

Cognitive Vulnerability to Depression and Axis II Personality Dysfunction

Jeannette M. Smith · Louisa D. Grandin ·
Lauren B. Alloy · Lyn Y. Abramson

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Abstract The study of common vulnerabilities to depression and personality disturbance is important given that depression and Axis II personality disorders exhibit high rates of comorbidity and comorbid personality dysfunction is associated with longer duration of depressive episodes, increased risk of relapse, and poorer outcome. Further, it has been demonstrated that a relationship exists between cognitive vulnerabilities to depression, such as negative cognitive style and dysfunctional attitudes, and personality dysfunction. This study sought to further explore the relationship between cognitive vulnerability to depression and Axis II pathology by examining links between personality dysfunction, cognitive risk status, and rumination in a sample with no current Axis I disorders, while controlling for current and past depression. At high vs. low cognitive risk for depression, 349 undergraduates completed diagnostic interviews and self-report questionnaires during Phase I, Phase II, and Time 1 of the Cognitive Vulnerability to Depression (CVD) project. As expected, cognitive risk and rumination were related to overall Axis II pathology. Cognitive risk status was also significantly related to several personality disorder dimensions, including paranoid, schizotypal, histrionic, narcissistic, avoidant, dependent, and obsessive-compulsive. Rumination was uniquely related to the borderline and obsessive-compulsive dimensions. Our findings confirm and expand upon the relationship between cognitive vulnerabilities to depression and personality dysfunction.

Keywords Personality dysfunction · Cognitive vulnerability · Depression · Rumination · Comorbidity

J. M. Smith (✉) · L. D. Grandin · L. B. Alloy
Department of Psychology, Temple University, Weiss Hall, 6th Floor, 1701 North 13th Street,
Philadelphia, PA 19122, USA
e-mail: jsmith03@temple.edu

L. Y. Abramson
Department of Psychology, University of Wisconsin, Madison, WI, USA

Introduction

Comorbidity has become an important area in depression research. Of particular interest is the common overlap of depressive disorders and personality pathology. Studies have reported that between 35% and 78% of individuals selected for depression have a comorbid personality disorder (Ilardi & Craighead, 1999; Marton et al., 1989; Rothschild & Zimmerman, 2002). Conversely, 80% of a sample diagnosed with borderline personality disorder (BPD) had co-occurring major depression (Grapentine, Picariello, Nurcombe, Seirfer, & Scioli, 1990). In a review, Farmer and Nelson-Gray (1990) conclude that studies of inpatients with depression suggest a high frequency of comorbid Cluster B personality pathology, such as BPD, whereas studies of depressed outpatients show greater overlap with Cluster C personality pathology, such as dependent, avoidant and obsessive compulsive personality disorder (OCPD).

In addition to its common co-occurrence with depression, comorbid personality dysfunction imparts several deleterious effects on depressed patients (for a review, see Farmer & Nelson-Gray, 1990). For example, patients with both depression and Cluster C personality pathology recover more slowly from their depression than patients with depressive diagnoses alone (Vinamaki et al., 2002). Additionally, longer episodes of depression were reported in depressed outpatients with a comorbid personality disorder than in patients with depression alone (Rothschild & Zimmerman, 2002). Comorbid personality pathology has also been implicated in relapse of depression; expected remission duration decreases by 8% with each Axis II criterion met (Ilardi, Craighead, & Evans, 1997). These studies highlight the additional impediments to recovery for depressed patients with personality pathology.

The frequent co-occurrence of depression and personality dysfunction warrants examination of risk factors that may simultaneously affect susceptibility to depression and personality pathology. One such risk factor may be a depressogenic cognitive style; several leading theories of depression have posited that the presence of a characteristic negative cognitive style increases vulnerability to the onset and maintenance of depression. For example, according to Beck's (1967) theory, certain individuals hold dysfunctional attitudes concerning the need to be perfect and gain others' approval and these maladaptive attitudes impart risk for depression. Likewise, in the Hopelessness Theory of depression, Abramson, Metalsky, and Alloy (1989) suggest that individuals who make stable and global attributions when faced with negative life events, or believe that such events will increase the likelihood of subsequent negative consequences, or infer negative characteristics about themselves in response to negative events, will be at increased risk for depression. Alternatively, Nolen-Hoeksema (1991) suggested that individuals who exhibit a ruminative response style, consistently focusing on the meanings and consequences of their negative mood, are likely to experience more severe and prolonged depression. Each of these cognitive styles has been found to prospectively predict depression (Abramson et al., 2002; Alloy et al., 1999, 2006; Nolen-Hoeksema, 2000; Robinson & Alloy, 2003; Spasojevic & Alloy, 2001), but it may be that they are also related to Axis II dysfunction.

In line with this, researchers have begun to demonstrate a relationship between these depressogenic cognitive styles and personality pathology. For example, Ilardi and Craighead (1999) reported that depressed patients with greater personality pathology were more likely to endorse dysfunctional attitudes and generated more global, stable and internal attributions than those with less personality dysfunction. Similarly, depressed patients with comorbid personality disorders had higher scores on the Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978) than those with depression alone (Marton et al., 1989). Abela, Payne, and Moussaly (2003) compared levels of cognitive vulnerability in individuals with BPD and

depression, depression alone, and control participants with no Axis I or II diagnoses. Their comorbid participants exhibited higher levels of cognitive vulnerability to depression, as measured by attributional style, hopelessness, dysfunctional attitudes, and rumination, than their depressed only or control participants. Similarly, among depressed inpatients, those with personality disorders, particularly BPD, were more likely to exhibit cognitive vulnerability than other depressed patients (Rose, Abramson, Hodulik, Halberstadt, & Leff, 1994). Although these studies suggest a relationship between depressive cognitive styles and Axis II dysfunction, they all do so only in patient samples and do not examine the full spectrum of personality pathology.

Not only are patients with comorbid Axis II dysfunction and depression more likely to exhibit negative cognitive styles, individuals with Axis II pathology often display depressogenic cognitive styles in the absence of current depression. For example, participants with BPD had elevated dysfunctional attitudes compared to normal controls, regardless of current levels of depression (O’Leary et al., 1991). Similarly, Hill, Oei, and Hill (1989) reported equivalent dysfunctional attitudes between depressed patients and non-depressed patients with Axis II diagnoses, suggesting that individuals with personality pathology may exhibit dysfunctional attitudes similar to those of depressed patients. Therefore, if depressogenic cognitive styles are associated with personality dysfunction, we expect that individuals who are selected based on cognitive vulnerability to depression, but do not currently have any Axis I disorders, may exhibit higher levels of personality pathology than individuals who are at low cognitive risk for depression.

The current study sought to further examine the relationship between Axis II dysfunction and depressogenic cognitive styles. Participants in the Temple – Wisconsin Cognitive Vulnerability to Depression project (CVD; Alloy & Abramson, 1999) were assessed for cognitive risk status (based on dysfunctional attitudes and negative inferential styles), rumination, and personality dysfunction, controlling for current and past history of depression. We predicted that depressogenic cognitive styles, including rumination, would be related to Axis II pathology independent of depression. Specifically, we expected that depressogenic thinking would be related to those personality disorders that are most often comorbid with depression, namely, borderline and the Cluster C personality disorders.

In addition, it has been suggested that negative cognitive styles may be an inherent characteristic of certain personality dysfunctions.¹ For example, the Diagnostic and Statistical Manual’s (DSM) general criteria for diagnosing a personality disorder describe deviant cognitions, “i.e., ways of perceiving and interpreting self, other people and events,” (American Psychiatric Association, 2000, p. 689) as part of the diagnostic picture of personality dysfunction.

In this study, we sought to determine whether there was a relationship between negative inferential styles, dysfunctional attitudes, and rumination and personality dysfunction above and beyond the dysfunctional thinking styles that are characteristic of personality disorders. In other words, we aimed to clarify whether the cognitive vulnerability to depression observed in personality-disordered patients in prior studies was meaningfully distinct from the dysfunctional thinking styles that define personality pathology. In these ways, the current study expands on prior literature on personality and cognitive vulnerability to depression and examines common vulnerabilities to depression and personality pathology among individuals at high vs. low cognitive risk for depression. Additionally, this study is only the second study to examine the relationship between rumination and Axis II dysfunction and the first to examine the association between depressogenic cognitive styles and Axis II dysfunction in a high-risk sample with no current Axis I disorders.

¹ We thank Reviewer 1 who raised this conceptual issue.

Method

Participants

One hundred and seventy nine Temple University (TU) and 170 University of Wisconsin (UW) students who participated in the CVD project were included in this study. The purpose of the CVD project was to follow prospectively non-depressed students, who were at high and low cognitive risk for depression, in order to predict onsets and recurrences of depressive disorders. Participants at cognitive high risk (HR) for depression scored in the upper quartile (most negative) on both the Cognitive Style Questionnaire (CSQ; Alloy et al., 2000) composite for negative events and the Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978). Participants at cognitive low risk (LR) for depression scored in the lower quartile (most positive) on both the CSQ composite and the DAS. Participants who met the HR or LR criteria and exhibited no current Diagnostic and Statistical Manual of Mental Disorders (3rd ed., rev.; DSM-III-R; American Psychiatric Association, 1987) or Research Diagnostic Criteria (RDC; Spitzer, Endicott, & Robins, 1978) Axis I disorders at the outset of the study, as well as no serious medical condition that would preclude them from being in a longitudinal study, were included in the CVD Project. Participants who met DSM-III-R or RDC criteria for a past mood disorder were retained in the final samples if their symptoms had been in remission for at least 2 months (for more details, see Alloy & Abramson, 1999 and Alloy et al., 2000).

In the CVD Project, the CSQ and DAS were administered to 5,378 (2,438 at TU; 2,940 at UW) freshmen, of which 619 potential HR (261 at TU; 358 at UW) and 585 potential LR (234 at TU; 351 at UW) were identified. Utilizing an expanded version of the Schedule for Affective Disorders and Schizophrenia-Lifetime (Exp-SADS-L; Endicott & Spitzer, 1978) Interview, a random subset of these participants were interviewed and 209 eligible HR (114 at TU; 95 at UW) and 201 eligible LR (110 at TU; 97 at UW) participants were identified. Of these eligible participants, 17 HR and 13 LR participants refused to participate in the prospective phase of the study and another 19 HR and 18 LR participants were dropped prior to study entry (for any one of the three reasons: inability to locate participant; 5 or more missed appointments; or foreign student with relatively poor English speaking ability). The final CVD sample, or those participants who completed the Time 1 baseline including the Personality Disorder assessment, were included in the current study ($N = 349$; $HR = 173$; $LR = 176$). The ethnic composition of our sample was 79% Caucasian, 14% African American, 2% Hispanic, 4% Asian, and 2% other. The mean age of the participants upon entering the study was 19.96 years and 235 (67%) were women (for demographics and other characteristics as a function of cognitive risk status and site, see Table 1). Only participants at the TU site were also administered the RSQ at the Time 1 baseline visit. Thus, for these analyses, the sample consisted of 83 HR and 87 LR participants.

Measures

Cognitive risk

Participants' cognitive vulnerability to depression was assessed using the CSQ and the DAS. The CSQ is an expanded and modified version of the Attributional Style Questionnaire (ASQ; Seligman, Abramson, Semmel, & von Baeyer, 1979). The CSQ uses 24 hypothetical events (12 negative and 12 positive) to assess the degree to which an individual's attributional style is internal, global and stable. Additionally, the CSQ assesses the individual's inferred

Table 1 Final sample: Demographic characteristics

	Temple University Site		University of Wisconsin Site	
	High Risk	Low Risk	High Risk	Low Risk
BDI ^a	6.59 (5.71)	3.73 (3.88)	5.71 (5.35)	2.35 (2.36)
SADS-L ^b	0.76 (0.89)	0.34 (0.52)	0.81 (0.99)	0.34 (0.62)
Rumination ^c	13.98 (7.58)	7.35 (4.51)	***	***
PDE ^d	12.46 (13.21)	6.91 (8.76)	9.36 (8.43)	4.22 (4.75)
Age (yrs)	19.43 (1.42)	20.63 (2.79)	19.84 (0.41)	19.94 (1.20)
Gender	66% Female	67% Female	69% Female	67% Female
Ethnic Group	68% Caucasian	58% Caucasian	96% Caucasian	92% Caucasian

***Data was not collected for this cohort

^aBeck Depression Inventory (represents number of current depressive symptoms)

^bSchedule for Affective Disorders and Schizophrenia-Lifetime (represents the number of past depressive episodes)

^cThe rumination subscale of the Response Style Questionnaire

^dPersonality Disorder Examination (represents the sum of the dimensional scores)

consequences and implications for self-concept in response to each of the 24 events. The CVD project utilized only the CSQ negative events composite (stability, globality, consequences, self-implications) score (in addition to the DAS) to establish the HR and LR groups. The DAS was expanded from 40 to 64 items for the CVD project. The DAS measures the degree to which an individual's attitudes involving evaluation and performance are maladaptive and pessimistic, as well as beliefs about control and attributional style. The CSQ and DAS have shown excellent internal consistency, retest-reliability over 1 year, and predictive validity for episodes of depression in the CVD project (Alloy et al., 1999; 2000; 2006).

Ruminative response style

The Response Style Questionnaire (RSQ; Nolen-Hoeksema & Morrow, 1991; Nolen-Hoeksema, Morrow, & Fredrickson, 1993) is a 71-item self-report questionnaire designed to examine how people tend to respond to depressed mood. The four subscales of the RSQ, rumination, distraction, problem-solving, and dangerous activities, represent four potential coping strategies for depressed mood. Only the rumination subscale, consisting of 21 items, was included in the present analyses. These particular items focus on responses to depressive symptoms and their possible causes and consequences, for example, "think about how hard it is to concentrate" and "go away by yourself and think about why you feel this way." For this subscale, internal consistency was high ($\alpha = 0.89$; Nolen-Hoeksema & Morrow, 1991) and test-retest reliability was moderate ($r = .47$ over 1 year; Just & Alloy, 1997) to high ($r = .80$ over 5 months; Nolen-Hoeksema, Parker, & Larson, 1994). The RSQ Rumination scale has also been found to predict prospective episodes of depression (Just & Alloy, 1997; Nolen-Hoeksema, 2000; Spasojevic & Alloy, 2001).

Personality dysfunction

The Personality Disorder Examination (PDE; Loranger, 1988) is a semi-structured diagnostic interview designed to assess the various DSM-III-R Axis II personality disorders. The PDE consists of 126 items which each assess part or all of a DSM-III-R personality disorder criterion and are rated on a three-point scale: 0 = absent or normal, 1 = exaggerated or accentuated, and 2 = meets criteria or pathological. Items have one or several primary

questions as well as follow-up questions. After these questions have been exhausted, a trained clinical interviewer may ask additional questions until he or she is able to score the item. The PDE has good reliability (e.g., all of the PDE items had interrater reliability coefficients greater than .71) and validity (Loranger et al., 1991; Loranger, Susman, Oldham, & Russakoff, 1987). Further, one study examined the stability of the personality disorder features in 250 undergraduate students in a 4-year prospective study with the PDE and Millon Clinical Multiaxial Inventory II (MCMI-II; Millon, 1997; Lenzenweger, 1999). Lenzenweger (1999) found that participants displayed relatively high levels of individual difference stability over the 4-year period as assessed by the PDE and MCMI-II. This study suggests that personality disorder features are stable in college student samples.

The PDE can be interpreted in several ways. It generates diagnoses for each Axis II personality disorder, as well as a continuous dimensional score for each disorder. Clinically significant items can also be summed as a measure of overall Axis II pathology. PDE interviewers were blind to participants' cognitive risk status and rumination scores. The interrater reliability for the dimensional scores based on 40 interviews independently rated by two interviewers in the CVD Project are as follows: paranoid ($\alpha = .73$), schizoid ($\alpha = .69$), schizotypal ($\alpha = .46$), antisocial ($\alpha = .88$), borderline ($\alpha = .76$), histrionic ($\alpha = .80$), narcissistic ($\alpha = .72$), avoidant ($\alpha = .80$), dependent ($\alpha = .73$), and obsessive-compulsive ($\alpha = .83$). In this study, we examined the diagnosis, PDE item sum, and dimensional score data.

Depressive symptoms

The Beck Depression Inventory (BDI; Beck, Rush, Shaw, & Emery, 1979) is a 21-item self-report inventory that assesses the presence and severity of current cognitive, motivational, affective, and somatic symptoms of depression. This measure has been validated for student samples (Bumberry, Oliver, & McClure, 1978; Hammen, 1980). In addition, in a non-clinical population, the internal reliability is good ($\alpha = 0.81\text{--}0.86$) and the test–retest reliability ranges from .48 to .86 (Beck, Steer, & Garbin, 1988; Groth-Marnat, 1990). The BDI was used as a measure of current depressive symptoms in this study.

Past history of depression

An expanded version of the Schedule for Affective Disorders and Schizophrenia-Lifetime (SADS-L; Endicott & Spitzer, 1978) diagnostic interview assessed the number of past depressive episodes as well as history of other disorders. For the CVD project, the SADS-L was modified and expanded in the following ways: (1) we added questions to aid in the assignment of DSM-III-R diagnoses as well as RDC diagnoses; (2) we expanded and improved upon the anxiety disorders section by incorporating aspects of the Anxiety Disorders Interview Schedule (DiNardo et al., 1985); (3) we added questions that assessed participants' percentage of waking hours of each day as well as the number of days that they felt depressed; and (4) we grouped questions according to their relevance to a particular diagnosis and questions assessing past episodes of a given disorder immediately followed the assessment of a current episode of that disorder. These modifications were made in consultation with Jean Endicott's group at the New York State Psychiatric Institute (the developers of the SADS-L).

Diagnostic interviewers were blind to participants' cognitive risk group as well as all other vulnerability measures (e.g., RSQ). Expanded-SADS-L interviewers were extensively trained in a program modeled after other ideal training programs (Amenson & Lewinsohn, 1981; Gibbon, McDonald-Scott, & Endicott, 1981). This program consisted of approximately 200 h of didactic instruction, role-playing, reading (e.g., case vignettes), watching videotaped

interviews, extensive practice conducting live interviews, and regular tests that must be passed. Interviewers received extensive feedback throughout their training and during the duration of the project. We also calibrated our diagnoses in several ways (see Alloy & Abramson, 1999). The interrater reliability for all depression diagnoses on the Exp-SADS-L interviews was good ($\kappa > 0.90$).

Procedure

Participants were selected based on a two-phase screening process. Phase I consisted of participants completing the CSQ and DAS to identify individuals who were hypothesized to be at high versus low cognitive risk for depression. A random subset of participants from Phase I who scored in the highest quartile (most negative), or the HR group, and the lowest quartile (most positive), or the LR group, were invited in for Phase II. Phase II consisted of administering the current episode and lifetime portions of the Exp-SADS-L as well as completing several other self-report questionnaires, such as the BDI. The time between Phase I and Phase II varied, but was approximately 2 months. Participants who met the Phase II screening criteria (see “Participants”) and agreed to participate in the prospective phase of the study were invited to do the Time I interview. At this baseline visit, several questionnaires and interviews were administered, including the PDE and RSQ. Again, only participants in the TU cohort were administered the RSQ. Time I occurred within 1 month of Phase II. To summarize, the measures specific to this study were completed at the following visits: the CSQ and DAS were completed in Phase I, the Exp-SADS-L and the BDI at Phase II, and the PDE and RSQ at Time I.

Results²

Preliminary Analyses: Group Differences

The TU and UW cohorts did not differ on age, sex, current depressive symptoms, or past depressive episodes, however the TU cohort had significantly more ethnic minority participants than the UW cohort ($\chi^2 = 56.27$, $p < 0.01$; see Table 1). This difference is likely explained by TU being situated in the ethnically diverse city of Philadelphia. The HR and LR groups did not differ on sex or ethnicity, however the LR group was significantly older than the HR group ($t(1, 333) = -3.38$, $p < .01$). As expected, the HR group reported more current depressive symptoms as well as a higher number of past depressive episodes than the LR group ($t(1, 340) = 6.32$, $p < .01$; $t(1, 347) = 5.75$, $p < .01$, respectively; see Table 1).

In order to control for these differences, sex, site, age, and ethnicity were controlled in all analyses. Also, because we were interested in the relationship of cognitive style and personality dysfunction independent of depression, current depressive symptoms and past history of depressive episodes were entered as covariates in all analyses. Finally, all analyses concerning

² The degrees of freedom varied across analyses due to missing data. The total sample size is 349 for analyses not including the RSQ, and thus, without any missing data our degrees of freedom for t -tests would be 348. For analyses including the RSQ, our degrees of freedom for t -tests without any missing data would be 169. Analyses that use data from several questionnaires had smaller degrees of freedom because participants were excluded if they had data from anyone questionnaire missing. For example, in examining the relationship of rumination and personality while controlling for cognitive risk, our degrees of freedom were reduced to 118, as participants were excluded from the analysis if they were missing data from the RSQ, PDE, DAS, or CSQ.

rumination included cognitive risk status as a covariate in order to isolate the effects of rumination independent of other cognitive vulnerabilities.

Tests of hypotheses

Several of the variables were significantly correlated ($p < .01$; see Table 2). For example, cognitive risk and rumination were positively correlated with each other and with current depression, past history of depression, and the PDE sum score. Current and past depressions were significantly correlated with each other, as well as with the PDE sum score.

To examine the relationship between cognitive risk and overall Axis II pathology, a logistic regression analysis was conducted on the presence versus the absence of any Axis II diagnosis, controlling for current and past depression, with cognitive risk as the predictor. As predicted, cognitive risk status was significantly related to diagnosable personality pathology, ($\beta = 5.19$, $Wald = 4.055$, $p = .04$). Only 1.8% of participants in the LR group had a diagnosable personality disorder, compared to 6.6% of participants in the HR group. Contrary to our hypothesis, rumination was not related to personality disorder diagnosis ($\beta = 1.03$, $Wald = 0.28$, $p = .60$); however, because only the TU sample completed the RSQ, this may be due to diminished power.

To more sensitively test for relationships with overall Axis II pathology, linear regression analyses, controlling for current and past depression, confirmed that cognitive risk status was significantly related to the PDE sum score ($t(320) = 3.78$, $p < .001$), such that HR individuals exhibited more overall Axis II pathology than LR individuals. Likewise, rumination was significantly related to greater overall personality dysfunction ($t(118) = 2.56$, $p = .03$), controlling for cognitive risk.

We also hypothesized that depressogenic thinking would be related to specific personality dimensions, particularly the ones most often comorbid with depression: borderline, and Cluster C disorders. To test these hypotheses, a multivariate analysis of covariance (MANCOVA) was conducted on the paranoid, schizoid, schizotypal, antisocial, borderline, histrionic, narcissistic, avoidant, dependent, and obsessive-compulsive dimensional scores of the PDE, controlling for current and past depression (see Table 3). Cognitive risk status was significantly related to several of the PDE dimensions, including paranoid, schizotypal, histrionic, narcissistic, avoidant, dependent, and obsessive-compulsive. Rumination was also uniquely related to the borderline and obsessive-compulsive dimensions, controlling for cognitive risk status.

Table 2 Correlation matrix for main variables

	RISK ^a	RUM ^b	BDI ^c	DEP ^d	PDE ^e
RISK ^a	1	.474 ^f	.324 ^f	.295 ^f	.276 ^f
RUM ^b	.474 ^f	1	.432 ^f	.260 ^f	.351 ^f
BDI ^c	.324 ^f	.432 ^f	1	.251 ^f	.292 ^f
DEP ^d	.295 ^f	.260 ^f	.251 ^f	1	.216 ^f
PDE ^e	.276 ^f	.351 ^f	.292 ^f	.216 ^f	1

^aCognitive risk status

^brumination subscale score of the Response Style Questionnaire

^cBeck Depression Inventory score, a measure of current depression

^dpast history of depression as measured by the Schedule for Affective Disorders and Schizophrenia, Lifetime version

^esum of all dimensional scores on the Personality Disorders Examination

^fcorrelation is significant at the 0.01 level (2-tailed)

Table 3 Results of MANCOVA analysis

	Cognitive Risk		Rumination	
	<i>F</i> -Statistic	<i>p</i> -value	<i>F</i> -Statistic	<i>p</i> -value
<i>PDE Dimension</i>				
Cluster A				
Paranoid	6.074	.014 ^a	.026	.872
Schizoid	3.277	.071	.386	.536
Schizotypal	5.799	.017 ^a	3.495	.064
Cluster B				
Antisocial	.028	.867	2.793	.097
Borderline	1.372	.242	12.604	.001 ^a
Histrionic	8.951	.003 ^a	2.810	.096
Narcissistic	4.716	.031 ^a	.000	.999
Cluster C				
Avoidant	15.459	.001 ^a	1.702	.195
Dependent	9.644	.002 ^a	1.802	.182
Obsessive-compulsive	26.600	.001 ^a	9.783	.002 ^a

^aSignificant at the $p < .05$ level

In order to address the conceptual concern that the cognitive vulnerabilities to depression assessed by the CSQ, DAS and RSQ are actually just part of characterological patterns of thinking inherent in personality dysfunction, we re-ran the analyses controlling for overlap between the definition of cognitive vulnerability as defined by Beck (1967) and Abramson and colleagues (1989) and the various cognitive criteria for personality disorder. To do this, the PDE items were assessed for conceptual similarity with depressogenic thinking, and the items that overlapped were removed from the dimensional scores. Eleven items from the paranoid, avoidant, and dependent dimensions were removed. The analyses were then re-run with these new dimensional scores, and cognitive risk remained significantly related to all three revised dimensions (paranoid: $F(1,292) = 18.22$, $p < .001$; avoidant: $F(1,292) = 23.04$, $p < .001$; dependent: $F(1,292) = 12.16$, $p = .001$). This suggests that the cognitive vulnerabilities to depression reported by participants are meaningfully different from the deviant cognitions that characterize paranoid, avoidant, and dependent personality dysfunction.

Similarly, two items from the obsessive-compulsive dimensional score that were conceptually similar to rumination, items that referred to a “preoccupation” with certain cognitive content, were removed. No items from the borderline dimension were determined to be overlapping with rumination. After removing the preoccupation items from the obsessive-compulsive dimensional score, rumination was no longer significantly related to obsessive-compulsive personality dysfunction ($F(1,105) = 2.01$, $p = .16$). This suggests that the relationship between rumination and obsessive-compulsive personality dysfunction may be due to conceptual overlap in the definition of rumination and the preoccupational thinking that defines obsessive-compulsive personality pathology.

Given that rumination was significantly related to overall personality dysfunction, as well as the BPD dimension of the PDE, while controlling for cognitive risk status, it is clear that rumination is associated with personality pathology above and beyond negative cognitive style and/or dysfunctional attitudes. This suggests that future research should examine rumination as a common risk factor for depression and personality dysfunction.

Discussion

The central aim of this study was to determine whether cognitive vulnerability to depression is associated with Axis II personality dysfunction and, thus, may help explain the common comorbidity of depression and Axis II pathology. Other researchers have found that individuals with borderline personality disorder endorse levels of cognitive distortions that are comparable to individuals with depression, regardless of current depression (Hill et al., 1989; O’Leary et al., 1991). This study provides further evidence that the cognitive styles typically associated with and predictive of future depression may be related to personality dysfunction, but are also distinct from the deviant cognitive styles that help define personality pathology. For example, cognitive risk status, based on negative inferential style and dysfunctional attitudes, was significantly related to the presence of Axis II diagnoses, the sum of the dimensional scores on the PDE, and several personality disorder dimensions (e.g., paranoid, schizotypal, histrionic, narcissistic, dependent, avoidant and obsessive-compulsive). Moreover, these relationships were significant independent of current or past history of depression and despite removing PDE items that conceptually overlapped with the cognitive vulnerabilities to depression. This suggests that maladaptive ways of thinking may not be uniquely related to depression, but may also be related to other types of pathology that overlap with depression, specifically personality dysfunction. In addition, cognitive vulnerability is associated with personality dysfunction regardless of current depression; thus, it may be that maladaptive patterns of thinking are not state-dependent in individuals with Axis II dysfunction.

It is noteworthy that the relationship between cognitive risk status and borderline personality disorder was not significant, which is discordant with other findings in the literature (Abela et al., 2003; O’Leary et al., 1991; Rose et al., 1994). Given the high-risk design, current and past depression were controlled in these analyses to isolate the effects of depressogenic thinking independent of depression history. However, current depression accounted for a large portion of the variance in the relationship between cognitive risk status and borderline personality pathology. When current depression is not controlled for, a statistically significant relationship between borderline personality pathology and cognitive risk is found ($F(1, 324) = 8.38, p = .004$), in line with previous findings. Thus, it is clear that controlling for current depression impacted the relationship between BPD and cognitive risk. Given that other investigators did not control for current depression (Abela et al., 2003; Marton et al., 1989; O’Leary et al., 1991; Rose et al., 1994), it will be important for future studies to examine the impact of current depressive symptoms on the association between BPD and depressogenic thinking.

This was the first study to examine the relationship between rumination and the full range of personality dysfunction. Specifically, after controlling for cognitive risk status, as well as current and past history of depression, rumination was significantly related to overall Axis II pathology, as measured by the PDE sum, as well as to borderline and obsessive-compulsive dimensions of the PDE. These findings coincide with Abela et al.’s (2003) results, which suggest that rumination is indeed uniquely related to BPD and that it may be even more pernicious in individuals with both BPD and depression. In addition, our finding that rumination was significantly associated with OCPD dysfunction, but only when the “preoccupation” items were included in the OCPD score, may be explained by such individuals’ preoccupation with, or repetitive thought process about, certain aspects of their environment. Obsessive behavior is similar to rumination, and thus, it may be that ruminative thinking processes are similar to the preoccupational thinking that is characteristic of OCPD. Further

research is needed to address whether the ruminative behavior reported in individuals with obsessive-compulsive pathology also contains the negative content that is typically associated with depressogenic thinking.

This was the first study to examine the association of depressogenic cognitive styles, including rumination, with a broad spectrum of Axis II personality dysfunction. In addition, no studies have examined these relationships in a high-risk sample with no other current Axis I pathology. Thus, this study presents a broader view of the overlap between cognitive vulnerabilities to depression and personality dysfunction. In addition, the overall relationship between personality dysfunction and depressogenic cognitive styles has important clinical implications. For example, the presence of maladaptive attitudes and inferences imparts vulnerability to the onset of depression (Alloy et al., 1999; 2006), and the presence of these negative cognitive styles in individuals with personality dysfunction may put these individuals at even increased risk for onset of depression. BPD and OCPD patients may be at especially high risk because they are also plagued by a characteristically ruminative style of responding to dysphoric mood in addition to the other cognitive vulnerabilities. Finally, the association of cognitive vulnerability with personality dysfunction independent of depression suggests that therapeutic techniques aimed at remediating negative cognitive styles may be useful in the treatment of Axis II disorders as well (Beck et al., 1990).

Despite its utility, several limitations of the present study must be acknowledged. For example, the sample was originally selected based on the presence or absence of cognitive vulnerabilities to depression and therefore, may show levels of current and past depression that would not be representative of a randomly chosen sample. In addition, our sample consisted of extremes on cognitive vulnerability, which may reduce the generalizability of our results. Future studies should replicate these findings with a more representative sample. Moreover, whereas our participants exhibited symptoms of personality dysfunction, only a minority met DSM-III-R criteria for a personality disorder. Thus, a study of individuals with clinical levels of personality pathology would clarify the role of cognitive style in more severely disordered Axis II populations. Finally, because our results are cross-sectional and correlational, no causal conclusions can be drawn. Thus, we cannot ascertain whether cognitive styles develop from certain personality characteristics, or if these styles serve as a vulnerability for personality dysfunction.

Conclusion

In sum, these results suggest that certain vulnerabilities to depression, such as maladaptive attitudes, negative inferential style, and rumination, may also be related to Axis II personality dysfunction. In addition, these cognitive vulnerabilities may be mood state independent in individuals with Axis II pathology. Given that individuals with personality dysfunction exhibit depressogenic thinking, future research should address whether interventions that target dysfunctional cognitive styles are effective for individuals with depression, personality dysfunction, or both. Given the clinical implications of these findings, future studies should examine further the genesis of these maladaptive ways of thinking that impart vulnerability to depression and personality dysfunction (see Alloy et al., 2001; Gibb et al., 2001).

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