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BRIEF REPORT

Relationships Between Depressive Rumination, Anger Rumination, and Borderline Personality Features

Ruth A. Baer and Shannon E. Sauer
University of Kentucky

We examined relationships between depressive rumination, anger rumination, and features of borderline personality disorder in a sample of 93 students with a wide range of borderline symptoms. All completed self-report measures of borderline features; trait-level negative affect; depressive and anger rumination; and current symptoms of depression, anxiety, and stress. Depressive and anger rumination were strongly associated with borderline features after controlling for comorbid symptoms of depression, anxiety, and stress. Both types of rumination showed significant incremental validity over trait-level sadness, anger, and general negative affect in predicting borderline features. Relationships with borderline features were stronger for anger rumination than for depressive rumination. Relationships between trait-level negative affect and borderline features were substantially reduced when anger rumination was included in regression models, suggesting the need for longitudinal analyses of mediation. Findings suggest that severity of borderline symptoms is influenced by ruminative thinking in response to negative affect, especially anger.

Keywords: borderline personality features, depressive rumination, anger rumination

Rumination is a maladaptive form of negatively valenced, self-focused, repetitive thinking about symptoms of distress and their causes, consequences, and implications. A large body of research has focused specifically on depressive rumination, in which individuals repetitively dwell on their symptoms when feeling sad, blue, or depressed. Depressive rumination has been shown to intensify and maintain negative mood, impair concentration, memory, and problem solving, reduce motivation for instrumental behavior, and predict the onset of future depressive episodes. Although depressive rumination is associated with neuroticism, it predicts variance in depression after controlling for neuroticism. Recent studies also have suggested that depressive rumination contributes to the etiology and maintenance of anxiety, posttraumatic stress, disordered eating, substance abuse, and emotional reactivity to stressful events (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008; Watkins, 2008).

Because of its strong associations with negative affect, emotional reactivity, and dysregulated behavior, several authors have suggested that depressive rumination may also be common in borderline personality disorder (BPD). Abela, Payne, and Moussaly (2003) found that patients with BPD and major depressive disorder (MDD) had higher levels of depressive rumination than those with only MDD. Smith, Grandin, Alloy, and Abramson (2006) and Selby, Anestis, Bender, and Joiner (2009) found that depressive rumination was significantly associated with BPD symptoms after controlling for current depression. However, none of these studies controlled for neuroticism, which is known to be high in BPD (Morey & Zanarini, 2000).

Although neuroticism increases the risk of psychopathology, it is possible to be high in neuroticism without having a mental disorder (Costa & McCrae, 1992). Recent work has suggested that how people respond to their negative affect is at least as important to their mental health as the frequency or intensity with which

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Ruth A. Baer and Shannon E. Sauer, Department of Psychology, University of Kentucky.

Correspondence concerning this article should be addressed to Ruth A. Baer, Department of Psychology, 115 Kastle Hall, University of Kentucky, Lexington, KY 40506-0044. E-mail: rbaer@email.uky.edu

negative affect arises (Hayes, Wilson, Gifford, Follette & Strosahl, 1996). For example, many people ruminate about sadness because they believe (mistakenly) that rumination is helpful in developing insight and solving problems that lead to depression (Papageorgieu & Wells, 2001). However, rumination typically exacerbates sadness and increases susceptibility to depressive episodes. A recently discussed mediational model suggests that high levels of neuroticism lead to depressive rumination as people attempt to reduce their negative affect by thinking repetitively about its causes and meanings. Rumination in turn increases the risk of psychopathology. Several studies of depression have supported this model. Nolan, Roberts, and Gotlib (1998) found that rumination at Time 1 mediated the relationship between neuroticism at Time 1 and depressive symptoms 8 to 10 weeks later. Similar evidence has been found in cross-sectional studies (Kuyken, Watkins, Holden, & Cook, 2006; Muris, Roelofs, Rassin, Franken, & Mayer, 2005). These findings support the hypothesis that depressive rumination is a mechanism through which neuroticism increases the risk of depressive episodes.

Selby and Joiner (2009) described a somewhat similar model for BPD. They proposed that BPD emerges from a vicious cycle in which rumination on negative affect leads to intensification of emotions, which leads to increased rumination. Dysregulated behavior, such as self-harm, substance abuse, or binge eating, then serves to distract attention from the negative affect and ruminative thoughts. Selby et al. (2009) found that severity of BPD symptoms was significantly correlated with rumination and that rumination mediated the relationship between BPD symptoms and dysregulated behavior, such as self-harm and binge eating. However, they did not examine the role of neuroticism.

Not all rumination occurs in response to sadness. Anger rumination is defined as repetitive thinking about anger experiences and the causes and consequences of angry feelings (Sukhodolsky, Golub, & Cromwell, 2001). Although anger rumination has been studied much less than depressive rumination, it has been shown to have maladaptive outcomes, including increased feelings of anger (Rusting & Nolen-Hoeksema, 1998), physiological arousal (Ray, Wilhelm, & Gross, 2008), and aggressive behavior (Bushman, Bonacci, Pederson, Vasquesz, & Miller, 2005). Selby, Anestis, and Joiner (2008) found significant relationships between anger rumination and BPD symptoms, bulimic symptoms, anxiety, depression, and alcohol use.

It is not surprising that both depressive and anger rumination are related to BPD. Depression is often comorbid with BPD, and problematic anger is a diagnostic criterion (American Psychiatric Association, 2000). However, differential relationships between these two types of rumination and BPD symptoms have not been investigated. Therefore, in the present study we examined the relative contributions of depressive and anger rumination to predicting variance in BPD features. We expanded on previous findings in several ways. First, because symptoms of depression, anxiety, and stress are common in BPD and are associated with rumination, we examined whether either type of rumination is associated with BPD features after controlling for these symptoms. Second, we examined the incremental validity of both types of rumination over neuroticism in predicting BPD features. Significant findings would suggest that severity of BPD symptoms is influenced both by the occurrence of negative affect and by ruminative thinking about it. Finally, we tested whether the relationship between neuroticism and BPD features is reduced when both neuroticism and rumination are included in regression models. Significant findings would suggest the need for longitudinal studies of rumination as a mediator of this relationship.

Method

Participants

Participants were 93 students in an introductory psychology course. Trull (1995, 2001) showed that BPD features are common in undergraduate samples. Students with raw scores over 37 ($T = 70$) on the Borderline Features Scale of the Personality Assessment Inventory (PAI–BOR; Morey, 1991) showed clinically significant characteristics of BPD and maladjustment in several domains (interpersonal and academic problems, Axis I symptoms, poor coping skills) similar to levels seen in clinical populations (Trull, 1995). These difficulties persisted over a 2-year period (Trull, Useda,

Conforti, & Doan, 1997). Trull (1995) also noted that clinical samples may represent only the upper range of BPD severity. Studies of cognitive and emotional functioning associated with BPD will have more power to detect effects if they include a wide range of BPD characteristics.

Participants signed up using an online registration system open to all students in introductory psychology, who are required to participate in ongoing studies for course credit. At the beginning of each semester, all students in this course complete a packet of screening measures for ongoing studies. Those who meet various criteria can then be invited to participate in particular studies. We included a short version of the PAI–BOR in the screening packet. Due to high demand for screening time by departmental researchers, length of screening instruments is limited. Therefore, our screener was a 10-item subset of PAI–BOR items that included two or three items from each of its four subscales. We made our study available to the entire participant pool through the online system. To obtain adequate representation of all levels of BPD features in our sample, we also sent emails to students in the pool who had obtained high or low scores on the PAI–BOR screener (scores that when prorated would be equivalent to *T* scores over 70 or below 50, respectively) inviting them to sign up for our study, if they had not already done so. Of the 93 students who completed the study (which included the full PAI–BOR scale), 27 (30%) had *T* scores of 70 or higher on the PAI–BOR, according to norms provided by Morey (1991). Mean age was 19 years ($SD = 1.76$, range = 18 to 33), 84% were women, and 87% were White. The distribution was not excessively skewed or kurtotic on the PAI–BOR scale (skewness = .25, $SE = .25$; kurtosis = −1.00, $SE = 0.50$).

Measures

The PAI–BOR (Morey, 1991) includes 24 items rated on a 4-point Likert scale (0 = false, not at all true; 4 = very true). It has four subscales, each assessing a general feature of BPD pathology: affective instability, identity problems, negative relationships, and self-harm (the self-harm scale also includes items about impulsivity). The PAI–BOR has high internal consistency and convergent correlations with other measures of BPD features (Trull, 1995). The four subscales are consistent with factor analytic research on the fundamental components of BPD pathology (Skodol et al., 2002). Morey (1991) reported that although BPD patients scored above the clinical threshold (raw score > 37) on the PAI-BOR, other diagnostic groups did not, including those with mood or anxiety disorders, schizophrenia, schizoaffective disorder, antisocial personality disorder, or substance abuse. Trull (1995) found that PAI–BOR scores predicted academic and interpersonal functioning in a student sample after controlling for Axis I pathology and neuroticism. Jacobo, Blais, Baity, and Harley (2007) found that PAI–BOR score was significantly correlated with presence of a BPD diagnosis as determined by structured interview, but not with diagnoses of narcissistic, histrionic, or antisocial personality disorders. These findings clearly suggest that high scores on the PAI–BOR are likely to reflect BPD-specific pathology rather than general distress or other disorders. In the present sample, internal consistencies for the PAI–BOR were .90 for the total score, .86 for affective instability, .70 for identity problems, .79 for negative relationships, and .70 for self-harm.

Depressive rumination was measured using the Ruminative Responses Scale (RRS) from the Response Styles Questionnaire (RSQ; Nolen-Hoeksema & Morrow, 1991), which assesses the tendency to engage in rumination when feeling sad, blue, or depressed. Scores are related to the onset and severity of depressive symptoms (Nolen-Hoeksema et al., 2008). However, several authors have argued that this relationship may be inflated by the presence of items that confound specific symptoms of depression with the tendency to ruminate about them (Treynor, Gonzalez, & Nolen-Hoeksema, 2000). For example, a high score on, "think about how alone you feel" might simply reflect feeling alone, rather than ruminating about feeling alone. Low scores on these items are similarly difficult to interpret. For example, a low score for "think about how hard it is to concentrate" could reflect either not having concentration problems or not ruminating about concentration problems (Segerstrom, Tsao, Alden, & Craske, 2000). Several authors (Treynor et al., 2000; Segerstrom et al., 2000) have addressed this problem by deleting items that include content related to specific depressive symptoms (poor concentration, feeling

alone, fatigued, passive, unmotivated) that a depressed person may not have. The remaining items focus on repetitive thinking about depression or sadness in general. This allows the total score to reflect the general tendency to ruminate when feeling sad or depressed, without confounding by the presence or absence of specific symptoms that not all depressed people experience. We followed the example of Segerstrom et al. (2000) by excluding all items referring to symptoms other than feeling sad or depressed. The remaining 10-item scale had an alpha coefficient of .90 in our sample. Example items include "isolate yourself and think about the reasons why you feel sad" and "go someplace alone to think about your feelings."

Anger rumination was measured using the Anger Rumination Scale (ARS; Sukhodolsky et al., 2001), which has 19 items assessing the tendency to focus attention on angry moods in a ruminative way (e.g., "when something makes me angry, I turn this matter over and over again in my mind"). The scale showed good internal consistency (α = .93) and test–retest reliability (r = .77) in the development sample. Sukhodolsky et al. (2001) found moderate correlations between ARS scores and anger-related constructs (anger expression, anger-out, anger-in), and a factor analysis showed that items representing these anger constructs loaded on a separate factor from anger rumination items, which all loaded on a single factor. These findings support the discriminant validity of anger rumination as distinct from anger. Selby et al. (2008) found an alpha of .91 and significant correlations with measures of psychological symptoms and dysregulated behavior. In the present sample, alpha was .95.

Current psychological symptoms were measured using the short form of the Depression Anxiety Stress Scales (DASS; Lovibond & Lovibond, 1995). This 21-item instrument assesses negative affect and bodily symptoms over the last week and provides scores for depression, anxiety, and stress as well as a total score. Although the DASS has no anger scale, stress items include feeling touchy, irritable, and intolerant of interruptions. We used only the total score. The DASS has alphas of .84 and higher (Lovibond & Lovibond, 1995) and strong correlations with other measures of anxiety and depression. In the present sample, alpha was .92.

The general tendency to experience negative affect was measured with the Positive and Negative Affect Schedule–Expanded Form (PANAS–X; Watson, & Clark, 1994), a 60-item self-report measure in which mood adjectives (e.g., "excited" and "irritable") are rated on a 5-point Likert scale (1 = very slightly or not at all; 5 = extremely). Multiple-time frames can be used with this instrument. To assess trait-level negative affectivity, participants were asked to rate how they generally feel. We used only the general negative affect, sadness, and hostility scales, which have shown good internal consistencies (alphas of .82 or higher) in previous samples and significant correlations in the expected directions with measures of related constructs. In the present sample, alphas were .85, .91, and .80 for negative affect, sadness, and hostility, respectively.

Procedure

All participants reported individually or in small groups to a room on the university campus. The study procedures were explained and the informed consent document was completed. Participants then completed a battery of questionnaires requiring 60 to 90 min (some for this project, others for projects not described here). Order of questionnaires within the battery was randomized. Participants then were thanked, provided with a written debriefing, and awarded research participation credit for their introductory psychology course.

Results

Intercorrelations between study measures were positive and significant and are shown in Table 1. All hypotheses were tested in the com-

Table 1
Intercorrelations Between Study Measures

Measure	DASS total	PANAS–NA	RRS	ARS
PAI-BOR total	.60**	.66**	.54**	.76**
DASS total	—	.62**	.51**	.54**
PANAS–NA	—	—	.48**	.56**
RRS	—	—	—	.57**

Note. DASS = Depression Anxiety Stress scales; PANAS–NA = Positive and Negative Affect Schedule–Negative Affect; RRS = Ruminative Responses Scale; ARS = Anger Rumination Scale; PAI–BOR = Personality Assessment Inventory, Borderline Features scale.
** $p < .01$.

bined sample ($N = 93$). Although BPD is more often diagnosed in females, in the present study the proportion of females was not significantly different among those with T scores above 70 (89% women) and below 70 (82% women) on the PAI–BOR total score, $\chi^2 = .71$, $p = .40$. The correlation between gender and PAI–BOR total score was only marginally significant ($r = .19$, $p = .06$). Therefore, the following analyses did not control for gender. The first hypothesis was that both types of rumination would be significantly correlated with BPD features when controlling for current symptoms of depression, anxiety, and stress. Partial correlations were computed between both of the rumination measures and PAI–BOR scores, controlling for DASS total score. To examine whether observed relationships are consistent across the range of BPD features, these correlations were computed for each of the PAI–BOR subscales as well as the total score. Findings are shown in Table 2. Nearly all correlations were significant, showing that BPD features are significantly related to both depressive and anger rumination and that this relationship is not attributable to comorbid symptoms of depression, anxiety, and stress. Correlations for anger rumination were consistently larger than for depressive rumination. T tests for the significance of the difference between dependent correlations (Howell, 1982) showed that these differences were significant for the PAI–BOR total score and the affective instability and negative relationships subscales (ps $< .05$). They were marginally significant for the identity problems and self-harm subscales (ps $= .09$ and .055, respectively).

Next we conducted a series of four hierarchical regression analyses to examine whether either type of rumination contributes to incremental variance in BPD features beyond that accounted for by trait-level negative affect (see Table 3). The first two analyses examined whether each specific type of rumination accounted for incremental variance in BPD features after controlling for the corresponding type of trait-level negative affect (sadness or anger). In Analysis 1, depressive rumination accounted for small but statistically significant variance in BPD features after controlling for trait-level sadness (R^2 increased from .36 to .42). In Analysis 2, anger rumination accounted for a statistically significant and substantially larger amount of variance in BPD features after accounting for trait-level anger (R^2 increased from .35 to .61).

Analyses 3 and 4 examined the incremental validities of both depressive and anger rumination after accounting for general negative affect. In both cases, general negative affect (PANAS–NA) entered at Step 1 and accounted for 43% of the variance in BPD features. In Analysis 3, depressive rumination entered next and was followed by anger rumination. Both forms of rumination showed significant incremental validity over trait negative affect in accounting for BPD features. In Analysis 4, the order of entry of the two forms of rumination was reversed. In this case, anger rumination showed significant incremental validity in predicting BPD features, whereas depressive rumination did not.

These findings suggest that anger rumination may have stronger relationships with BPD features than depressive rumination. To determine whether this pattern is consistent across the range of BPD features, the regression analyses just described were repeated for each of the subscales of the PAI–BOR separately. Results are shown in Table 4. In each case, the findings

Table 2
Partial Correlations Between Depressive and Anger Rumination and Borderline Personality Features, Controlling for Current Symptoms of Depression, Anxiety, and Stress in Total Sample

PAI–BOR scale	Depressive rumination	Anger rumination	t	p
Affective Instability	.32**	.54***	2.24	.03
Identity Problems	.26*	.44***	1.72	.09
Negative Relationships	.31**	.56***	2.57	.02
Self-Harm/Impulsivity	.17	.38***	1.95	.055
Total score	.35***	.64***	3.20	.002

Note. $N = 93$. PAI–BOR = Personality Assessment Inventory–Borderline Features scale.
* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 3
Hierarchical Regression Analyses Examining the Incremental Validity of Rumination Over Trait-Level Negative Affect in Predicting Borderline Personality Features in Total Sample

Analysis	DV	Step	Predictor(s)	Change in R^2	Total R^2
1	PAI–BOR total	1	PANAS–Sadness	.36*	.36
		2	Depressive rumination	.06*	.42
2	PAI–BOR total	1	PANAS–Anger	.35*	.35
		2	Anger rumination	.26*	.61
3	PAI–BOR total	1	PANAS–Negative Affect	.43*	.43
		2	Depressive rumination	.06*	.49
		3	Anger rumination	.16*	.65
4	PAI–BOR total	1	PANAS–Negative Affect	.43*	.43
		2	Anger rumination	.22*	.65
		3	Depressive rumination	.00	.65

Note. $N = 93$. DV = dependent variable; PAI–BOR = Personality Assessment Inventory Borderline Features Scale; PANAS = Positive and Negative Affect Scale.
* $p < .05$.

were the same as for the PAI–BOR total score. After accounting for trait-level negative affect, anger rumination had incremental validity over depressive rumination in predicting each PAI–BOR subscale, but depressive rumination had no incremental validity over anger rumination.

Previous studies have suggested that rumination may mediate the relationship between neurot-

Table 4
Hierarchical Regression Analyses Examining the Incremental Validity of Depressive and Anger Rumination Over Trait-Level Negative Affect in Predicting the PAI–BOR Subscales

Analysis	DV	Step	Predictor(s)	Change in R^2	Total R^2
1	Affective instability	1	PANAS–Negative Affect	.39*	.39
		2	Depressive rumination	.05*	.44
		3	Anger rumination	.11*	.55
2	Affective instability	1	PANAS–Negative Affect	.39*	.39
		2	Anger rumination	.15*	.54
		3	Depressive rumination	.01	.55
3	Identity problems	1	PANAS–Negative Affect	.29*	.29
		2	Depressive rumination	.07*	.36
		3	Anger rumination	.11*	.47
4	Identity problems	1	PANAS–Negative Affect	.29*	.29
		2	Anger rumination	.17*	.46
		3	Depressive rumination	.01	.47
5	Negative relationships	1	PANAS–Negative Affect	.30*	.30
		2	Depressive rumination	.06*	.36
		3	Anger rumination	.15*	.51
6	Negative relationships	1	PANAS–Negative Affect	.30*	.30
		2	Anger rumination	.20*	.50
		3	Depressive rumination	.01	.51
7	Self-harm/impulsivity	1	PANAS–Negative Affect	.19*	.19
		2	Depressive rumination	.01	.20
		3	Anger rumination	.08*	.28
8	Self-harm/impulsivity	1	PANAS–Negative Affect	.19*	.19
		2	Anger rumination	.09*	.28
		3	Depressive rumination	.00	.28

Note. $N = 93$. DV = dependent variable; PAI–BOR = Personality Assessment Inventory Borderline Features Scale; PANAS = Positive and Negative Affect Scale.
* $p < .05$.

icism and psychopathology. Although our data are cross-sectional, we explored this issue in a preliminary way by examining whether the beta coefficient for PANAS–NA was reduced when anger rumination was included in the regression model (with BPD features as the dependent variable) and whether the indirect pathway through anger rumination was statistically significant. The beta coefficient for PANAS–NA as a single predictor of PAI–BOR was .66 ($p < .001$). When anger rumination was added as a second predictor, the beta coefficient for PANAS–NA was reduced to .34 ($p < .001$). (Depressive rumination was not included because it showed no incremental validity over anger rumination in the previous analyses.) The Sobel z test (1982) showed that the indirect pathway through anger rumination was significant ($z = 4.91$, $p < .001$). We repeated this analysis using the PANAS–Anger scale as the independent variable. Results were similar. The beta coefficient for PANAS–Anger predicting PAI–BOR was .60. When anger rumination was added as a second predictor, the beta coefficient for PANAS–Anger was reduced to .25. The indirect path through anger rumination again was significant ($z = 4.93$, $p < .001$). These findings suggest that the relationship between trait negative affect (and anger in particular) and BPD features may be at least partially accounted for by anger rumination.

Discussion

The present study replicated previous findings showing that BPD features are strongly associated with both depressive and anger rumination. Partial correlations revealed that these relationships are not attributable to comorbid symptoms of depression, anxiety, or stress. Rumination was shown to account for variance in BPD features that is not accounted for by the trait-level tendency to experience negative affect. This is important because it suggests that severity of BPD symptoms is influenced both by the occurrence of negative affect and by ruminative thinking about it. Associations with BPD features were stronger for anger rumination than for depressive rumination. Overall, findings suggest the need for longitudinal analyses to determine whether rumination is a risk factor (Kraemer, 2003) for the development of BPD and whether it mediates the relationship between trait-level negative affect and BPD features.

The present study measured only two types of rumination. Recent studies described other types that may be related to BPD. For example, Robinson and Alloy (2003) defined stress-reactive rumination as repetitively dwelling on negative inferences ("it was all my fault" or "things like this always happen to me") following stressful life events. Stress-reactive rumination predicts future depressive episodes (Robinson & Alloy, 2003) but has not been studied in BPD. Potential differences between specific types of rumination also should be investigated. For example, perceived reasons for engaging in anger rumination or beliefs about its effects may differ from those that have been documented for depressive rumination.

The present findings may have implications for the treatment of BPD. Empirically supported treatments for BPD, such as dialectical behavior therapy (DBT; Linehan, 1993) and psychodynamic therapies (Bateman & Fonagy, 2008) may implicitly address rumination through mindfulness training or reflection on mental states (Selby & Joiner, 2009). However, other treatments are more explicit in teaching participants about the nature and consequences of rumination, how to recognize when they are ruminating, and strategies for responding to rumination. These treatments include rumination-focused cognitive–behavioral therapy (Watkins et al., 2007), behavioral activation (Dimidjian et al., 2006), metacognitive therapy (Wells, 2000) and mindfulness-based cognitive therapy (Segal, Williams, & Teasdale, 2002). They have promising support for their efficacy with anxiety and depression but have not been studied in BPD. Future research should examine whether explicit targeting of rumination is helpful in treating BPD.

Several limitations must be considered when interpreting these findings. Although instruments with good psychometric properties were used, self-report methods may be subject to biases. The current sample had limited gender and ethnic diversity and was comprised entirely of students, who may not represent the upper range of symptom severity. Trull (1995, 2001) showed that levels of distress and impairment are clinically significant in student samples with high scores on the PAI–BOR. However, it is important to replicate the current findings in a

more diverse clinical sample with BPD diagnoses identified through structured interviews. The present study did not measure variables that may influence the development of BPD, such as a history of trauma or abuse. Finally, because the data were cross-sectional, conclusions cannot be drawn about the direction of effects. Previous work suggests that neuroticism and rumination are risk factors for psychopathology and that rumination may mediate the effects of neuroticism. Although the present findings are consistent with this model, they do not establish a temporal sequence in which neuroticism precedes the development of rumination, which in turn precedes the onset of BPD features, and therefore cannot provide convincing support of this model in BPD (Kraemer, 2003).

Despite these limitations, the current study contributes to the small literature on rumination in BPD by examining relationships with trait-level negative affect and by suggesting the potentially important role of anger rumination in particular. Findings add to a growing body of literature suggesting that cognitive processing styles, which have been studied extensively in Axis I disorders, are also important in BPD (Smith et al., 2006). Future research should include more diverse samples and more objective methods for assessing specific types of rumination and BPD symptoms. Longitudinal studies of rumination and BPD symptoms, as well as laboratory studies of the effects of rumination on behavioral measures, will also be very useful in clarifying the influence of rumination on severity of BPD.

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