoms. Thus, Study 3 shows that HT's and BT's cognitive vulnerability-stress interaction predicts changes in depressive symptoms specifically because the timing of the assessment of vulnerability, stressor, and affective symptoms was precisely dated and assessed, and as a result, the known timing of stressor occurrence and changes in affective symptoms rules out the possibility (from Studies 1 and 2) that the stressors experienced over the prospective follow-up may have preceded the increase in emotional symptoms. This replication across studies enhances confidence in the cognitive vulnerability-stress hypotheses and their affective specificity.

GENERAL DISCUSSION

We tested the hypothesis that cognitive vulnerability for depression would interact with negative events to predict depression specifically compared with anxiety in three independent, prospective studies. Negative events were hypothesized to operate as a nonspecific risk factor for both anxiety and depression. These hypotheses were investigated prospectively using multiple affective symptom measures with

enhanced specificity and structured diagnostic interviews in a subsample of participants to assess for clinically significant anxiety and depressive disorder.

The results across all three prospective studies were consistent with these hypotheses. Cognitive vulnerability interacted with negative life events to predict prospective depressive symptoms and disorder specifically, but not anxiety symptoms or disorders. Negative events were nonspecifically associated with future anxiety and depression. These findings are consistent with hypotheses from cognitive vulnerability-stress theories of depression (Abramson, et al., 1989; Beck, 1987; Hankin & Abramson, 2001) that cognitive vulnerability interacting with negative events will be an etiological specific risk factor for depression and that negative life events will be a nonspecific risk factor for anxiety and depression.

The findings from these three studies add to a growing body of literature (e.g., see Abramson et al., 2002; Ingram et al., 1998, for reviews) that has examined prospectively the interaction of cognitive vulnerability with stressors and its association with depression. The pattern of results across studies shows that prospective elevations in depression, even when different measures of depression are used, are best predicted when individuals with heightened cognitive vulnerability experience stressors over time.

In addition to replicating the findings from previous studies, several features of the present report extend the prior research and advance the evidence for the cognitive vulnerability-stress theories of depression. First, we examined the affective specificity of the cognitive vulnerability-stress component in three prospective studies with different time intervals and designs. We used a 2 time-point prospective panel design in two separate studies, one over a short-term (5-weeks) and another over a long-term (2-years) interval, as well as a third academic midterm study with 3 time points over several weeks. Each of the studies and designs, on their own, has limitations, but the consistent, replicated pattern of results across the different studies and designs suggests a strong, robust set of findings. Second, we used multiple measures of anxiety and depression with improved affective specificity and discriminant validity. The results were maintained even after initial levels of both anxiety and depressive symptoms and initial levels of stressors were controlled. Third, we tested affective specificity with clinically significant anxiety and depressive disorders in addition to symptom measures. We discuss the importance of investigating the etiological factors at the level of symptoms and disorder later. Across the different time intervals, study designs, symptom measures of anxiety and depression, and severity level (symptoms to disorder), the same pattern of results was found. This enhances the confidence in a replicable, robust set of findings.

We also compared the etiological components of the two cognitive theories. First, in the analyses examining each theory on its own, the vulnerability-stress components from both HT and BT were equally effective in predicting depression. Second, when both cognitive vulnerability-stress components were included in the same analysis to compare HT and BT more directly, across the three studies, neither HT's nor BT's cognitive vulnerability-stress component uniquely predicted elevations in anhedonic depressive symptoms, and in the first two studies, neither cognitive theory's vulnerability-stress component remained as a significant predictor of composite depressive symptoms. Only in Study 3 did BT's Dysfunctional attitudes $\times$ Stress

interaction remain as a unique predictor of composite depressive symptoms, and even in this analysis, the magnitude of the effect size for BT was largely reduced after including HT's Cognitive vulnerability × Stress interaction. In sum, these results suggest that both theories' vulnerability-stress components largely overlapped in predicting depression. Still, as the present investigation is one of the first to compare directly the cognitive vulnerability-stress components from HT and BT in predicting affective symptom specificity (see also Lewinshohn et al., 2001 for vulnerability-stress comparisons in adolescents and Haeffel et al., 2003, for cognitive vulnerability comparisons in adults), further research examining the unique predictive power of each cognitive theory's components would be helpful.

The findings from these three studies help to clarify the co-occurrence of anxiety and depression. One approach to understanding the overlap of anxiety and depression is utilizing a structural theory, such as the tripartite theory of anxiety and depression, that explicitly models both the overlapping and the relatively specific aspects of depression and anxiety. In addition to the structural models that specify *what* is common and unique to anxiety and depression, another complementary approach to understanding affective overlap is focusing on etiological models that illustrate *how* and *why* these symptoms develop. In these studies, we tested the etiological specificity of HT's and BT's cognitive vulnerability-stress interaction to predict prospective elevations in depression using the more precise affective symptom measures based on the tripartite model, whereas most past studies have used less affectively specific anxiety and depression measures that primarily assessed broad negative affect. Our results suggest that the occurrence of stressors leads to elevations in negative affect (i.e., anxiety and depression). Of interest, the interaction of cognitive vulnerability with stress was a more etiologically specific predictor of later depression (i.e., both specific anhedonic depression and composite depressive symptoms). We did not test nor find an etiological factor specific to anxiety.

In all three of our studies, we used these more affectively specific measures based on the tripartite theory to improve discriminant validity and to provide a stronger test of cognitive vulnerability-stress models predicting depression specifically compared with anxiety. We conducted analyses using both the tripartite theory specific symptom factors and the composite symptoms because more recent structural models (e.g., Mineka et al., 1998) now view the tripartite theory's anxious arousal symptoms as most related to panic attack symptoms, rather than the full range of anxiety symptoms and disorders. The pattern of our results (i.e., cognitive vulnerability-stress predicting depression, but only stressors predicting anxiety) were the same for the composite measure of depression (general distress—depression, anhedonic depression, and BDI), the composite measure of anxiety (general distress—anxiety and anxious arousal), and for the specific tripartite model symptoms of anhedonic depression or anxious arousal.

In addition to more precise affective symptom measures, the current investigation included a structured diagnostic interview to assess for the occurrence of depressive and anxiety disorders over the 2 years in a subset of participants in Study 2. Examining depressive disorder is particularly important given previous critiques (e.g., Coyne & Gotlib, 1983) that the cognitive vulnerability theories are relevant to mild levels of depressive symptoms but not to clinically significant depression. Our

results show that the cognitive vulnerability-stress components from HT and BT are associated with the prospective occurrence of depressive disorder in addition to depressive symptoms. Thus, these results, combined with other research (Alloy et al., 2000; Haeffel et al., 2003; Lewinsohn et al., 2001), address directly the previous criticisms and show that the factors featured in the cognitive theories apply to clinically significant depressive disorder as well as symptoms.

Moreover, assessing both depressive symptoms and disorder helps inform the debate over the continuity of depression (Coyne, 1994; Flett, Vredenburg, & Krames, 1997). All of our etiological findings applied to both depressive and anxious symptoms and disorder. These results more consistently support the continuity hypothesis that the latent structure of depression as well as the risk factors and processes contributing to mild depression may not differ significantly from those leading to clinical depression (Hankin, Fraley, Lahey, & Waldman, in press; Lewinsohn, Solomon, Seeley, & Zeiss, 2000; Ruscio & Ruscio, 2000, 2002).

It is important to consider potential limitations from our three prospective studies. First, fewer participants were interviewed for clinically significant anxiety and depressive disorder compared with the number of participants who completed questionnaires assessing affective symptoms. This may have limited the power to detect significant effects for disorder. For example, neither dysfunctional attitudes nor cognitive style alone predicted depression after controlling for initial depression and stressor levels. However, significant results were obtained for predicting depressive disorder based on the cognitive vulnerability-stress component. These findings suggest that cognitive vulnerability in interaction with negative events more powerfully predicts depression than does cognitive vulnerability by itself. Also, the lack of power as an explanation for the nonsignificant findings of cognitive vulnerability-stress in predicting anxiety disorder seems unlikely because the cognitive vulnerability-stress component predicted occurrence of depressive disorder in this sample. Moreover, the other studies had large enough samples with sufficient power for analyses involving anxiety symptoms, but still only negative life events, and not the cognitive vulnerability-stress component, predicted anxiety symptoms. Last, other studies with larger sample sizes (e.g., Alloy et al., 2000; Haeffel et al., 2003; Lewinsohn et al., 2001) have found that cognitive vulnerability is associated with depressive, but not nondepressive, disorders. Still, replication of our findings with a larger sample of participants interviewed for anxiety and depressive disorders would be helpful.

Second, university undergraduates served as participants in all 3 studies. Some authors (e.g., Coyne, 1994) have criticized the use of college students in research examining emotional distress. However, others (e.g., Flett, Vredenburg, & Krames, 1997) have countered that using student samples is appropriate because there is reasonable continuity in depression between student and clinical samples. Further, late adolescence to young adulthood is a time when many individuals experience their first depressive disorder (Hankin et al., 1998). Indeed, the incidence rate of 18% clinical depression over the 2-years period in Study 2 from late adolescence to early adulthood is consistent with other research with young adults (Haeffel et al., 2003; Hankin et al., 1998). Thus, studying undergraduates in these studies seems justifiable.

Third, concerns arise with the timing and assessment of stressors in vulnerability and stress studies. Theoretical and empirical research (e.g., Monroe & Simons, 1991) highlights the possibility that a self-report measure of negative life events may be inadequate. As a result, our use of a self-report checklist of stressors in Studies 1 and 2 may not have been as powerful as using a contextual threat interview to assess events. However, addressing this potential concern, recent research (Lewinsohn, Rohde, & Gau, 2003) has directly compared self-report and interviewer-based measures of negative life events. Of interest, the majority of self-reported stressors were verified in the more comprehensive stressor interview, and the interview-identified stressors showed the same associations with depression as did the self-reported stressors (but see McQuaid, Monroe, Roberts, Kupfer, & Frank, 2000). Thus, this research suggests that interviewer-based and self-report measures of negative life events yield comparable results in the prediction of depression. Further, Study 3 used the academic midterm design in which a known, naturalistic stressor (midterm exam) was used as the stressor and the timing of changes in affective symptoms is known, and results from Study 3 were remarkably consistent with findings from Studies 1 and 2 that employed self-report checklists of events. Thus, Study 3 directly addresses the potential concern with the dating of stressors and symptoms and with the temporal precedence of stressors leading to elevations in depressive symptoms. In sum, our use of different vulnerability-stress designs (a prospective follow-up design with typically occurring, multiple stressors in Studies 1 and 2 and a midterm design with a specific, naturalistic stressor in Study 3) shows that the set of findings is not tied to a particular design or methodology, but rather, cognitive vulnerability interacting with stress as a specific predictor of depressive symptoms was replicated robustly across three studies with different time intervals and designs.

Fourth, the effect sizes for the significant findings may appear small to moderate at first glance. For example, after controlling for initial depression and stressors, the cognitive vulnerability-stress component explained a 2–4% increment in variance in depressive symptoms (small effect size; Cohen, 1988). However, there are two statistical considerations to keep in mind when interpreting these effect sizes. First, McClelland and Judd (1993) noted that "moderator effects are so difficult to detect that even those explaining as little as 1% of the total variance should be considered important," especially when using dimensional variables in prospective field research. Second, controlling for initial levels of depression, anxiety, and stressors provides a very conservative test of the cognitive vulnerability-stress theories component because any variance shared among cognitive vulnerability, negative life events, and affective symptoms is allocated to these initial levels of emotional distress and stressors.

In sum, we tested whether etiological risk factors from cognitive vulnerability-stress theories of depression predicted future depression, compared with anxiety, using more affectively specific symptoms measures, based on a structural model of anxiety and depression, and structured clinical interviews. Findings from three independent, prospective studies show that negative life events were a nonspecific risk factor for prospective elevations in depression and anxiety. Importantly, the interaction of cognitive vulnerability for depression with negative events served as a specific risk factor for future depression.

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