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Ruminative Response Style and Vulnerability to Episodes of Dysphoria: Gender, Neuroticism, and Episode Duration

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A number of recent laboratory and prospective field studies suggest that the tendency to ruminate about dysphoric moods is associated with more severe and persistent negative emotional experiences (e.g., Morrow & Nolen-Hoeksema, 1990; Nolen-Hoeksema & Morrow, 1991). The current paper reports two studies that tested the hypotheses that (a) ruminative response styles act as a trait vulnerability to dysphoria, particularly to relatively persistent episodes of dysphoria; (b) aspects of rumination that are not likely to be contaminated with the presence and severity of previous symptomatology (introspection/self-isolation, self-blame) demonstrate vulnerability effects; and (c) rumination mediates the effects of gender and neuroticism on vulnerability to dysphoria. Consistent support was found for each of these hypotheses. Overall, our data suggest that rumination might reflect an important cognitive manifestation of neuroticism that increases vulnerability to episodes of persistent dysphoria.

KEY WORDS: dysphoria; ruminative response style; neuroticism.

INTRODUCTION

Investigators examining vulnerability to negative emotional states such as dysphoria have become increasingly interested in how individuals respond to these

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moods. Individual differences in cognitive responses to dysphoric mood states are hypothesized to determine whether or not those moods persist and spiral into more severe and persistent clinical disorders (Teasdale, 1988). For example, a number of theorists have suggested, that once a dysphoric mood is established, it can be maintained and exacerbated by mood-congruent biases in memory and attention (e.g., Gotlib & Hammen, 1992; Persons & Miranda, 1992; Segal & Ingram, 1994; Teasdale, 1988). In a similar manner, Nolen-Hoeksema (1987, 1991) proposed that individuals who respond to dysphoric moods with a ruminative response style are vulnerable to persistent dysphoria. "Ruminative responses involve repetitively focusing on the fact that one is depressed; on one's symptoms of depression; and on the causes, meanings, and consequences of depressive symptoms" (Nolen-Hoeksema, 1991, p. 569).

To date, Nolen-Hoeksema and her colleagues have garnered strong support for her model across a variety of study designs with nonclinical samples. In an early test, Morrow and Nolen-Hoeksema (1990) found that, following a depressive mood induction, individuals who were assigned to a physically active, distracting, task exhibited the greatest alleviation of dysphoric mood. In contrast, persons who were assigned to a physically passive, ruminative , task remained the most dysphoric. Similar results were found with naturally occurring nonclinical dysphoria (Nolen-Hoeksema & Morrow, 1993). To examine characteristic or typical levels of rumination, Nolen-Hoeksema and Morrow (1991) developed a self-report measure of response style. This questionnaire asks participants to report the types of thoughts and behaviors that they typically engage in when feeling dysphoric. In prospective studies with nonclinical samples, ruminative response styles have been found to predict higher levels of dysphoria over time, even after statistically controlling for the severity of initial dysphoria (Nolan, Roberts, & Gotlib, in press; Nolen-Hoeksema & Morrow, 1991; Nolen-Hoeksema, Parker, & Larson, 1994). Further, in another study based on 30 consecutive daily measurements of mood and responses to those moods, Nolen-Hoeksema, Morrow, and Fredrickson (1993) found that rumination predicted the duration of dysphoria. In this study, individuals tended to be consistent in their responses to dysphoric mood over time, suggesting that ruminative response style is a trait-like characteristic of individuals who are vulnerable to prolonged periods of dysphoric affect.

Interestingly, there are data to suggest that rumination might mediate the effects of other risk factors for dysphoria and depression. For example, the higher prevalence of depression in females than in males might be explained by the tendency of females to ruminate in response to dysphoria, whereas males tend to actively distract themselves from these negative moods (Nolen-Hoeksema, 1987; Nolen-Hoeksema *et al.*, 1993). In support of this hypothesis, Nolen-Hoeksema *et al.* (1994) found that, after statistically controlling for rumination, gender failed to predict changes in dysphoria over a 6-month period in a sample of bereaved individuals. However, gender did predict ruminative response style, which, in turn, was

associated with changes in dysphoria. These results suggest, therefore, that the effects of gender are mediated, at least in part, by ruminative response styles.

Theoretically, relatively global personality factors implicated in vulnerability to dysphoria and depression might also be mediated by ruminative response style. In particular, there is growing evidence that neuroticism is associated with risk for dysphoria and clinical depression. Neuroticism is generally considered to involve a heightened sensitivity to life stressors, high levels of worry, and a tendency to experience negative emotions (Eysenck & Eysenck, 1985). In terms of vulnerability, Hirschfeld *et al.* (1989) found in a prospective study that elevated levels of neuroticism predicted first episodes of clinical depression in initially nondepressed persons. Consistent with this finding, Kendell and DiScipio (1968) found that previously depressed individuals reported higher levels of neuroticism than did never depressed persons (see also Roberts & Gotlib, 1997a). Several other investigators have also found that elevated levels of neuroticism are associated with a poor long-term course of clinical depression (e.g., Duggan, Lee, & Murray, 1990; Weissman, Prusoff, & Klerman, 1978). Together, these findings suggest a robust association between neuroticism and vulnerability to dysphoria and clinical depression. It is unclear, however, whether this association reflects a causal process, or alternatively, is spurious and due to item overlap between measures of neuroticism and symptoms of affective distress. It is also possible that this association is due to a third variable, such as shared genetic underpinnings (Roberts & Gotlib, 1997b). Watson and Clark (1984) suggested a causal role for neuroticism: They argued that the negative affectivity associated with neuroticism results in part from these individuals' tendency to ruminate and to be self-focused. To the extent that neuroticism causally contributes to dysphoria and depression, it may be that individuals with high levels of neuroticism manifest this personality characteristic in a tendency to ruminate in response to dysphoric moods (cf. Nolen-Hoeksema *et al.*, 1994).

Although findings from laboratory and prospective field studies suggest that ruminative response style might be an important vulnerability factor to dysphoria, no research to date has examined whether the tendency to ruminate persists after episodes of dysphoria remit. Such "remission" designs are useful in testing whether putative risk factors are merely state-dependent features of dysphoria, or whether they act as trait-like characteristics that potentially predispose individuals to dysphoria. If rumination acts as a risk factor for dysphoria, as suggested by Nolen-Hoeksema (1991), elevated levels of rumination should be present both during *and* following episodes (as well as prior to episodes). If, however, rumination is merely a state characteristic (i.e., a symptom of dysphoria), elevated levels of rumination should be apparent *only* during episodes, and should return to normal ranges following remission. We should caution, however, that, on their own, remission designs cannot clearly determine whether a psychological characteristic acts as a trait vulnerability factor. Elevations on the characteristic among previously depressed indi-

viduals could also reflect a residual consequence or "scar" of the disorder itself (see Rohde, Lewinsohn, & Seeley, 1990).

Another concern with previous research involves measurement issues with Nolen-Hoeksema and Morrow's (1991) self-report measure of rumination. Previous investigations employing this questionnaire have failed to consider the possibility that rumination effects were due to confounds within this measure between the severity of previous dysphoric symptoms and the tendency to ruminate about those symptoms (i.e., criterion contamination). Specifically, a large proportion of items ask about typical responses to symptoms of dysphoria when one feels sad (e.g., "think about how hard it is to concentrate"; "think about your feelings of fatigue and achiness"). It is possible, therefore, that the presence and severity of previous symptoms contaminate the measurement of ruminative responses to those symptoms. That is, the rumination questionnaire requires individuals to make the fine distinction between *having* a particular symptom of dysphoria and *thinking* about that symptom. Therefore, scores on this inventory could represent a proxy indicator of the presence and severity of previous symptoms of dysphoria, rather than measuring rumination per se. For example, it is not possible for individuals to ruminate about concentration difficulties if their previous experiences with dysphoria typically did not include this symptom.

The present paper reports two studies utilizing remission designs to examine the role of ruminative response style in vulnerability to episodes of dysphoria. Although dysphoria has come to refer to nonspecific elevations of self-reported depressive symptoms (see Kendall, Hollon, Beck, Hammen, & Ingram, 1987), in the present study we defined dysphoria as episodes of symptomatology that meet symptom criteria for major depression according to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.) (DSM-IV; American Psychiatric Association, 1994). We used self-report measures to assess these episodes—the Inventory to Diagnose Depression (IDD; Zimmerman, Coryell, Corenthal, & Wilson, 1986) and the IDD-Lifetime Version (IDD-L; Zimmerman & Coryell, 1987). We refer to these episodes as dysphoria rather than as major depression because of the controversy concerning whether self-report instruments can validly diagnose major depression, particularly in nonclinical college samples (Coyne, 1994; Kendall *et al.*, 1987; Tennen, Hall, & Affleck, 1995). We should point out, however, that our dysphoric participants reported the full constellation of symptoms that defines major depression. In terms of potentially generalizing to clinical depression, classifying individuals on the basis of these specific diagnostic criteria is a clear advantage over simply examining persons with diffuse, elevated levels of depressive symptoms, which is the practice in the majority of studies based on nonclinical samples.[5]

[5]Although our samples were composed of college students, epidemiologic data indicate that up to 25% of individuals who develop major depressive disorder in their lifetime experience their first full episode before the age of 18 years, and that 54% experience their first episode by the age of 24 years (Sorenson, Rutter, & Aneshensel, 1991). These findings suggest that college samples are composed of a reasonable

In Study 1, we hypothesized that both currently dysphoric (CD) *and* previously dysphoric (PD) individuals would report greater rumination than would never dysphoric (ND) persons. In Study 2, we attempted to replicate and extend these results in a larger sample. If rumination leads to prolonged periods of dysphoria, ruminative response styles should be associated with relatively protracted dysphoric episodes. Therefore, we were interested in examining whether ruminative response styles are associated with the duration of episodes of dysphoria. Individuals whose worst lifetime episode of dysphoria met both DSM-IV symptom criteria for major depression *and* the 2-week duration criterion were classified as having protracted previous dysphoria (PPD), whereas those who met symptom criteria, but not duration criteria, were classified as having brief previous dysphoria (BPD). Based on Nolen-Hoeksema's (1991) formulation, we predicted that individuals whose episodes persisted for 2 weeks or longer (PPD) would report higher levels of rumination than would those with briefer episodes (BPD). Both of these groups were predicted to ruminate more than were ND individuals, even after controlling for the severity of current dysphoric symptoms.

In addition to the issue of duration, Study 2 examined the possibility that effects were due to potential confounds within the self-report measure of rumination between the presence and/or severity of previous symptoms of dysphoria and responses to those symptoms. In order to increase confidence in the vulnerability explanation, we wanted to determine whether those responses to dysphoria that were relatively independent of symptoms (e.g., "listen to sad music") would demonstrate the same pattern of results as was obtained with the complete rumination scale. In particular, we examined the relation among several dimensions of ruminative response style (determined through factor analysis) and history of dysphoria. Finally, we tested a path model linking gender, neuroticism, and rumination to vulnerability to dysphoric episodes. The model posits that females experience higher levels of neuroticism (Roberts & Gotlib, 1997a), which, in turn, are associated with greater rumination. In the final step of the model, rumination is posited to directly increase vulnerability to episodes of dysphoria. In this model, ruminative response style is seen as a specific cognitive and behavioral manifestation of neuroticism; therefore, rumination would be a more proximal contributor to vulnerability to episodes of dysphoria than would neuroticism. In addition, to help establish the consistency of findings, Study 2 was replicated on an independent sample of participants.

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number of individuals who have had bouts of relatively severe depression. We also should note that, in a study employing structural equation modeling of twin data, a simple self-report assessment of lifetime history of major depression was found to be a stronger predictor of true liability to major depression than were diagnoses based on structured interviews (Kendler, Neale, Kessler, Heath, & Eaves, 1993b). As suggested by these authors, participants from the community might respond more openly and honestly to an anonymous self-report measure of depression than to a face-to-face interview with a stranger.

STUDY 1

Method

Participants

Participants in Study 1 were 62 college students (34 female, 18 male) selected from a mass testing session (total In = 151) at Northwestern University during the Spring quarter of 1994. We selected 20 currently dysphoric (CD), 21 previously dysphoric (PD), and 21 never dysphoric (ND) individuals (based on their responses to the IDD and IDD-L) to participate in this study. As discussed previously, dysphoria was defined as meeting DSM-IV symptom criteria for major depression, regardless of the 2-week duration criterion. Later that academic quarter, participants were invited to the laboratory in individual sessions and completed measures of rumination and depressive symptoms. As recommended by Kendall *et al.* (1987), participants who changed dysphoria status between the mass testing and laboratory sessions (CD individuals with Beck Depression Inventory [BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961] scores < 10; PD and ND individuals with BDI scores > 9) were eliminated from all analyses. This procedure resulted in the loss of seven CD (final n = 13), eight PD (final n = 13), and two ND (final n = 19) participants.

Measures

Rumination. The Ruminative Response Scale of the Response Style Questionnaire (Nolen-Hoeksema & Morrow, 1991) was used to assess how participants tended to respond to their own feelings and symptoms of dysphoria. The Ruminative Response Scale consists of 21 items that assess responses to dysphoric mood that are focused on the self (e.g., "Think 'Why do I always react this way?'"), focused on symptoms (e.g., "Think about how hard it is to concentrate"), or focused 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 inventory also includes a number of behavioral responses (e.g., "Listen to sad music" and "Write down what you are thinking about and analyze it").

Dysphoria. The Inventory to Diagnose Depression (Zimmerman *et al.*, 1986) was used to classify participants categorically according to DSM-IV symptom criteria of major depression. When completed within 2 days of each other, the IDD shows good agreement with the Diagnostic Interview Schedule (k = .8; Zimmerman & Coryell, 1988). Items on the IDD can also be summed to create an overall severity index. This index correlates highly with total scores on the Beck Depression Inventory (r = .87) and the Hamilton Rating Scale (r = .80; Zimmerman *et al.*, 1986). The IDD-Lifetime Version (Zimmerman & Coryell, 1987) was used to assess

participants' worst lifetime period of dysphoria. The IDD-L has good sensitivity (74%) and specificity (93%) when compared to the Diagnostic Interview Schedule (k = .6; Zimmerman & Coryell, 1987). Finally, the BDI was used to assess severity of current depressive symptomatology.

Results and Discussion

Preliminary analysis of the data demonstrated that the three dysphoria groups differed in terms of current BDI scores, $F(2,42) = 68.09$, $p < .001$, as expected by our selection procedure. As presented in Table I, follow-up Tukey tests indicated that CD participants were more symptomatically depressed on the BDI than were PD ($p < .001$) and ND individuals ($p < .001$), who did not differ significantly from each other ($p > .2$).

Of most interest, the three dysphoria groups also differed significantly in terms of rumination, $F(2,42) = 18.80$, $p < .001$. As presented in Table I, follow-up Tukey tests indicated that CD ($p < .001$) and PD individuals ($p < .01$) reported greater rumination than did ND participants, but did not differ significantly from each other ($p > .15$). Because PD individuals reported slightly (although not significantly) higher scores on the BDI than did ND participants, a one-way analysis of covariance (ANCOVA) was conducted, comparing these two groups on rumination with current BDI scores treated as a covariate. Importantly, PD individuals continued to show significant elevations in rumination even after current BDI scores were covaried, $F(1,29) = 11.88$, $p < .01$.

The results of Study 1 suggest that rumination might act as a trait characteristic that persists subsequent to remission of dysphoric episodes. In particular, PD individuals demonstrated levels of rumination as high as those exhibited by CD persons, and both of these groups of participants indicated that they ruminated in response to dysphoric moods to a greater extent than did ND individuals. Although the results of this study are consistent with the possibility that rumination acts as a trait-

Table I. Mean Depression and Rumination Scores across Depression Groups in Study 1[a]

Measures	Currently dysphoric (n = 13)		Previously dysphoric (n = 13)		Never dysphoric (n = 19)	
	M	*SD*	*M*	*SD*	*M*	*SD*
Current BDI	16.8_a	4.5	5.1_b	3.0	3.5_b	2.5
Rumination	55.8_a	10.5	48.1_a	11.5	33.0_b	10.3

[a]BDI = Beck Depression Inventory. Means with identical subscripts are not statistically different at $p < .05$.

like characteristic that increases vulnerability to dysphoria, these findings could be spurious. As discussed in the Introduction, it is possible that the presence and/or severity of prior symptoms of dysphoria and ruminative responses to those symptoms are confounded within the rumination scale. This potential confound could account for the findings of Study 1, and was examined directly in Study 2.

STUDY 2

In addition to confirming the findings from the previous investigation in a larger sample, Study 2 was conducted to (a) determine whether rumination is associated with the duration of individuals' worst lifetime episode of dysphoria, (b) examine whether the relatively less symptom-contaminated components of ruminative style are related to history of dysphoria, and (c) test whether rumination mediates the effects of gender and neuroticism on dysphoria. Data were gathered during two mass testing sessions: one in the fall quarter of 1994 (n = 299) and one in the winter quarter of 1995 (n = 317). In order to have a "built-in" replication, these two samples were analyzed separately.

Method

Participants

In Sample 1, based on their responses to the IDD and IDD-L, eight participants[6] (five female, three male) were classified as currently dysphoric (CD), 41 (26 female, 15 male) as prolonged previously dysphoric (PPD), 75 (51 female, 24 male) as brief previous dysphoric (BPD), and 175 (80 female, 95 male) as never dysphoric (ND). In Sample 2, 16 participants[7] (11 female, five male) were classified as CD, 46 (26 female, 20 male) as PPD, 74 (49 female, 23 male) as BPD, and 181 (100 female, 81 male) as ND.

Measures

Measures were identical to those used in Study 1, except that the Eysenck Personality Inventory (EPI; Eysenck & Eysenck, 1964) was included to measure neuroticism, and the IDD, rather than the BDI, was used to evaluate severity of current symptoms.

[6]Seven of these participants met the DSM-IV 2-week duration criterion for either their current or worst lifetime episode of depression.

[7]Fourteen of these participants met the DSM-IV duration criterion for either their current or worst lifetime episode of depression.

Results

Differences Across Dysphoria Groups

Sample 1. As can be seen in Table II, the four dysphoria groups in Sample 1 differed in severity of current depressive symptoms, $F(3,295) = 59.25$, $p < .001$. Consistent with our grouping procedure, follow-up Tukey tests indicated that CD individuals were more symptomatically depressed on the IDD than were any of the other three groups (all ps $< .001$). Further, PPD and BPD individuals were more symptomatic than were ND participants (both ps $< .001$), but did not differ significantly from each other ($p > .2$). The four dysphoria groups also differed on neuroticism, $F(3,295) = 26.37$, $p < .001$. As can be seen in Table II, ND individuals reported lower levels of neuroticism than did any participants in the other three groups (ps $< .001$). Further, BPD individuals reported less neuroticism than did CD participants ($p < .01$).

Of most theoretical interest, the four dysphoria groups differed significantly in total rumination, $F(3,295) = 35.79$, $p < .001$. Consistent with our hypotheses, and as can be seen in Table II, follow-up Tukey tests indicated that ND individuals endorsed less rumination than did participants in any of the other three groups (all ps $< .001$). CD individuals endorsed levels of rumination statistically equivalent to that of PPD and BPD participants (ps $> .1$). Nonetheless, PPD individuals were more ruminative than were BPD participants ($p < .01$). This finding suggests that rumination is associated with relatively prolonged episodes of dysphoria. Because PPD and BPD participants differed from ND individuals in current depressive symptomatology and neuroticism, a conservative follow-up ANCOVA was conducted comparing these groups on rumination, with current IDD and neuroticism

Table II. Mean Depression, Neuroticism, and Rumination Scores across Dysphoria Groups in Sample 1 of Study 2[a]

	CD (n = 8)		PPD (n = 41)		BPD (n = 75)		ND (n = 175)	
Measures	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Control variables								
Current IDD	33.2_a	6.1	13.0_b	5.6	11.2_b	6.6	7.8_c	5.1
Neuroticism	16.9_a	3.7	13.4_{ab}	3.6	11.3_b	4.8	8.5_c	4.0
Rumination								
Total rumination	54.1_{ab}	12.8	51.8_a	11.9	45.4_b	10.2	36.4_c	9.8
Symptom-Based	18.9_a	3.7	15.8_a	4.5	13.5_b	3.3	11.2_c	3.2
Introspection/Self-Isolation	10.5_{ab}	3.9	12.8_a	4.2	11.8_a	3.9	8.8_b	3.3
Self-Blame	8.0_a	3.2	7.5_a	2.6	6.7_a	1.8	5.7_b	1.9

[a]CD = currently dysphoric, PPD = prolonged previous dysphoria, BPD = brief previous dysphoria, ND = never dysphoric, IDD = Inventory to Diagnose Depression, Symptom-Based = Symptom-Based Rumination. Means with similar subscripts are not statistically different at $p < .05$.

treated as covariates. Again, the three groups differed significantly, $F(2,286) = 23.20$, $p < .001$, with ND individuals ruminating less than PPD and BPD participants (both ps $< .001$), and BPD individuals ruminating less than PPD participants ($p < .05$). Thus, even after controlling for differences in current subclinical depressive symptomatology and neuroticism, rumination was associated with previous dysphoric episodes, and particularly with relatively prolonged episodes.

Sample 2. Preliminary analysis indicated that the four dysphoria groups in Sample 2 differed significantly on severity of current depressive symptomatology, $F(3,313) = 107.37$, $p < .001$. As can be seen in Table III, follow-up Tukey tests indicated that CD participants were more symptomatically depressed on the IDD than were any of the other three groups (all ps $< .001$). Further, PPD and BPD individuals endorsed more symptomatology than did ND participants (both ps $< .001$), but did not differ significantly from each other ($p > .2$). The four dysphoria groups also differed on neuroticism, $F(3,313) = 25.66$, $p < .001$, and these group differences paralleled those found on depressive symptoms.

Consistent with our previous findings, the four dysphoria groups in Sample 2 differed in level of total rumination, $F(3,313) = 42.22$, $p < .001$. As can be seen in Table III, ND individuals were less ruminative than were participants in each of the other three groups (all ps $< .001$). PPD individuals were more ruminative than were BPD participants ($p < .05$), suggesting again that rumination is associated with relatively prolonged episodes of dysphoria. In contrast to Study 1 and Sample 1, CD individuals reported greater rumination than did PPD ($p < .05$) and BPD participants ($p < .001$). Because PPD and BPD participants were more symptomatically depressed and neurotic than were ND individuals, a conservative analysis was conducted comparing these three groups, with current IDD and neuroticism treated as a covariates. Consistent with our earlier findings, the results of this AN-

Table III. Mean Depression, Neuroticism, and Rumination Scores Across Dysphoria Groups in Sample 2 of Study 2[a]

	CD (n = 16)		PPD (n = 46)		BPD (n = 74)		ND (n = 181)	
Measures	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Control variables								
Current IDD	34.3_a	6.7	14.5_b	5.6	12.9_b	7.0	8.4_c	5.1
Neuroticism	19.2_a	3.2	13.9_b	4.6	13.2_b	4.1	10.7_c	4.3
Rumination								
Total rumination	60.4_a	9.4	51.4_b	11.1	46.5_c	10.8	38.3_d	9.7
Symptom-Based	19.4_a	3.8	15.5_b	4.0	14.1_b	3.4	11.6_c	2.9
Introspection/Self-Isolation	13.4_a	3.9	12.5_a	3.9	11.0_a	3.8	9.2_b	3.3
Self-Blame	8.9_a	1.9	7.6_{ab}	2.2	7.0_b	1.9	5.8_c	1.8

[a]CD = currently dysphoric, PPD = prolonged previous dysphoria, BPD = brief previous dysphoria, ND = never dysphoric, IDD = Inventory to Diagnose Depression, Symptom-Based = Symptom-Based Rumination. Means with similar subscripts are not statistically different at $p < .05$.

COVA indicated that these groups continued to differ after controlling for current depressive symptoms and neuroticism, $F(2,296) = 20.85$, $p < .001$. PPD individuals were marginally more ruminative than were BPD participants ($p < .06$), and both groups were more ruminative than were ND individuals (both ps $< .001$).

Factor Components of Rumination

Sample 1. In order to examine components of rumination that are not confounded with severity of previous dysphoric symptoms, a principal components factor analysis with varimax rotation was conducted on the rumination scale. Four factors with eigenvalues greater than 1 emerged. However, the fourth factor was uninterpretable and a scree test indicated that it accounted for little additional variance. Therefore, the analysis was rerun selecting for three factors. These three factors accounted for 55.7% of the total variance. Factor scales were constructed based on items that loaded higher than .4 on only a single factor. Items were summed with unit weighting. Factor 1 consisted of seven items ($\alpha = .81$) and was labeled Symptom-Based Rumination (e.g., "think about how passive and unmotivated you feel"). It accounted for 17.4% of unique variance. Factor 2 was composed of five items ($\alpha = .84$) and was labeled Introspection/Self-Isolation (e.g., "Go someplace alone to think about the reasons why you feel sad"). This factor accounted for 21.2% of the unique variance. Factor 3 consisted of three items ($\alpha = .71$) and was labeled Self-Blame (e.g., "Think 'Why do I always react this way?'"). It accounted for 17.1% of the unique variance. Interestingly, Factor 1 was composed of exactly those types of items that we believed could be highly confounded with the severity of previous depressive symptoms. In contrast, Factors 2 and 3 were conceptually independent of depressive symptomatology. Factor loadings are presented in Table IV.

Females reported higher levels of Symptom-Based Rumination, $t(289) = 2.11$, $p < .05$, and Introspection/Self-Isolation, $t(289) = 2.82$, $p < .01$, than did males, but did not differ from males on Self-Blame, $t(289) = 1.59$, $p > .10$. The three dimensions of rumination were all significantly intercorrelated (rs ranged from .39 to .45, all ps $< .001$). Each dimension was associated with both current depressive symptoms (rs = .26 to .43, all ps $< .001$) and worst lifetime depressive symptoms (rs = .39 to .45, all ps $< .001$). Symptom-Based Rumination ($r = .33$, $p < .001$), Introspection/Self-Isolation ($r = .19$, $p < .01$), and Self-Blame ($r = .43$, $p < .001$) each correlated significantly with neuroticism.

To examine the possibility that confounding within the rumination scale between previous dysphoric symptoms and ruminative responses accounted for our vulnerability findings, the four dysphoria groups were compared on each of the three subscales of rumination (Symptom-Based Rumination, Introspection/Self-Isolation, Self-Blame). The "confounding" hypothesis posited that group differences would be apparent only on Symptom-Based Rumination, whereas the "vulnerabil-

Table IV. Factor Loadings of Ruminative Response Scale[a]

	Factor 1	Factor 2	Factor 3
1. Think about your feelings of fatigue and achiness	**.715** (.275)	.089	.011
2. Think about how passive and unmotivated you feel	**.685** (.644)	.227	.201
3. Think "I won't be able to do my job/work because I feel so badly"	**.674** (.411)	.147	.231
4. Think about how you don't feel up to doing anything	**.671** (.606)	.079	.376
5. Think about how hard it is to concentrate	**.631** (.466)	.108	.130
6. Think about how alone you feel	**.451** (.391)	.379 (.278)	.376
7. Think about how you don't seem to feel anything any more	**.435** (.495)	.333	.142
8. Go someplace alone to think about your feelings	.158	**.849** (.916)	.107
9. Isolate yourself and think about the reasons why you feel sad	.208	**.838** (.797)	.220
10. Go away by yourself and think about why you feel this way	.113	**.775** (.929)	.227
11. Write down what you are thinking about and analyze it	.063	**.703** (.375)	−.089
12. Listen to sad music	.250	**.466** (.452)	.061
13. Think "Why do I always react this way?"	.084	.140	**.740** (.566)
14. Think about a recent situation wishing it had gone better	.151	.204	**.672** (.460)
15. Think about how angry you are with yourself	.358	.173	**.667** (.693)
16. Try to understand yourself by focussing on your depressed feelings	.199	.645	.412
17. Analyze your personality to try to understand why you are depressed	.049	.631	.547
18. Analyze recent events to try to understand why you are depressed	.123	.605	.414
19. Think about all your shortcomings, failings, faults, mistakes	.429	.092	.694
20. Think about how sad you feel	.492	.297	532
21. Think "Why can't I get going?"	.449	.042	.453

[a]Loadings for items selected for factor scales are represented in boldface. Factor loadings for Sample 2 are presented in parentheses.

ity" hypothesis would be supported by the presence of differences on Introspection/Self-Isolation and Self-Blame. With respect to Symptom-Based Rumination, a one-way analysis of variance (ANOVA) indicated that the four dysphoria groups differed significantly, $F(3,295) = 33.09$, $p < .001$. As can be seen in Table II, CD individuals exhibited greater Symptom-Based Rumination than did BPD and ND participants (both ps $< .001$). PPD persons reported more Symptom-Based Rumination than did BPD individuals ($p < .01$), and both groups were more ruminative than were ND individuals (both ps $< .001$). The four groups also differed with respect to Introspection/Self-Isolation, $F(3,295) = 21.65$, $p < .001$, with PPD and BPD individuals showing greater introspection/self-isolation than ND participants (both ps $< .001$). Finally, the four groups differed on Self-Blame, $F(3,295) = 13.41$, $p < .001$. Although CD individuals were no more self-blaming than were PPD and

BPD participants (all *p*s > .3), individuals in all three dysphoria groups were more self-blaming than were ND individuals (all *p*s < .01).

Sample 2. In order to replicate the factor structure obtained in Sample 1, a confirmatory factor analysis was conducted with LISREL 7 (Joreskog & Sorbom, 1989) on data from Sample 2. This three-factor model provided an adequate fit to the data: $\chi^2(87, n = 317) = 256.77$, $p < .001$, χ^2/df ratio = 2.95, root mean square (RMS) = .052, goodness-of-fit index (GFI) = .900, adjusted GFI = .863, and all factor loadings were significant. Nonetheless, the residual of an item loading on Symptom-Based Rumination ("think about how alone you feel") had large correlations (*r*s > .15) with the residuals of two items loading on Introspection/Self-Isolation ("go away by yourself and think about why you feel this way" and "isolate yourself and think about the reasons why you feel sad"). These correlations suggest that the item "think about how alone you feel" might load on both Symptom-Based Rumination and Introspection/Self-Isolation. The modified model, which included this dual loading, provided an improved fit to the data, $\chi^2(86, n = 317) = 232.58$, $p < 001$, χ^2/df ratio = 2.70, RMS = .046, GFI = .910, Adjusted GFI = .874. Factor loadings, including the additional dual loading, were all statistically significant and are presented in parentheses in Table IV. Coefficient alphas were .79 for Symptom-Based Rumination, .81 for Introspection/Self-Isolation, and .66 for Self-Blame.

Although females in Sample 2 reported higher levels of Introspection/Self-Isolation than did males, $t(299) = 2.96$, $p < .01$, there were no significant gender differences on Symptom-Based Rumination, $t(299) = 1.48$, $p > .10$, or Self-Blame, $t(299) = 1.38$, $p > .10$. Consistent with Sample 1, the three dimensions of rumination were significantly intercorrelated (*r*s = .35 to .56, all *p*s < .001). Further, the three dimensions were all significantly associated with current depressive symptoms (*r*s = .23 to .41, all *p*s < .001) and worst lifetime depressive symptoms (*r*s = .34 to .45, all *p*s < .001). Symptom-Based Rumination ($r = .41$, $p < .001$), Introspection/Self-Isolation ($r = .16$, $p < .01$), and Self-Blame ($r = .44$, $p < .001$) were each significantly correlated with neuroticism.

As in Sample 1, the four dysphoria groups were compared on each of these subscales of rumination. As can be seen in Table III, Symptom-Based Rumination differed across the four dysphoria groups, $F(3,313) = 43.71$, $p < .001$. ND individuals were less ruminative than were CD, PPD, and BPD participants (all *p*s < .001). BPD and PPD individuals were less ruminative than were CD participants ($p < .001$), but did not differ from each other ($p > .10$). The four groups also differed with respect to Introspection/Self-Isolation, $F(3,313) = 16.16$, $p < .001$: ND individuals were less self-isolating than were CD ($p < .001$), PPD ($p < .001$), and BPD ($p < .01$) persons. No other pair-wise comparisons were significant. Finally, the four groups differed significantly on Self-Blame, $F(3,313) = 23.59$, $p < .001$. ND individuals were less self-blaming than were participants in each of the three other groups (all *p*s < .001), and CD individuals were more self-blaming than were BPD participants ($p < .01$). No other pair-wise comparisons were significant.

Path Model

Sample 1. Our path model portrayed in Fig. 1 maintains that females have a higher level of neuroticism than do males, which is associated with greater rumination. Rumination, in turn, is posited as directly affecting vulnerability to episodes of dysphoria. Gender was treated as the most "upstream" variable because it is logically prior to any of the other psychological variables in the model. Neuroticism was considered to be causally prior to rumination because neuroticism is thought to reflect a rather broad dimension of personality involving ge neral sensitivity to stres s, whereas rumination is thought to involve more specific cognitive and behavioral responses.

Consistent with the overall model, females reported higher levels of depressive symptoms than did males, both currently (10.8 vs. 9.2), $t(297) = 1.99$, $p < .05$, as well as during their worst lifetime episodes of dysphoria (25.9 vs. 21.3), $t(294) = 3.10$, $p < .01$. Also consistent with our model, females reported greater neuroticism (11.1 vs. 8.9), $t(297) = 4.22$, $p < .001$, and a higher level of rumination (43.1 vs. 39.0), $t(297) = 2.94$, $p < .01$. Importantly, neuroticism and rumination were positively correlated ($r = .43$, $p < .001$).

To further test our path model, a series of three simultaneous regression analyses were conducted (Cohen & Cohen, 1983, pp. 352-378; see Nolen-Hoeksema *et al.*, 1994, for another recent example). As discussed below, in each of these analyses current depressive symptoms were treated as a covariate and were controlled statistically. In the first analysis, neuroticism was regressed on gender and current depressive symptoms to determine whether gender contributed to neuroticism after controlling for current depressive symptoms. In the second analysis, ruminative response style was regressed on gender, neuroticism, and current depressive symptoms. This analysis tested whether neuroticism made an independent contribution to the prediction of rumination after controlling for more "upstream" and control variables (gender, current symptoms). In addition, this analysis tested whether gender made direct contributions to rumination, or whether its association was entirely mediated by neuroticism. Finally, lifetime episodes of dysphoria was regressed on gender, neuroticism, ruminative response style, and current depressive symptoms. This analysis tested whether rumination made a contribution to the prediction of vulnerability to episodes of dysphoria after controlling for theoretically prior and control variables (neuroticism, gender, current symptoms). In addition, this analysis also tested whether gender and neuroticism made direct contributions to vulnerability to dysphoria, or whether their associations were entirely mediated by rumination.

For the purpose of these regression analyses, ND individuals were coded 1 on lifetime episodes of dysphoria, BPD individuals were coded 2, and PPD persons were coded 3. Consistent with our earlier work (Roberts & Gotlib, 1997a), CD participants were excluded from analyses because of the likelihood that current depressive symptomatology and neuroticism would be contaminated (Larsen, 1992). Further, current depressive symptoms were statistically controlled in each of these regression analyses

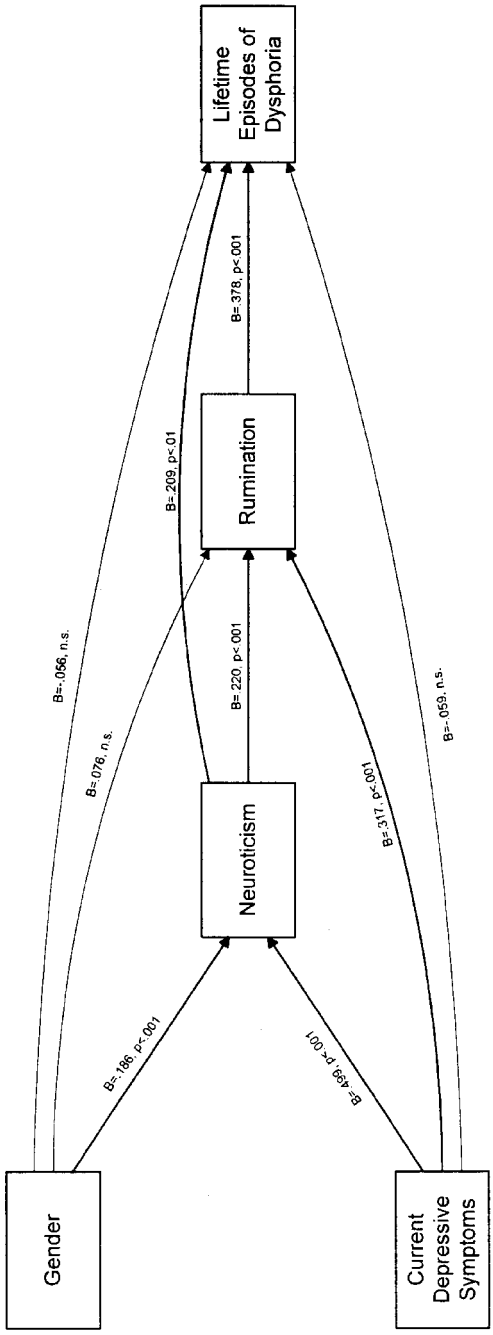

Fig. 1. Path model of relations among gender, current depression, neuroticism, rumination, and lifetime dysphoric episodes in Sample 1. Nonsignificant paths are represented with thinner lines.

in order to provide a conservative test of the model by reducing the possibility that concurrent dysphoria biases or contaminates other self-report measures. Gender was dummy coded, with males equal to 0 and females equal to 1.

As can be seen in Fig. 1, gender, $t(288) = 3.76$, $p < .001$, and current depressive symptoms, $t(288) = 10.09$, $p < .001$, each made significant unique contributions to the prediction of neuroticism. Females and persons who were more symptomatically depressed reported higher levels of neuroticism. In turn, higher levels of neuroticism, $t(287) = 3.56$, $p < .001$, and current depressive symptoms, $t(287) = 5.26$, $p < .001$, were each associated with greater rumination. Finally, higher levels of neuroticism, $t(286) = 3.46$, $p < .01$, and rumination, $t(286) = 6.69$, $p < .001$, each made significant independent contributions to lifetime dysphoric episodes, even after statistically controlling for gender and current depression. Interestingly, gender was not directly associated with lifetime episodes of dysphoria after controlling for neuroticism and rumination. Gender contributed to dysphoria only indirectly through its impact on neuroticism and rumination. In Fig. 1, paths are represented with standardized beta coefficients.[8] Nonsignificant paths are portrayed with thinner lines.

Sample 2. Although gender was unrelated to current, $t(315) < 1$, or past levels of depressive symptomatology, $t(314) = 1.23$, $p > .05$, females reported higher levels of both neuroticism (12.8 vs. 11.3), $t(315) = 2.74$, $p < .01$, and rumination (44.4 vs. 41.5), $t(315) = 2.10$, $p < .05$, than did males. Consistent with Sample 1, neuroticism and rumination were positively correlated ($r = .43$, $p < .001$).

Consistent with the results obtained with Sample 1, and as can be seen in Fig. 2, gender, $t(298) = 2.98$, $p < .01$, and current depression, $t(298) = 13.26$, $p < .001$, each made significant contributions to the prediction of neuroticism. Ruminative response style was predicted by both neuroticism, $t(297) = 3.18$, $p < .01$, and current depressive symptoms, $t(297) = 3.57$, $p < .001$, whereas lifetime episodes of dysphoria was associated with higher levels of rumination, $t(296) = 6.41$, $p < .001$, and current depressive symptoms, $t(296) = 4.22$, $p < .001$. In contrast to Sample 1, neuroticism failed to make a significant direct contribution to the prediction of vulnerability to dysphoric episodes. Its effect was indirect and was entirely mediated by rumination.[9]

[8]As noted by a reviewer, because our dependent measure—lifetime episodes of dysphoria—is ordinal, linear regression was not the most appropriate data analytic approach. Consequently, we reran the final step of the path analysis using logistic regression. For the purpose of this analysis, ND and BPD individuals were coded as 0 and PPD participants (who met both symptom *and* duration criteria for major depression) were coded as 1. Importantly, the results of this analysis were essentially identical to those based on the linear regression analysis. In particular, both rumination (Wald = 19.81, $p < .001$) and neuroticism (Wald = 9.88, $p < .01$) continued to make significant contributions to the prediction of lifetime episodes of dysphoria, whereas current depressive symptoms and gender were not significant.

[9]We conducted a logistic regression analysis similar to that described in footnote 8. Again, the results were essentially identical to those obtained with linear regression. In particular, rumination (Wald = 19.71, $p < .001$) and current depressive symptoms (Wald = 6.89, $p < .01$) continued to make significant contributions to the prediction of lifetime episodes of dysphoria, whereas neuroticism and gender were not significant.

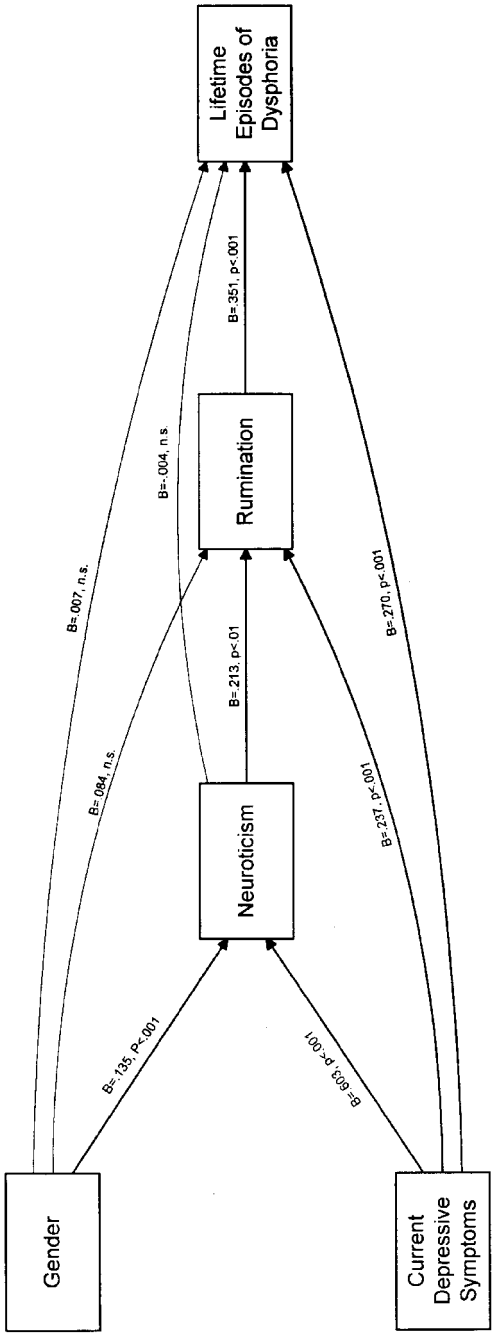

Fig. 2. Path model of relations among gender, current depression, neuroticism, rumination, and lifetime dysphoric episodes in Sample 2. Nonsignificant paths are represented with thinner lines.

GENERAL DISCUSSION

Across three samples (total $n = 661$), elevated levels of rumination were found in currently dysphoric and previously dysphoric individuals compared to never dysphoric controls. This pattern of results indicates that rumination might be an important trait vulnerability characteristic in dysphoria. Not only are ruminative tendencies apparent during dysphoric episodes, but they also persist once these episodes have remitted. Further, rumination was associated with relatively prolonged episodes of dysphoria. Individuals who previously experienced dysphoric episodes lasting 2 weeks or longer reported greater rumination than did individuals who previously experienced briefer episodes. Finally, the present data are consistent with a model that posits that rumination accounts for the effects of gender and neuroticism on vulnerability to episodes of dysphoria. Combined with data from both clinical (Just & Alloy, 1997) and nonclinical prospective field studies (e.g., Nolen-Hoeksema & Morrow, 1991; Nolen-Hoeksema *et al.*, 1994), and laboratory studies (Morrow & Nolen-Hoeksema, 1990; Nolen-Hoeksema & Morrow, 1994), it appears that rumination both precedes and follows periods of dysphoria. Consequently, rumination might be a trait characteristic that increases individuals' vulnerability to developing relatively protracted episodes of dysphoria. These protracted episodes of dysphoria might then spiral into more severe episodes of clinical depression.

Importantly, our results were not due to confounds between rumination and current depressive symptomatology or neuroticism. Although previously dysphoric individuals reported greater subclinical depressive symptomatology and higher levels of neuroticism than did never dysphoric persons, significant group differences on rumination remained after these variables were controlled statistically. Therefore, potential response biases resulting from current depressive symptomatology or neurotic personality characteristics could not account for our results. Further, these findings were not due to confounds within the self-report measure of rumination between responses to *symptom-based* items and the *presence* and *severity* of those same symptoms during previous episodes of dysphoria. As we discussed earlier, many items on the ruminative response scale require persons to report the degree to which they generally think about certain depressive symptoms (e.g., concentration difficulties) when they feel dysphoric. It is unclear how accurately individuals can report ruminative responses to depressive symptoms independent of the severity of those symptoms. For example, persons who do not experience concentration difficulties when they feel dysphoric (perhaps because they only experience mild dysphoria) would be unable to ruminate about this problem. Potentially, these items could create spurious associations between previous episodes of dysphoria and ruminative response styles. The present findings demonstrate that those components of the rumination scale that are relatively independent of symptoms (e.g., aspects of Introspection/Self-Isolation, such as "Go away by yourself and think about why you feel this way" and Self-Blame, such as "Think 'Why do I always react this

way?") are also associated with vulnerability to episodes of dysphoria. We should note, however, that it is still possible that introspection/self-isolation and self-blame are consequences or scars of past episodes rather than causal factors. However, when considered together with laboratory studies that experimentally manipulate rumination (e.g., Morrow & Nolen-Hoeksema, 1990) and prospective field studies (e.g., Nolen-Hoeksema *et al.*, 1994), we believe that the vulnerability hypothesis is more tenable.

The path model that we proposed and tested suggests that rumination is an important cognitive manifestation of neuroticism: relatively neurotic individuals tend to focus their attention on dysphoric moods and experiences, which, in turn, amplifies these feelings and leads to more persistent and severe episodes of dysphoria. These findings are important because they help to elucidate the mechanism by which neuroticism is associated with vulnerability to dysphoria. Previous research has demonstrated that neuroticism predicts the onset of depression (Hirschfeld *et al.*, 1989; Kendler, Kessler, Neale, Heath, & Eaves, 1993a), as well a more pernicious course in patients diagnosed with clinical depression (e.g., Weissman *et al.*, 1978). Similarly, in an earlier study, we found that previously dysphoric individuals reported higher levels of neuroticism than did never dysphoric subjects, and that the association between gender and vulnerability to dysphoric episodes was mediated by neuroticism (Roberts & Gotlib, 1997a). However, none of these studies investigated the *process* by which neuroticism leads to vulnerability. The present research suggests that neuroticism is associated with a cognitive style involving attentional focus on dysphoric moods, which, in turn, increases vulnerability (see also Derryberry & Reed, 1994; Martin, 1985). Further, our data suggest that neuroticism primarily contributes to specific dimensions or aspects of rumination, namely, Symptom-Based Rumination and Self-Blame. Although its association with Introspection/Self-Isolation was statistically significant, these correlations were small (rs = .16 and .19 in the two samples in Study 2).

The present findings are also consistent with Nolen-Hoeksema's (1987, 1991) hypothesis concerning gender differences in depression. In our data, to the extent that females were at greater risk for dysphoria, this vulnerability was entirely due to females higher levels of neuroticism and tendency to ruminate in response to depressive moods. Females with lower levels of neuroticism, who tended to ruminate less, were at no greater risk for dysphoric episodes than were males. Of the three dimensions of rumination derived through factor analysis, females appeared to be at particular risk to become introspective and to isolate themselves in response to negative moods. Thus, the current data suggest that introspection and self-isolation might contribute to gender differences in depression.

More generally, the current data also have important practical implications concerning the manner in which normal control groups are selected in depression vulnerability research. These groups typically exclude individuals with previous episodes of major depression in order to increase homogeneity. However, our findings

suggest that, even after these selection procedures, normal control groups likely remain heterogenous. In particular, they include a large number of individuals who have met symptom criteria for major depression at some point in their lives, but not the duration criterion. Our findings suggest that these individuals with brief episodes of symptomatology are distinguishable from persons who have never met symptom criteria for depression with respect to ruminative response style and neuroticism. These results are consistent with findings from our previous study (Roberts & Gotlib, 1997a), which focused exclusively on neuroticism. Across these studies, approximately 25% of unselected student samples reported that they had previously experienced these briefer episodes, which we refer to as episodes of brief previous dysphoria, whereas approximately 14% had previously experienced episodes meeting the DSM-IV 2-week duration criterion.[10] Interestingly, Angst, Merikangas, Scheidegger, and Wicki (1990) have recently described a subtype of depression they refer to as recurrent brief depression. This subtype is characterized by at least monthly recurrence of a depressive syndrome of shorter duration than that described in DSM-IV (i.e., less than 2 weeks). Given that in our studies individuals with episodes of brief previous dysphoria reliably differed from never dysphoric persons on theoretically important psychological characteristics, we believe that it is crucial to examine these individuals separately from never dysphoric persons (see also Gotlib, Lewinsohn, & Seeley, 1995).

The current research suggests a number of important directions for future work. Perhaps most importantly, our findings need to be replicated with patient groups that have been diagnosed through structured interviews. Although 104 participants in Study 2 indicated that they had received some form of therapy for depression (15 reported that they had been treated with pharmacotherapy), the majority of our dysphoric and previously dysphoric participants likely experienced episodes that were substantially less severe and prolonged than those that are typical of patient groups (Coyne, 1994). It is an open question as to whether remitted depressed *patients* would show elevations on rumination. In addition to clinically depressed patient groups, the inclusion of nondepressed psychiatric controls would be important in order to examine whether the effects of rumination are specific to dysphoria and depression.

Although prior research has prospectively investigated rumination as a risk factor for elevated depressive symptoms (Nolen-Hoeksema *et al.*, 1994), future studies need to investigate whether rumination is prospectively associated with the duration of episodes of dysphoria and clinical depression. Several studies have demonstrated that depressed patients with higher levels of neuroticism tend to have relatively protracted episodes of depression (e.g., Duggan *et al.*, 1990). The present research

[10]We should note that this lifetime prevalance of 14% is comparable to results from a recent national epidemiologic study that employed structured diagnostic interviewing and found a lifetime prevalance of major depressive disorder of 15.7% for persons aged 15 to 24 years old (Blazer, Kessler, McGonagle, & Swartz, 1994).

suggests that the association between neuroticism and episode duration might be due to depressive rumination. That is, depressed patients with elevated levels of neuroticism are more likely to focus on their depressive symptoms, amplifying their dysphoric feelings and leading to more persistent episodes. If so, more rapid treatment gains might be achieved by directly modifying ruminative response styles, such as the proclivities to focus on symptoms, to introspect, to self-isolate, and to engage in self-blame.

Finally, we need to move beyond our heavy reliance on self-report measures of rumination. Although Nolen-Hoeksema (1994; Morrow & Nolen-Hoeksema, 1990) has developed clever experimental manipulations of rumination, self-reports are the only means currently available to assess a person's characteristic tendency to ruminate. Behavioral and information processing approaches need to be developed to assess individual differences in ruminative response style. In this context, measures that focus directly on individuals' actual ruminative behaviors and attentional processes would represent a significant advancement (cf. Gotlib, Gilboa, & Sommerfeld, in press). Likewise, experience-sampling methods, which use beepers or hand-held microcomputers to assess psychological and behavioral phenomenon close to the time of their actual occurrence, would also represent an improvement over the current methodology in this area.

In summary, the present findings suggest that ruminative response style is an important trait vulnerability characteristic to episodes of persistent dysphoria. Elevated levels of rumination were found in individuals with current dysphoria, as well as with previous episodes of dysphoria, when compared to persons who have never experienced such episodes. Further, individuals whose episodes lasted 2 weeks or longer reported greater rumination than did those whose episodes did not last a full 2 weeks, suggesting that rumination is associated with the duration of episodes of dysphoria. Finally, support was obtained for a model that posits that rumination accounts for the effects of gender and neuroticism on vulnerability to dysphoria. Importantly, these findings were not due to confounding between depressive symptoms and the self-report measure of ruminative response style.

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