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Neuroticism and Ruminative Response Style as Predictors of Change in Depressive Symptomatology

Susan A. Nolan[1] John E. Roberts,[2] and Ian H. Gotlib[3,4]

Several investigations have demonstrated that neuroticism and ruminative response style are associated with increased risk for depression. The current study examined the effects of neuroticism and ruminative response style on changes in depressive symptoms over an 8- to 10-week interval. Analyses indicated that the effects of neuroticism and ruminative response style were moderated by initial level of depressive symptomatology. Specifically, neuroticism and ruminative response style predicted changes in depressive symptoms more strongly in individuals who were initially higher in levels of depression than they did in those with lower initial levels of depressive symptoms. These data were consistent with a path model in which ruminative response style mediated the effect of neuroticism on depression.

KEY WORDS: rumination; neuroticism; depression; response styles; depressive symptoms.

INTRODUCTION

A number of studies have implicated neuroticism in vulnerability to depression (cf. Clark, Watson, & Mineka, 1994). In particular, not only do currently depressed individuals report elevated levels of neuroticism (e.g., Saklofske, Kelly, & Janzen, 1995), but previously depressed individuals do as well (e.g., Roberts & Gotlib, 1997). These findings suggest that neuroticism is a trait characteristic of depression that persists beyond remission of the depressive episode. Further, neuroticism has also been found to predict depression in prospective investigations. For example, in a sample of depressed inpatients, elevated neuroticism scores were associated both with the duration of depression and with poor overall outcome over a period of 18 years (Duggan, Lee, & Murray, 1990). Similarly, Boyce, Parker, Barnett, Cooney, and Smith (1991) found that high levels of neuroticism were associated with a three-fold increase in the risk for the development of depressive episodes over a 6-month

[1]Northwestern University, Evanston, Illinois 60208.
[2]State University of New York at Buffalo, Buffalo, New York 14260.
[3]Stanford University, Stanford, California 94305.
[4]Address all correspondence concerning this article to Ian H. Gotlib, Department of Psychology, Bldg. 420, Jordan Hall, Stanford University, Stanford, California 94305.

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period. Although it is not clear precisely how a global personality dimension such as neuroticism might increase risk for depression, studies suggest that neuroticism can lead to negative biases in attention (e.g., Derryberry & Reed, 1994) and memory (see Martin, 1985, for a review), as well as to a cognitive and behavioral style of a ruminative focus on depressive symptoms (Roberts, Gilboa, & Gotlib, 1998). In turn, each of these processes might contribute to the onset and maintenance of depressive symptoms.

Nolen-Hoeksema's (1987, 1991) model of vulnerability to depression posits that a ruminative style of responding to dysphoric mood contributes to the severity and persistence of depression. In particular, Nolen-Hoeksema suggested that an attentional focus on depressive symptomatology, as well as on the possible antecedents and consequences of depression, leads to longer and more severe depressive episodes. Relatively consistent support has been obtained for this hypothesis. For example, in a study in which participants tracked their moods and responses to their moods for 30 days, Nolen-Hoeksema, Morrow, and Frederickson (1993) found that increased use of ruminative responses led to longer durations of depressed mood. Similarly, Nolen-Hoeksema and Morrow (1991) found that the presence of ruminative responses in the 10 days following a natural disaster predicted high levels of depressive symptoms seven weeks later. Finally, in a laboratory experiment, Morrow and Nolen-Hoeksema (1990) had participants either distract or ruminate following a negative mood induction. Participants who actively distracted themselves showed the greatest improvement in mood, whereas participants who passively ruminated showed the least improvement (see also Nolen-Hoeksema & Morrow, 1993). Nolen-Hoeksema (1991) hypothesized that rumination influences depressive mood by interfering with attention and concentration, by enhancing the recall of negative events, and by increasing the likelihood of using depressogenic explanations for negative life events.

Although naturalistic prospective studies are important in testing the hypothesis that neuroticism and rumination act as causal agents in depression, third variables, such as a history of previous episodes of depression, can render the results of these investigations difficult to interpret. In particular, a number of studies have demonstrated that a history of depressive episodes is associated not only with subsequent depressive symptoms (cf. Belsher & Costello, 1988; Gotlib & Hammen, 1992), but also with elevated levels of neuroticism (see Clark et al., 1994, for a review) and rumination (Roberts et al., 1998). Therefore, it is possible that high levels of rumination or neuroticism are due to prior symptoms of depression. Indeed, the concept of neuroticism is ambiguous, and may simply reflect the past or current experience of strong negative affect. Thus, it is difficult to determine whether the prospective associations that have been documented between neuroticism and rumination on the one hand, and the onset or the duration of depression on the other, are direct, or alternatively, are due to the adverse effects of previous depression on neuroticism, rumination, and subsequent depressive symptoms. In the present study, therefore, we examined whether neuroticism and rumination would predict subsequent depressive symptoms after controlling for previous depression.

It is also important to note that earlier studies have found that psychosocial risk factors may be differentially associated with changes in depressive symptoms as a

function of whether individuals are initially asymptomatic or mildly depressed (Barnett & Gotlib, 1988; Roberts & Monroe, 1992). Such findings suggest that different processes might be involved in the onset versus the maintenance of depression (Depue & Monroe, 1986). Therefore, in addition to past depression, naturalistic prospective studies also need to examine the role of initial level of depressive symptomatology in mediating the relations between risk factors and subsequent depression.

The present study was conducted to examine the roles of neuroticism and ruminative response style in the onset and maintenance of depressive symptoms over an 8- to 10-week prospective interval. We hypothesized that initial levels of neuroticism and rumination would be associated prospectively with depressive symptomatology, such that higher levels of these variables would predict more severe depressive symptoms at follow-up. In addition, we also tested a path model in which ruminative response style was proposed to mediate the relation between neuroticism and change in depressive symptomatology. We noted earlier that neuroticism and rumination have both been associated with increases in depressive symptomatology. In a previous investigation in our laboratory, we (Roberts et al., 1998) speculated that individuals with high levels of neuroticism may have a tendency to engage in rumination in response to depression. More specifically, we hypothesized that rumination may mediate the effects of neuroticism on depression. In testing this path model in the present study, we statistically controlled for severity of worst lifetime episode of depressive symptomatology in all analyses to facilitate interpretation of the obtained results.

METHOD

Participants

Participants were 135 undergraduates (67 female) at Northwestern University who participated in this study in exchange for course credit. Participants were selected on the basis of a quartile split on the Ruminative Responses Scale of the Response Style Questionnaire (see below), which was completed during group testing sessions. Sixty-seven participants had rumination scores of 35 or less and 68 participants had scores of 48 or greater.

Measures

Depression

The Inventory to Diagnose Depression (IDD; Zimmerman, Coryell, Corenthal, & Wilson, 1986) is a 22-item self-report inventory that assesses the presence and severity of depressive symptoms. Although this instrument was designed to classify subjects categorically in terms of diagnostic criteria for major depression, when the items are summed it also yields an index of severity of depressive symptomatology. Treated as a continuous measure, the IDD correlates highly with both the Beck Depression Inventory (r = .87) and the Hamilton Rating Scale for Depression (r = .80; Zimmerman et al., 1986). In contrast to other commonly used self-report

instruments, such as the Beck Depression Inventory and the Center for Epidemiologic Studies Depression Scale, the IDD is a relatively pure measure of depressive symptomatology as defined by the Diagnostic and Statistical Manual of Mental Disorders (4th ed.) (DSM-IV; American Psychiatric Association, 1994).

The lifetime version of the IDD, the IDD-L (Zimmerman & Coryell, 1987), is similar to the IDD but focuses on the period of time during which the participant felt most depressed. Zimmerman and Coryell administered the IDD-L to a nonpatient sample and found it to have high internal consistency and split-half reliability.

Rumination

The Ruminative Response Scale (RRS) of the Response Style Questionnaire (Nolen-Hoeksema & Morrow, 1991) was used to assess how the subjects tended to respond to their own symptoms of depressed mood. The RRS consists of 21 items that assess responses to depressed mood that are focused on the self (e.g., "Think 'why do I always react this way?'"), on symptoms (e.g., "Think about how hard it is to concentrate"), or on possible consequences and causes of moods (e.g., "Think 'I won't be able to do my job/work because I feel so badly'"). The RRS also includes a number of behavioral responses to depressive mood (e.g., "Listen to sad music" and "Write down what you are thinking about and analyze it"). The RRS is scored by summing each subject's ratings for each of the 21 items.

Neuroticism

Neuroticism was assessed using the neuroticism subscale of the Eysenck Personality Inventory (EPI; Eysenck & Eysenck, 1964). This subscale consists of 24 items, such as "Are your feelings rather easily hurt?" and "Do you worry about awful things that might happen?" A total neuroticism score is computed by summing subjects' responses to the 24 items on the subscale. The EPI is a widely used, well-validated measure (Eysenck & Eysenck, 1964).

Procedure

Participants were assessed in a group testing session (Session 1), during which initial levels of depression, rumination, neuroticism, and past history of depression were evaluated using the measures described above. Eight to ten weeks later ($M = 9$ weeks; Session 2), participants returned to the laboratory and completed the IDD once again.

RESULTS

Descriptive Statistics and Preliminary Analyses

Mean IDD scores were 9.78 at Session 1 and 9.89 at Session 2. These scores were highly correlated, $r = .74$, $p < .001$, and were not significantly different, $t(133) < 1$. As

Table I. Gender Differences in Depression, Rumination, and Neuroticism

Variable	Cronbach's alpha	Males	Females	*t*-Statistic	*p*-Value
Session 1 IDD	.90	8.71	10.87	1.47	n.s.
Session 2 IDD	.91	8.29	11.51	2.08	< .05
Lifetime IDD	.91	22.46	27.05	1.77	n.s.
Rumination	.92	41.22	46.82	2.02	< .05
Neuroticism	.83	10.89	12.58	1.92	< .06

[a]IDD = Inventory to Diagnose Depression; n.s. = $p > .05$.

Table II. Intercorrelations Among Measures of Depression, Rumination, and Neuroticism[a]

Scale	1	2	3	4	5
1. Session 1 IDD		.74	.67	.53	6.9
2. Session 2 IDD			.57	.57	.63
3. Lifetime IDD				.61	.58
4. Rumination					.57
5. Neuroticism					

[a]$N = 135$; all correlations are significant at $p < .001$. IDD = Inventory to Diagnose Depression.

can be seen in Table I, compared with males, females reported greater depressive symptomatology at Session 2, greater rumination at Session 1, and marginally higher levels of neuroticism at Session 1. As can be seen in Table II, rumination and neuroticism at Session 1 were both significantly correlated positively with depressive symptoms at Session 1 and at Session 2, and with severity of worst lifetime depressive episode.

Prospective Analyses

To examine the prediction that depressive symptomatology at Session 2 would be predicted by initial levels of neuroticism and rumination, we conducted two regression analyses.[5] In each of these analyses, depressive symptoms at Session 2 served as the dependent variable. In the first analysis (neuroticism), depressive symptoms at Session 1, severity of worst lifetime depressive episode, neuroticism at Session 1, and the interaction of neuroticism and Session 1 depression were entered as independent variables. This analysis yielded a significant main effect of neuroticism, beta = .30, $t(130) = 3.61$, $p < .001$, such that higher levels of neuroticism at Session 1 predicted higher levels of depressive symptoms at Session 2. There also was a significant interaction of neuroticism and Session 1 depression, beta = .24, $t(130) = 3.23$, $p < .01$. Both this main effect and interaction were significant regardless of whether severity of worst lifetime depressive episode was included in the regression. The form of this interaction was examined by plotting data points according to the regression equation. The mean depression score at Session 1 and the scores 1 standard deviation above and below the mean were entered to produce three equations. Three regression lines were then calculated for varying levels of

[5]We conducted multiple-regression analyses to analyze our data even though we used a quartile split on rumination to select our subjects. One of the assumptions of regression procedures is that the *residuals* are normally distributed, which they were in this study; the *observations* need not be normally distributed to use multiple regression analysis (see Glass & Hopkins, 1984, p. 141, for a more complete discussion of this issue).

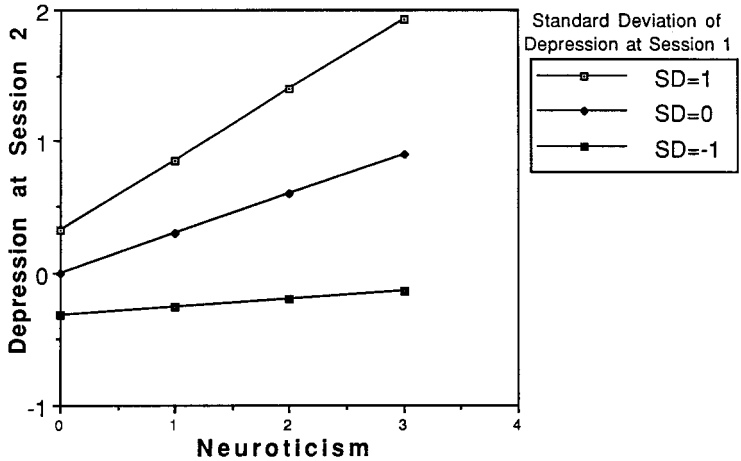

Fig. 1. Levels of depression at Session 2 as a function of neuroticism for three levels of depression at Session 1.

neuroticism. The graph derived from these calculations, presented in Fig. 1, indicates that neuroticism had a greater effect on depression at Session 2 as level of depression at Session 1 increased.

In the second regression analysis (rumination), depressive symptoms at Session 2 again served as the dependent variable, and depressive symptoms at Session 1, severity of worst lifetime depressive episode, rumination at Session 1, and the interaction of rumination and Session 1 depression were entered as independent variables. This analysis yielded a significant main effect of rumination, beta = .24, $t(130) = 3.43$, $p < .001$, indicating that higher levels of rumination at Session 1 predicted higher levels of depressive symptoms at Session 2. The interaction of rumination and Session 1 depression was also significant, beta = .17, $t(130) = 2.64$, $p < .01$. Again, this main effect and interaction were significant regardless of the whether severity of worst lifetime depressive episode was included in the analysis. As was the case with the first regression analysis, the form of this interaction was examined by plotting data points according to the regression equation. The mean depression score at Session 1 and the scores 1 standard deviation above and below the mean were entered to produce three equations. Three regression lines were then calculated for varying levels of rumination. Presented in Fig. 2, this graph indicates that rumination had a greater effect on depression at Session 2 as level of depression at Session 1 increased.[6,7]

[6]We also conducted all of the regression analyses, as well as the path analyses discussed below, with gender as an additional factor. In no case did gender change the relations among neuroticism, rumination, and depression, nor did gender contribute significantly to the path analyses. We also conducted an analysis of covariance (ANCOVA) examining the interaction of rumination and gender on Session 2 depression, covarying depression at Session 1. If gender were, in fact, a moderator of rumination, this interaction would be significant; it was not, $F(1,130) < 1$. It appears, therefore, that, once an individual ruminates, the effects are the same, regardless of gender. To facilitate presentation of the results, we did not include gender in these analyses.

[7]We also conducted this analysis treating rumination as a categorical variable. As was the case in the "continuous variable" analysis, the main effect of rumination was significant here as well, beta = .14, $t(132) = 2.19$, $p < .05$.

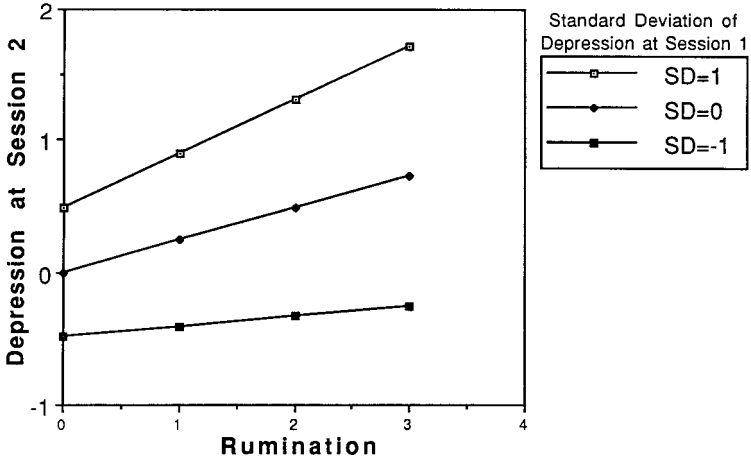

Fig. 2. Levels of depression at Session 2 as a function of rumination for three levels of depression at Session 1.

Path Model

We predicted a path model in which the relation between neuroticism and changes in depressive symptomatology was mediated by rumination (cf. Roberts et al., 1995). This model was tested with two regression analyses. The first analysis regressed rumination on Session 1 neuroticism, worst lifetime depressive symptoms, and Session 1 depressive symptoms. This equation was significant, with both neuroticism, beta = .29, $t(131) = 3.1$, $p < .002$, and worst lifetime depressive symptoms, beta = .39, $t(131) = 4.31$, $p < .001$, predicting rumination; Session 1 depressive symptoms did not predict rumination, beta = .08, $t(131) < 1$.[8] The second analysis regressed Session 2 depression on Session 1 rumination, Session 1 neuroticism, worst lifetime depressive symptoms, and Session 1 depressive symptoms. This equation was also significant, with both rumination, beta = .19, $t(130) = 2.56$, $p < .02$, and Session 1 depressive symptoms, beta = .52, $t(130) = 5.93$, $p < .001$, directly predicting Session 2 depressive symptoms. Neither neuroticism [beta = .15, $t(130) = 1.85$, $p > .05$] nor worst lifetime depressive symptoms [beta = .03, $t(130) < 1$] were significant direct predictors of Session 2 depressive symptoms. Thus, our path model, depicted in Fig. 3, demonstrated that, consistent with our prediction, neuroticism and severity of worst lifetime depression affected depression at Session

[8]We also conducted this regression analysis in two other ways. First, we conducted the analysis treating rumination as a categorical variable. The results of this logistic regression were virtually identical to those of the continuous variable analysis, with both neuroticism (Wald = 5.36, $p < .02$) and worst lifetime depressive symptoms (Wald = 9.39, $p < .002$), but not Session 1 depressive symptoms (Wald < 1), significantly predicting rumination (goodness of fit = 119.87, $p < .001$). Second, we conducted the analysis using the full range of rumination data, rather than data only from subjects in the top and bottom quartiles. The results of this analysis of neuroticism predicting rumination were even stronger than the quartile analysis (beta = .56, $p < .01$). Thus, eliminating the middle quartiles does not appear to cause any artifactual associations among these variables. For the sake of consistency throughout this paper, we present analyses using subjects only from the top and bottom quartiles on the measure of rumination.

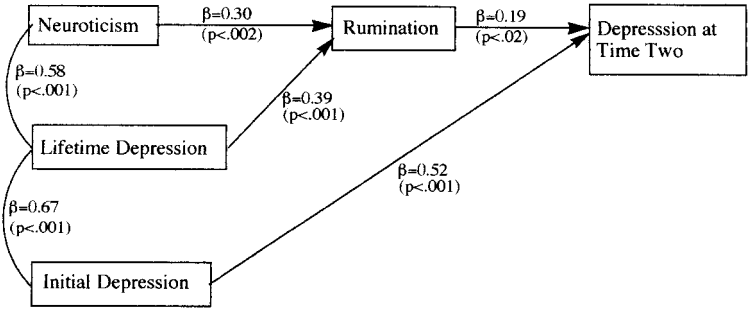

Fig. 3. Path model (only statistically significant paths are included).

2 indirectly through rumination. In contrast, rumination and depression at Session 1 both exerted direct effects on depression at Session 2.

Finally, we conducted an additional regression analysis to test an alternative, unpredicted, path model in which neuroticism mediates the relation between rumination and Session 2 depression. We regressed neuroticism on rumination, worst lifetime depression, and initial depressive symptoms. This equation was significant, with both rumination, beta = .24, $t(131) = 3.10$, $p < .01$, and initial depression, beta = .51, $t(131) = 6.15$, $p < .001$, predicting neuroticism. Contrary to the first model, lifetime depression did not significantly predict rumination, $p > .05$. As we described above, the regression of Session 2 depression on rumination, neuroticism, worst lifetime depression, and initial depressive symptoms was also significant, with rumination and initial depression, but not neuroticism or lifetime depression, directly predicting depressive symptoms at Session 2. There is no direct path, therefore, between neuroticism and depressive symptoms at Session 2, indicating that neuroticism does not mediate the relation between rumination and subsequent depression. Thus, our hypothesized model, in which rumination mediates the effects of neuroticism on subsequent depression, appears to be a more accurate description of relations among these variables.

DISCUSSION

This prospective study was conducted to examine the ability of neuroticism and ruminative response style to predict changes in depressive symptoms over an 8- to 10-week interval. As hypothesized, levels of neuroticism and rumination at Session 1 were associated prospectively with depressive symptomatology at Session 2, such that initially elevated levels of these variables led to higher levels of depressive symptoms eight to ten weeks later. Significant interactions of neuroticism and rumination with initial level of depression indicated that both these constructs are stronger predictors of subsequent depressive symptoms in individuals who are already mildly symptomatic than they are in initially asymptomatic persons. Moreover, these effects continued to be statistically significant after controlling for severity of worst lifetime depressive symptomatology. This pattern of results indicates

that, although a history of depression is associated with elevated levels of neuroticism and rumination, it is these latter constructs, and not history of depression, that lead to subsequent elevated depressive symptoms. These findings provide further support for the conceptualization of neuroticism and ruminative response style as vulnerability factors for depression.

Importantly, these data were also consistent with a hypothesized path model in which ruminative response style serves to mediate the effects of neuroticism and severity of previous depression on change in depressive symptomatology. Both neuroticism and history of previous depression were found to predict future depression only indirectly through elevated levels of rumination. In contrast, initial depressive symptomatology and ruminative response style appear to exert direct effects on subsequent depression. Importantly, the data also indicate that neuroticism does not similarly meditate the relation between rumination and depression.

The present findings are consistent with the formulation that neuroticism does not affect mood directly, but instead affects mood through cognitions, specifically through ruminative response style. This central role of cognition in affecting mood is consistent with cognitive theories of depression. In general, these theories maintain that negative schemata lead to cognitive biases, such as negative information processing and dysfunctional attitudes, which in turn contribute to the etiology and exacerbation of depressive symptoms (e.g., Beck, 1976, 1987; Gotlib & MacLeod, 1997). Nolen-Hoeksema (1991) distinguished her rumination theory of depression from other cognitive theories, contending that whereas cognitive theories generally focus on *content*, such as negative automatic thoughts and dysfunctional attitudes, rumination is a *style*, a pattern of behaviors and thoughts in which content is superfluous. However, Nolen-Hoeksema (1993) also stated that a ruminative style may be entwined with dysfunctional attitudes and cognitions, leading depressed individuals into the vicious cycle that maintains their depression. In this cycle, negative cognitions lead to more depressive symptomatology, which leads to more negative information processing and dysfunctional attitudes, and so on. If neuroticism does affect depression through cognitions, rumination may be the pathway.

This area of research may also provide clues to the identification of factors underlying the gender difference in the prevalence of depression. Epidemiologic studies have consistently found that the prevalence of depression is twice as high in women as it is in men. Numerous theories, focusing on artifactual, hormonal, and psychosocial variables, have been formulated in attempts to explain this difference (cf. Weissman & Klerman, 1977). Despite considerable research, however, the reasons for the elevated prevalence of depression among women remain unclear. Investigations assessing rumination have demonstrated that, when confronted with depressive thoughts, women are more likely to ruminate, whereas men are more likely to distract themselves (e.g., Butler & Nolen-Hoeksema, 1994). Rumination style, therefore, may be an important factor in accounting for the gender difference in depression.

It is important to note that, to date, studies have not found that the effects of rumination or distraction are moderated by gender (Nolen-Hoeksema, 1990). Thus, although women may be more likely to ruminate and men to distract, once these processes are undertaken the consequences are not affected by gender. Con-

sistent with Nolen-Hoeksema's (1991) theory, females in the present study exhibited higher levels of rumination than did males. Interestingly, females also demonstrated higher levels of neuroticism than did males. If rumination is linked with neuroticism, and levels of both rumination and neuroticism are generally higher in women, these two variables may both play key roles in the etiology and maintenance of depression. Indeed, their elevation among women may be an important factor underlying the gender difference in depression. It remains for future research to examine this formulation more explicitly.

Finally, we should note a number of limitations of the current study. First, participants in this study were university students, and a number of investigators have raised concerns about the use of such samples to test hypotheses about clinical depression. Clearly, therefore, these results should be replicated with a sample of clinically depressed individuals. Of related concern, it is important to determine whether neuroticism and rumination are vulnerabilities that are specific to depression or whether they increase risk to other psychological conditions and disorders, such as anxiety. Second, the current study used a relatively brief prospective interval of 6 to 8 weeks. Many individuals who might have been at risk for depression may have remained relatively low in depressive symptoms during this brief period. Clearly, future research would benefit from longer prospective intervals. Finally, the present study did not examine what factors might trigger or potentiate the processes that we investigated. For example, it is possible that these processes are activated by life stressors. Thus, individuals with higher levels of neuroticism might be more prone to ruminate when they experience stress than would less neurotic individuals. In contrast, these individuals would be no more likely to ruminate during stress-free periods than would less neurotic individuals. It remains for future research to test these hypotheses more explicitly.

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