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Rumination Predicts Heightened Responding to Stressful Life Events in Major Depressive Disorder and Generalized Anxiety Disorder

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Abstract

Although studies have documented heightened stress sensitivity in major depressive disorder (MDD) and generalized anxiety disorder (GAD), the mechanisms involved are poorly understood. One possible mechanism is the tendency to ruminate in response to stress. We used ecological momentary assessment to study ruminative thoughts following stressful events in 145 adults with MDD, GAD, comorbid MDD-GAD, or no psychopathology. Diagnosed individuals reported more event-related rumination than controls, even after adjusting for event stressfulness. Rumination was equally common in MDD and GAD and was especially severe among comorbid cases. More rumination immediately after the event predicted poorer affect, more maladaptive behavior, and more MDD and GAD symptoms at the next signal, even when pre-event levels of these variables were controlled. Rumination mediated, but did not moderate, the association of stress with affect and with symptoms. Stress-related rumination was more deleterious for diagnosed than healthy individuals, more intense for more severe clinical cases, and more persistent for cases with a greater temperamental vulnerability for emotional disorders. These results implicate rumination as a mechanism of stress sensitivity and suggest pathways through which it may maintain depression and anxiety in everyday life.

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Keywords

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Stressful life events have long been linked with emotional disorders. Depressed and anxious individuals not only experience greater life stress (Blazer, Hughes, & George, 1987; Kendler, Karkowski, & Prescott, 1998) but exhibit heightened sensitivity to stress, reacting more strongly than healthy individuals even to relatively minor stressors (Bale, 2006; Vrieze & Claes, 2009; Wichers et al., 2009). Stress sensitivity has been identified as a risk factor for the development and maintenance of emotional disorders (Mezulis, Funasaki, Charbonneau, &, Hyde, 2010; Morris, Ciesla, & Garber, 2010; Morris, Rao, & Garber, 2012; Siegrist, 2008). However, little is known about the mechanisms underlying stress sensitivity, nor the pathways through which they contribute to symptoms in daily life.

Rumination may be one mechanism linking stress with depression and anxiety (Brosschot, Gerin, & Thayer, 2006; Ciesla, Felton, & Roberts, 2011; Verkuil, Brosschot, Gebhardt, &Thayer, 2010). Rumination reflects a negative, repetitive style of thinking about present and past symptoms, loss, and failure (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Whereas initial conceptualizations of rumination emphasized its focus on symptoms and their causes and consequences, more recent elaborations have broadened the concept to include attention to negative life events that the individual has experienced (Alloy et al., 2000; Smith & Alloy, 2009). Moreover, while rumination originally was proposed as a causal and maintaining factor for depression, a growing body of research suggests that rumination may be equally associated with anxiety (e.g., Nolen-Hoeksema, 2000), underscoring its potential for illuminating the link between life stress and these frequently co-occurring disorders.

Findings from cross-sectional research support the presumed association between stress and rumination. Rumination is a common and spontaneous response to induced stress and is associated with heightened physiological responding, delayed recovery from the stressor, and increased reactivity to a subsequent upsetting event (Watkins, 2004; Watkins, Moberly, & Moulds, 2008; Zoccola, Dickerson, & Zaldivar, 2008; Zoccola, Quas, & Yim, 2010). Other research has paired rumination, in turn, with depression and anxiety. Rumination inductions increase depressed and anxious affect (Andrews & Borkovec, 1988; McLaughlin, Borkovec, & Sibrava, 2007). Additionally, spontaneous rumination is elevated in depressed and anxious individuals relative to controls following a laboratory stressor (Ruscio, Seitchik, Gentes, Jones, & Hallion, 2010), with higher levels of negative thought intrusions predicting a stronger stress response (Ruscio, Seitchik, Gentes, Jones, & Hallion, 2011).

Longitudinal studies further support a link between rumination and negative emotional outcomes following stress. Individuals who report using more ruminative responses around the time of a stressor report higher levels of depressive symptoms over the following weeks and months, even after accounting for pre-stressor levels of depressive symptoms (Nolen-Hoeksema, McBride, & Larson, 1997; Nolen-Hoeksema & Morrow, 1991; Nolen-Hoeksema, Parker, & Larson, 1994). Moreover, rumination mediates the longitudinal

relationship between stressful life events and symptoms of depression and anxiety in the community (Michl, McLaughlin, Shepherd, & Nolen-Hoeksema, 2013).

Despite their contributions, past studies have left important gaps in our understanding of rumination as a mechanism of stress sensitivity. Cross-sectional studies have typically answered between-persons questions (e.g., Is trait rumination associated with symptoms?) rather than within-person questions addressing mechanisms of action (e.g., Does an individual experience more symptoms following more intense rumination on a stressor?). Longitudinal studies that have examined within-person questions have tended to use lengthy follow-up periods (i.e., weeks or months) that lack the temporal resolution to illuminate rapidly unfolding processes in the wake of stressful events. Past studies have also focused either on stressors that can be created in the laboratory or on major events in respondents' lives (e.g., divorce, death of a loved one). We know much less about how people respond to day-to-day stressors, despite their far greater frequency of occurrence and their documented cumulative impact on mental health (Nolen-Hoeksema, Larson, & Grayson, 1999).

Ecological momentary assessment (EMA) has several unique advantages for addressing these gaps in the literature (Wenze & Miller, 2010). EMA is an ambulatory data collection method in which participants report on their experiences multiple times per day. A particular strength of EMA lies in its ability to examine processes as they unfold within the individual over time, making it ideal for studying pathways through which rumination may heighten sensitivity to stress. One pathway, suggested by experimental research (Andrews & Borkovec, 1988; McLaughlin et al., 2007), is that rumination directly increases the negative emotions and symptoms that are central to emotional disorders. Alternatively, rumination may heighten sensitivity through its influence on behavior. Theorists have speculated that rumination increases maladaptive behaviors such as withdrawal, inactivity, and avoidance that, in turn, exacerbate depression (Wisco & Nolen-Hoeksema, 2008). To date, no study has investigated the behavioral concomitants of rumination in daily life, despite their potential to explain how rumination operates and their value as intervention targets. In contrast to these direct effects, rumination may moderate or mediate the effect of the stressor itself. For example, theorists have suggested that rumination prolongs the impact of a stressor by keeping the stressor "alive"—and the individual activated—even after the stressor has ended (Brosschot et al., 2006; Verkuil et al., 2010).

Researchers are just beginning to tap the potential of EMA for studying these pathways, especially in clinical populations. In the few EMA studies of reactions to stressful events in major depressive disorder (MDD), depressed individuals reported more negative events and perceived their daily events as more stressful than nondepressed controls (Bylsma, Taylor-Clift, & Rottenberg, 2011; Thompson et al., 2012). Depressed individuals also exhibited a larger or more persistent negative emotional response to daily stressors than controls in some studies (Bylsma et al., 2011; Peeters, Nicolson, Berkhof, Delespaul, & de Vries, 2003). Even fewer studies have examined rumination in the context of stressful events. In the only EMA study on this topic to date, ruminative self-focus and negative affect (NA) were sampled eight times daily for one week in an unselected community sample, with participants recording in a paper diary any event that elicited negative emotions (Moberly & Watkins, 2008). Higher ruminative self-focus after a negative event was associated with

greater subsequent NA. In addition, high trait ruminators reported greater NA after negative events than low trait ruminators. In a second study that used daily diary methods, undergraduates rated at bedtime (a) their current mood and (b) the most unpleasant event of the day and their ruminative response to it (Genet & Siemer, 2012). Rumination moderated the effect of stress on mood, with stress predicting NA only when rumination was high. In both studies, rumination partially mediated the effect of stressful events on NA.

Remarkably, no EMA study has investigated ruminative responses to stress in depressed individuals. As a result, we know little about how often depressed individuals respond to stress with rumination, how severe it tends to be, and which clinical features predict who is most vulnerable to experiencing the most severe and persistent rumination after stress. Additionally, the specificity of rumination as a mechanism of stress sensitivity for depression is poorly understood. Emerging evidence that rumination may share an equally strong association with anxiety (Nolen-Hoeksema et al., 2008) implies that rumination may also be elevated in individuals with anxiety disorders. Generalized anxiety disorder (GAD) may be particularly useful for evaluating specificity, both because of its close relationship to MDD (Goldberg, Kendler, Sirovatka, & Regier, 2010) and because its cardinal feature of worry shares important similarities with rumination (Ehring & Watkins, 2008; Watkins, 2008). Determining whether depressed individuals are especially susceptible to rumination and its consequences, or whether levels of rumination are similar in GAD or even higher in comorbid cases, is needed to determine who should be offered rumination-focused interventions to curb reactivity to stress.

The present investigation used EMA to study ruminative responses to stressful events in the daily lives of persons with MDD, GAD, comorbid MDD-GAD, or no psychopathology. We hypothesized that diagnosed individuals would rate daily events as more stressful and would experience more frequent and severe rumination after stress than controls. We posed competing hypotheses about differences in rumination between MDD and GAD, with the traditional pairing of rumination with depression arguing for higher levels in MDD, but more recent evidence linking rumination with anxiety—and particularly with worry—suggesting similar elevations in MDD and GAD. A further possibility was that rumination would be highest in comorbid MDD-GAD, given the generally greater clinical severity and higher trait rumination of persons with co-occurring depression and anxiety (e.g., Nolen-Hoeksema, 2000). We expected that higher rumination levels after the stressor would be associated with poorer affect, more symptoms, and greater engagement in maladaptive behaviors, speculating that the magnitude of these associations would be larger in diagnosed than healthy individuals. In addition to any direct associations of rumination with affect, symptoms, and behavior, we hypothesized that rumination would interact with stressfulness in predicting these outcomes. Finally, we hypothesized that rumination would emerge as a mediator of the relationship between event stressfulness and post-stress outcomes.

Method

Participants

Participants were recruited from the Philadelphia community and from the student body of a private university. Eligibility was determined using the Anxiety Disorders Interview

Schedule–Lifetime Version (ADIS-IV-L; DiNardo, Brown, & Barlow, 1994). Individuals who were assigned a current, principal (most severe) *DSM-IV* diagnosis of MDD or GAD were eligible to participate, excluding those with current suicidal intent, psychosis, or substance-related disorders. Individuals with no current or past psychopathology were eligible for the control group.

Of 151 individuals who began the study, three withdrew due to time constraints, two had data lost due to malfunction of the electronic device, and one failed to return the device. The final sample consisted of an MDD group (n = 38) diagnosed with MDD but not GAD, a GAD group (n = 36) diagnosed with GAD but not MDD, a comorbid group (n = 38) diagnosed with both MDD and GAD, and a control group (n = 33) with no psychopathology. Past major depressive episodes were reported by a majority of the GAD group (58%), whereas past GAD was rare in the MDD group (6%). The groups did not differ in race-ethnicity, education, or marital status but did differ in age (MDD group older than the control group) and sex (GAD group more female than the comorbid group; see Table 1). To account for these differences, all multilevel models adjusted for age and sex.

Procedure

During the first session, participants were administered clinical interviews by a Master's- or Bachelor's-level diagnostician who had undergone extensive training and demonstrated high interrater agreement with the supervising psychologist. Interrater reliability for MDD (K = 0.88) and GAD (K = 1.00) diagnoses was high for a randomly selected subset of recorded interviews (n = 32) rated independently by a diagnostician blind to initial diagnoses. Diagnostic decisions and clinical severity ratings were finalized by the full assessment team following discussion of each case.

Eligible participants returned to the laboratory for an orientation session. They met individually with a research assistant and completed two full practice assessments. The seven-day sampling week began the morning after the orientation session. Participants carried an electronic device (Palm Pilot Z22) that signaled them with a tone eight times per day during the 12-hour period they identified as most convenient (typically 10 AM-10 PM). Following a time-stratified random sampling strategy, participants were signaled once at a random time in each 90-minute block, with the constraint that signals be separated by at least 20 minutes. Participants were able to delay a signal for one hour if they were entering a situation in which responding would be infeasible (e.g., a business meeting) or dangerous (e.g., while driving). This option could only be used to delay future signals; once a signal was delivered, reports not completed within 15 minutes were coded as missing.

At each signal, participants completed a two-part assessment. In the first part, participants rated their thoughts, feelings, behaviors, and symptoms at the moment they were signaled (Time 1, or T1). In the second part, they described the most significant negative or positive event that had occurred since the previous signal, operationalized for participants as the event that had the biggest impact on them. Participants rated characteristics of the event, then reported on the thoughts and feelings they experienced immediately after the event (Time of event, or T_E). Thus, T_E and T1 ratings were made at the same assessment, with T1

ratings reflecting the participant's current state and T_E ratings providing a retrospective account of an event occurring between 0 and 90 minutes earlier.

Participants were telephoned on day 2 of the sampling week to check adherence and address any problems. After completing the sampling week, participants returned the device, were debriefed, and were compensated for their participation.

Measures

EMA variables

Event-related variables—Participants rated the stressfulness of each T_E event on a Likert-type scale ranging from 0 (*not at all*) to 4 (*very much*). Events rated 2 (*moderately*) or higher on this scale were considered stressful events in analyses.

Participants also rated the extent to which they ruminated immediately after the event using the same 0–4 scale. Rumination was assessed by two items written to capture two facets of the construct. The first item (*I was dwelling on my mistakes, failures, or losses*) assessed perseverative thoughts about negative personal attributes involving themes that are central to rumination (Nolen-Hoeksema et al., 2008). The second item (*I kept thinking about something negative that has happened*) assessed perseverative thoughts about past negative events that are an important focus of rumination and distinguish it from worry (Nolen-Hoeksema et al., 2008; Smith & Alloy, 2009). Cronbach's alpha for the two items, averaged across events within individuals, was $\alpha = .89$. A single momentary rumination variable, constructed by averaging the two items for each event, served as the measure of rumination in all analyses. Its validity was supported by strong associations (both $r = .57$) with the Ruminative Responses Scale (Nolen-Hoeksema & Morrow, 1991) and the Rumination scale of the Rumination-Reflection Questionnaire (Trapnell & Campbell, 1999).

Outcomes—Participants rated their emotions immediately after the T_E event and at each signal using three NA (*sad, anxious, dissatisfied with myself*) and three PA (*happy, proud, determined*) terms. Each emotion was rated on a 0–4 scale. Item triplets were averaged at each time point to form momentary NA ($\alpha = .79$-$.81$) and PA ($\alpha = .76$–$.78$) variables. Trait NA, assessed by the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988), correlated highly with momentary NA immediately after the event ($r = .60$) and at the signal ($r = .59$). Similarly, trait PA on the PANAS correlated highly with momentary PA after the event ($r = .59$) and at the signal ($r = .62$), providing evidence of validity.

At each signal, participants were shown all *DSM-IV* symptoms of MDD and GAD and were asked to check the box next to each symptom that they were currently experiencing. Symptoms were presented four at a time; the exception was suicidal ideation, which was presented alone and, if endorsed, triggered the display of emergency referrals. These dichotomous symptom ratings were summed into MDD (15 items) and GAD (9 items) composites that correlated strongly with ADIS-IV-L clinical severity ratings for MDD ($r = .57$) and GAD ($r = .51$), respectively.

At each signal, participants also rated the extent to which they were currently engaging in each of four behaviors: social withdrawal (*distancing or isolating myself from others*), inactivity (*unable to make myself get up and do things*), behavioral avoidance (*avoiding doing something difficult or unpleasant*), and cognitive avoidance (*avoiding thinking about something difficult or unpleasant*). These behaviors were chosen for their conceptual links with rumination and related forms of perseverative thought and their hypothesized roles as mechanisms through which rumination may influence depression (Nolen-Hoeksema et al., 2008). Each behavior was rated on a separate 0–4 scale.

Clinical predictors—Clinical characteristics assessed at the first session, prior to the sampling week, were examined as predictors of participants' ruminative response to stress. Interviewers rated the overall severity of MDD (ICC = 0.97) and GAD (ICC = 0.97) for each participant using a 0–8 scale. Interviewers also rated the severity of depression and anxiety using the Hamilton rating scales (Hamilton, 1959, 1960; ICC = 0.96–0.97). Measures of the course of MDD and GAD, derived from the ADIS-IV-L, included current episode duration among current cases and age of onset of the first episode, history of single vs. recurrent episodes, and total months in episode over the lifetime (lifetime persistence) among lifetime cases.

Other clinical predictors included the number of current and past comorbid disorders (out of 13 anxiety, mood, and substance-related disorders other than MDD and GAD assessed by the ADIS-IV-L) and history of mental health treatment (pharmacotherapy or psychotherapy). Finally, trait NA and PA, two well-established symptom and vulnerability dimensions for depressive and anxiety disorders (see Brown & Barlow, 2009), were assessed using the PANAS.

Statistical Analyses

In addition to comparisons of means by diagnostic group using SPSS v20 (IBM corporation, 2011), we performed multilevel analyses using Hierarchical Linear and Nonlinear Models 6.03 (Raudenbush, Bryk, & Congdon, 2005). EMA observations were nested within individuals using a two-level model. All models adjusted for age and sex. One set of models examined associations between variables assessed at the same signal; these included event-related variables rated retrospectively (T_E) and current outcomes rated at the signal (T1). A second set of models examined time-lagged associations in which event-related variables (T_E) were used to explain variance in outcomes rated two signals after the event (T2). To test for moderation by diagnostic group, we first constructed three dichotomous variables to compare the MDD, GAD, and comorbid groups to the control group, while in subsequent models we constructed three dichotomous variables to compare the three clinical groups to each other. The three clinical groups did not differ in any moderation analyses other than those noted specifically below. As one illustration of the analyses performed, the relationship of post-event rumination with behavioral inactivity can be described by the following equation:

$$\text{inactivity}_{ij} = \beta_{0j} + \beta_{1j}(\text{rumination}_{ij} - \text{rumination}_{.j}) + r_{ij}$$

where $inactivity_{ij}$ is the inactivity rating for individual *j* at observation *i*; intercept β_{0j} is the expected inactivity rating when the rumination score is equal to the individual's average rumination score, $rumination_{.j}$ (scores are examined relative to the individual's own mean by centering around $rumination_{.j}$); slope β_{1j} is the expected change in inactivity for a unit deviation from the average rumination score for individual *j*; $rumination_{ij}$ is the rumination score following a stressful event at observation *i* for individual *j*; and r_{ij} is the error term associated with observation *i* for individual *j*. These Level 1 intercepts and slopes for individual *j* can then be predicted at Level 2 by the following equations:

$$\beta_{0j}=\gamma_{00}+\gamma_{01}(\text{Age})+\gamma_{02}(\text{Sex})+\gamma_{03}(\text{MDD})+\gamma_{04}(\text{GAD})+\gamma_{05}(\text{Comorbid})+U_{0j}$$

$$\beta_{1j}=\gamma_{10}+\gamma_{11}(\text{Age})+\gamma_{12}(\text{Sex})+\gamma_{13}(\text{MDD})+\gamma_{14}(\text{GAD})+\gamma_{15}(\text{Comorbid})+U_{1j}$$

where γ_{00} is the average intercept across individuals; coefficients γ_{01} through γ_{05} indicate the expected change in the average intercept attributable to between-person variance in age, sex, or diagnostic status; u_{0j} is the unique increment to the intercept associated with individual *j*; γ_{10} is the average regression slope across individuals (i.e., the pooled within-person regression coefficient); coefficients γ_{11} through γ_{15} indicate the expected change in the average regression slope attributable to between-person variance in age, sex, or diagnostic status; and u_{1j} is the unique increment to the slope associated with individual *j*. This model subsequently was repeated to include a covariate adjusting for the status of behavioral inactivity at the previous observation ($inactivity_{i-1}$). Our main focus in this investigation was on the average within-person regression coefficients (γ_{10}) and their moderation by diagnostic group (γ_{13}- γ_{15}). We supplemented these analyses with tests of within-person mediated effects using procedures detailed in Bauer, Preacher, and Gil (2006) and Bolger and Laurenceau (2013) and performed in Mplus v7.2 (Muthén & Muthén, 1998–2012).

Results

Preliminary Analyses

Participants were administered 7,988 pre-programmed signals over the sampling week and submitted 5,724 completed assessments, corresponding to a mean response rate per participant of 72% (SD = 12.7, range 41% – 98%). Prior validation studies have found similar response rates with depressed and anxious samples (Husky et al., 2010; Johnson et al., 2009). Missing data were addressed in the multilevel models by calculating within-person coefficients before pooling across individuals. Importantly, the number of assessments completed did not differ by diagnostic group or by any clinical predictor. Neither assessment completion rates nor event-related rumination levels differed by time of day or day of study.

Frequency and Severity of Stressful Life Events in MDD and GAD

Participants reported a total of 5,700 events during the sampling week. Events ranged in stressfulness from 0 (*not at all*) to 4 (*very much*), with the average event rated as mildly stressful ($M = 1.08$, $SD = 1.22$). Mean stress levels were highest in the comorbid group ($M = 1.36$), lower in the GAD group ($M = 1.12$), and lowest in the control group ($M = 0.49$), all $\gamma > 0.295$, all $p < .017$, with the MDD group ($M = 1.32$) differing marginally from the GAD group.

In total, 34% of events ($n = 1,965$) were rated as moderately or more stressful, meeting our operational definition of a stressful event. On average, stressful events constituted 45% of all events reported by comorbid participants, 42% of events among MDD participants, 36% of events among GAD participants, and 15% of events among controls.

Ruminative Responses to Stress in MDD and GAD

Rumination was a common response to stress in all groups, but was reported more often by depressed and anxious participants (80% to 87%) than by controls (55%). Mean levels of rumination immediately after the stressor were highest for comorbid cases ($M = 1.93$), lower for GAD cases ($M = 1.48$), and lowest for controls ($M = 0.59$), all $\gamma > 0.504$, all $p < .002$, with MDD cases differing marginally from both other clinical groups ($M = 1.70$). Group differences in rumination levels remained significant even after adjusting for event stressfulness, all $\gamma > 0.645$, all $p < .001$.

To better understand individual differences in the tendency to respond to stress with rumination, we used clinical features assessed prior to the sampling week to predict ruminative experiences following stressful events. In a first set of models predicting severity of rumination, each feature was used to explain the average level of rumination immediately after the stressor (T_E). In a second set of models predicting persistence of rumination, each feature was tested as a moderator of the association between rumination level immediately after a stressful event (T_E) and rumination level two signals after that event (T2).

The clinical characteristics considered here more consistently predicted the severity than the persistence of rumination after stress (see Table 2). Rumination was more severe among individuals with more severe depression and anxiety. Rumination was also higher among individuals with more extensive comorbidity, with a history of mental health treatment, and with higher trait NA and lower trait PA. Measures of clinical course were unrelated to the magnitude of the ruminative response to stress. By contrast, rumination was more persistent after stress among individuals with a more adverse course of GAD (i.e., an earlier first onset) as well as those with greater temperamental vulnerability (i.e., high trait NA). Rumination was less persistent among individuals with a larger number of remitted comorbid disorders.

Is Ruminating on Stressful Events Associated with Adverse Outcomes?

In the next set of models, the intensity of rumination experienced immediately after the stressor was used to predict the level of each affective, symptom, and behavioral outcome (see Table 3, left column). Rumination was associated with affect concurrently (T_E) and at

the signal (T1), with more robust associations observed for negative than positive affect. Greater rumination predicted more symptoms of MDD and GAD at the signal (T1). It also predicted higher levels of social withdrawal, inactivity, and behavioral avoidance—but not cognitive avoidance—at the signal (T1). Lastly, rumination predicted elevations in NA two signals after the stressor (T2). Many of these associations were moderated by diagnostic status, with rumination more strongly associated with adverse outcomes in the clinical groups than controls, but no differences in the effects of rumination observed between clinical groups.

To evaluate rumination as a predictor of change in these outcomes, we reran each model controlling for the level of the outcome variable at the signal preceding the event (T0) as well as for age and sex (see Table 3, right column). Those who ruminated more in the aftermath of the stressor experienced larger increases in NA and larger decreases in PA, both concurrently (T_E) and at the signal (T1). Those who ruminated more also experienced a larger upsurge in MDD and GAD symptoms and increased difficulty engaging in motivated activity at the signal (T1). In the total sample, rumination was not a significant predictor of change in any outcome two signals after the stressor (T2), all $\gamma < 0.355$, all $p > .076$. However, within the clinical groups, more rumination after the stressor predicted greater deterioration on many of these measures.

Does Rumination Moderate the Association of Stress With Adverse Outcomes?

In addition to examining the direct associations of rumination with affect, symptoms, and behavior, we tested whether rumination moderated the associations of event stressfulness with these outcomes. In a dataset including all 5,700 events, we evaluated perceived stressfulness of the event, severity of post-event rumination, and their interaction as predictors of each outcome. After controlling for age, sex, and pre-event (T0) level of the outcome variable, rumination did not moderate the effect of stress on any outcome, all $\gamma < 0.218$, all $p > .051$.

Does Rumination Mediate the Association of Stress With Adverse Outcomes?

In the same dataset of 5,700 events, we evaluated rumination as a within-person mediator of the relationship between event stressfulness and post-stress outcomes. Each model controlled for age, sex, and pre-event (T0) level of the outcome variable. Models providing evidence of mediation were rerun including group contrasts to test whether the mediating role of rumination varied by diagnostic group.

We first examined concurrent relationships between the perceived stressfulness of an event (T_E) and the rumination and affect experienced in its immediate aftermath (T_E). There were strong within-person associations between stressfulness and rumination ($\gamma = 0.430$, $SE = 0.024$, $p < .001$) and between rumination and NA immediately after the event ($\gamma = 0.488$, $SE = 0.021$, $p < .001$). As hypothesized, rumination mediated the association between stressfulness and NA (mediated effect = 0.227, $SE = 0.017$, $p < .001$). To provide a sense for the size of the mediated effect, we calculated proportion mediated effect (PME; MacKinnon, Fairchild, & Fritz, 2007), a ratio of the mediated effect over the total effect of stressfulness on NA. Rumination mediated 62% of the association between event stressfulness and post-

event NA. Similarly, rumination was associated with PA immediately after the event (γ = −0.235, *SE* = 0.024, $p < .001$) and mediated 64% of the association between stressfulness and PA (mediated effect = −0.105, *SE* = 0.013, $p < .001$).

Follow-up multilevel models revealed that the association between event stressfulness and rumination was stronger for all three clinical groups compared to controls, all $\gamma > 0.260$, all $p < .001$. Rumination, in turn, was more strongly associated with NA for the MDD group than for controls ($\gamma = 0.141$, *SE* = 0.066, $p = .032$), with the GAD and comorbid groups exhibiting a similar pattern that did not reach statistical significance (both $\gamma < 0.124$, both $p > .082$). The association between rumination and PA, in contrast, did not differ by diagnostic group, all $|\gamma| < 0.075$, all $p > .485$.

Next, we evaluated rumination (T_E) as a mediator of the relationship between event stressfulness (T_E) and outcomes at the signal (T1). Greater rumination in the aftermath of the event was associated with higher NA ($\gamma = 0.399$, *SE* = 0.022, $p < .001$) and lower PA (γ = −0.185, *SE* = 0.020, $p < .001$) at the signal. Further, rumination mediated the relationship of stressfulness with both affect variables (both mediated effects > 0.085 in absolute value, both $p < .001$), accounting for 72% of the association with NA and 82% of the association with PA at the signal. Group moderated the link between stressfulness and rumination (all $\gamma > 0.255$, all $p < .001$) but did not moderate the link between rumination and either affective outcome (all $\gamma < 0.120$, all $p > .141$).

Rumination (T_E) also emerged as a mediator of the relationship between stressfulness (T_E) and symptoms reported at the signal (T1). Higher levels of rumination after the event predicted more symptoms of MDD at the signal ($\gamma = 0.609$, *SE* = 0.061, $p < .001$); rumination explained 88% of the association between event stressfulness and momentary depression symptoms (mediated effect = 0.286, *SE* = 0.034, $p < .001$). Similar results were obtained for GAD symptoms, with rumination predicting more symptoms at the signal (γ = 0.513, *SE* = 0.046, $p < .001$) and accounting for 62% of the association between stressfulness and symptoms (mediated effect = 0.236, *SE* = 0.025, $p < .001$). The magnitude of both mediation effects differed by group, with stronger relationships observed between stress and rumination, and between rumination and symptoms, for clinical participants than controls, all mediated effects > 0.249, all $p < .033$.

Event stressfulness did not predict participants' self-reported behaviors at the signal (T1), nor did it significantly predict any outcomes at the following signal (T2). Consequently, mediation analyses were not performed for these outcomes.

Discussion

The present study used EMA to investigate rumination as a mechanism of stress sensitivity for depression and anxiety. Rumination was a common response to daily stressors among persons with MDD and GAD. It was more detrimental for diagnosed than healthy individuals and more intense for individuals with more severe psychopathology. Rumination predicted a variety of adverse outcomes after stress and mediated the relationship of stress with symptoms of both disorders. These findings offer new insights into the experience of

rumination among depressed and anxious individuals and shed light on how ruminative thoughts may contribute to psychopathology in daily life.

The present study revealed multiple pathways through which rumination may heighten sensitivity to stress. First, rumination was associated with deteriorations in affect and increases in depression and anxiety symptoms following the stressful event. Its robust prediction of emotional reactivity to events that were, on average, mildly stressful suggests that rumination may sensitize individuals even to minor stressors, perhaps helping to explain how MDD and GAD persist in the absence of major stressors. Additionally, rumination mediated the association of event stressfulness with emotion and symptom outcomes. These findings replicate prior EMA studies in which rumination partially or fully mediated the association of stressful life events with negative and positive affect (Genet & Siemer, 2012; Moberly & Watkins, 2008; Peeters et al., 2003). Our study adds to this literature by revealing a pathway from stressful events through rumination to MDD and GAD symptoms, and by showing that this pathway is stronger among individuals diagnosed with MDD and GAD than among healthy controls.

Second, our results suggest that rumination may heighten sensitivity indirectly through its influence on behavior. Rumination was associated with social withdrawal, inactivity, and behavioral avoidance in the wake of stressful events. Rumination also predicted changes in activity levels following stress, with more intense rumination predicting increased difficulty engaging in the very behaviors that could improve the stressful situation or repair mood. The possibility that rumination intensifies or prolongs the impact of stress by inhibiting instrumental action is consistent with an experiment wherein dysphoric individuals, after being induced to ruminate, reported decreased willingness to engage in pleasant activities despite expecting these events to be enjoyable (Lyubomirsky & Nolen-Hoeksema, 1993). The present study is the first to examine this and other behavioral outcomes of rumination in the lives of depressed and anxious individuals. Our results align with observations by Nolen-Hoeksema and colleagues (2008) that rumination serves the function of establishing that one’s situation is hopeless, justifying withdrawal, inaction, and avoidant coping. We found less evidence linking rumination with cognitive avoidance, consistent with the hypothesis that cognitive avoidance is more a function of worry than of rumination (Nolen-Hoeksema et al., 2008) but inconsistent with the findings of a daily diary study in which cognitive avoidance predicted rumination in healthy adolescents (Dickson, Ciesla, & Rielly, 2012). Further investigation of the behavioral concomitants of rumination—particularly testing whether behaviors predict symptoms, and mediate the relationship of rumination with symptoms—would aid in identifying mechanisms of action through which rumination influences anxiety and depression. This could, in turn, inform the design of behavioral as well as cognitive interventions for disrupting the cycle of negative thinking and promoting successful coping after stress.

The present study revealed that, even among individuals with no history of psychopathology, greater rumination after stress predicts poorer outcomes. However, compared with healthy controls, depressed and anxious individuals appear to be doubly vulnerable to the effects of rumination. Not only did diagnosed participants report more frequent and severe rumination, but rumination was associated more strongly with adverse

emotional, behavioral, and symptom outcomes in these participants than controls. Contrary to traditional views of rumination as particularly relevant to depression, ruminative responses to stress were similar in MDD and GAD participants, were predicted by clinical measures of anxiety as well as depression, and were predictive of increases in GAD as well as MDD symptoms. These results provide support for an emerging view of rumination (Nolen-Hoeksema & Watkins, 2011), and of perseverative thought more broadly (Ehring & Watkins, 2008), as a transdiagnostic rather than a disorder-specific process. Our observation that rumination levels were highest in the comorbid group, even after accounting for higher perceived stress in this group, further hinted that the tendency to respond to stress with negative, repetitive thinking may constitute a mechanism of comorbidity for MDD and GAD (cf. Ruscio et al., 2011). Collectively, these findings suggest that patients with GAD, as well as those with MDD, may benefit from interventions targeting rumination (e.g., Watkins et al., 2011) and that comorbid cases may be especially good candidates for these interventions.

The current findings should be interpreted within the context of several study limitations. One limitation was that events were rated from memory, 0 to 90 minutes after they occurred, introducing the potential for retrospective recall bias. Although this approach to sampling events is far from perfect, the potential for bias is still greatly reduced compared to traditional longitudinal designs. Moreover, the alternative approach of reporting on events as they occur (i.e., event-contingent reporting) has its own limitations, especially for research on stress. Relying on participants to initiate reports of stressful events runs the risk that differential stress sensitivity will lead to bias in the detection and reporting of stressors. By prompting all participants to engage in a memory search for the most significant event since the last signal, we sought to reduce the risk of differential reporting by group as well as the risk that more minor stressors would go underreported. A related limitation was that T_E and T1 ratings were made at the same sampling occasion. This suggests the need for caution in interpreting event-related variables as temporally preceding, and therefore potentially causing, experiences measured at the time of the signal. To enhance separation, we had participants rate their current experiences before rating the earlier event and their reactions to it, and we examined associations with outcomes at T2 as well as T1, although the lengthy interval between T_E and T2 may have weakened our ability to detect proximate consequences of rumination. Investigating the typical duration of rumination episodes would aid in designing future studies that better separate predictors and outcomes while remaining sensitive to the potentially brief effects of minor stressors.

A further limitation of our study was that stress was measured via participant reports rather than through independent, contextual ratings of objective event severity. This made it difficult to distinguish true sensitization from proportionate responses to objectively greater stress. We demonstrated that participants with MDD and GAD ruminated more than healthy participants at the same level of perceived stress, providing evidence of sensitization and suggesting that subsequent findings could be construed as consequences of this sensitization. However, more definitive results await the validation of objective rating systems that are appropriate for, and sensitive to, the minor stressors that predominate in the everyday environment. A related challenge concerns the feasibility of acquiring detailed event descriptions or extensive response ratings using an EMA approach. For example, we

assessed rumination using two items which, although yielding a reliable composite that correlated highly with trait measures, could not capture all features that may moderate the sensitizing influence of rumination (e.g., abstract versus concrete processing of the stressor; content of accompanying self-beliefs; Watkins et al., 2008). The brief assessments necessitated by EMA represent a limitation of the study. These must be weighed against unique strengths of EMA, including the repeated assessment of stressors and responses in the natural environment, close in time to their occurrence.

These limitations highlight the importance of a multimethod approach to studying stress sensitivity, especially in clinical populations that are known to evidence distorted appraisals of life events (Espejo, Hammen, & Brennan, 2012). EMA studies, which investigate real-world stressors in the natural environment, and laboratory experiments, which administer standardized stressors under controlled conditions, yield complementary lines of evidence about the nature of stress and stress responding. Harnessing the strengths of both methods, and seeking convergence across them, may yield the most rapid advances in understanding how mechanisms such as rumination contribute to stress sensitivity in psychopathology.

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Table 1

Demographic and Clinical Characteristics of the Sample by Group

Variable	Control (*n* = 33)	GAD (*n* = 36)	MDD (*n* = 38)	Comorbid (*n* = 38)
Demographic characteristics				
Age*	28.61 (10.42)^{a}	31.62 (9.24)^{a},b	36.38 (12.33)b	33.60 (11.35)^{a},b
% Female*	66.7^{a},b	83.3^{a}	71.1^{a},b	52.6b
% Caucasian	54.5	63.9	54.1	56.8
Marital Status				
Never married	75.0	47.2	60.5	71.1
Married or cohabiting	15.6	44.4	26.3	18.4
Previously married	9.4	8.3	13.2	10.5
Education				
High school or lower	6.1	8.3	10.8	10.5
Some college	48.5	27.8	29.7	31.6
College degree or higher	45.5	63.9	59.5	57.9
Clinical characteristics				
GAD severity***	0.54 (0.91)^{a}	4.89 (0.66)b	3.99 (1.60)c	4.95 (1.02)b
MDD severity***	0.18 (0.53)^{a}	2.19 (1.13)b	5.16 (0.82)c	5.16 (0.74)c
Current comorbid disorders^{a}***	0.00 (0.00)^{a}	0.81 (0.82)b	0.90 (0.96)b	1.33 (1.36)b
Past comorbid disorders^{a}**	0.00 (0.00)^{a}	0.88 (1.56)b	0.77 (1.10)b	1.00 (1.12)b

Note. GAD = generalized anxiety disorder; MDD = major depressive disorder. *M (SD)* are presented for dimensional variables; all other values represent percentages. Values in the same row that do not share subscripts differ at $p < .05$.

^{a}Number of anxiety, mood, and substance-related disorders, excluding GAD and MDD.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

Table 2

Clinical Features Predicting the Severity and Persistence of Post-Stress Rumination Across Groups

	Severity		Persistence	
Predictor	**γ**	***SE***	**γ**	***SE***
Depression features				
HAM-D	1.175	0.126***	0.075	0.109
MDD clinical severity	0.192	0.025***	0.021	0.018
MDD current episode duration	−0.001	0.001	−0.001	0.001
MDD age of onset	0.002	0.008	−<0.001	0.004
MDD recurrence	−0.067	0.161	−0.006	0.098
MDD lifetime persistence	−<0.001	0.001	<0.001	0.001
Anxiety features				
HAM-A	0.822	0.112***	0.037	0.084
GAD clinical severity	0.191	0.028***	0.019	0.021
GAD current episode duration	−<0.001	0.001	<0.001	<0.001
GAD age of onset	0.003	0.008	−0.010	0.003**
GAD recurrence	−0.087	0.143	−0.015	0.087
GAD lifetime persistence	<0.001	0.001	<0.001	<0.001
Other clinical features				
Current comorbid disorders	1.004	0.302**	0.103	0.143
Past comorbid disorders	0.800	0.281**	−0.477	0.149**
Current mental health treatment	0.163	0.124	−0.044	0.070
Lifetime mental health treatment	0.304	0.134*	0.019	0.072
PANAS – NA	0.492	0.062***	0.091	0.039*
PANAS – PA	−0.379	0.072***	−0.106	0.058

Note. HAM-D = Hamilton Rating Scale for Depression; MDD = major depressive disorder; HAM-A = Hamilton Anxiety Rating Scale; GAD = generalized anxiety disorder; PANAS = Positive and Negative Affect Schedule; NA = Negative Affect; PA = Positive Affect.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

Table 3

Rumination Predicting Level of, and Change in, Outcomes Following Stressful Events

	Level of Outcome			Change in Outcome		
Outcome	**γ**	***SE***	**Moderation by Group**	**γ**	***SE***	**Moderation by Group**
Immediately after event (T_E)						
Negative affect	0.649	0.073**	MDD, GAD > Con	0.571	0.102**	-
Positive affect	−0.301	0.115*	-	−0.289	0.119*	-
One signal after event (T1)						
Negative affect	0.524	0.097***	MDD, GAD > Con	0.497	0.112***	MDD, GAD, Com > Con
Positive affect	−0.327	0.072***	-	−0.289	0.090**	-
MDD symptoms	0.850	0.256**	MDD, GAD, Com > Con	0.759	0.258**	MDD, GAD, Com > Con
GAD symptoms	0.806	0.181***	MDD, GAD, Com > Con	0.654	0.218**	MDD, GAD, Com > Con
Social withdrawal	0.277	0.113*	MDD, GAD, Com > Con	0.187	0.142	-
Inactivity	0.270	0.092**	-	0.240	0.105*	-
Behavioral avoidance	0.195	0.095*	-	0.213	0.132	-
Cognitive avoidance	0.030	0.105	-	−0.033	0.132	-
Two signals after event (T2)						
Negative affect	0.264	0.119*	MDD, GAD > Con	0.226	0.127	-
Positive affect	−0.064	0.073	MDD, GAD > Con	−0.052	0.119	GAD, Com > Con
MDD symptoms	0.371	0.253	MDD, Com > Con	0.222	0.267	MDD, Com > Con
GAD symptoms	0.399	0.239	MDD > Con	0.354	0.244	MDD, GAD, Com > Con
Social withdrawal	0.148	0.160	-	0.050	0.191	-
Inactivity	0.099	0.124	-	0.131	0.165	-
Behavioral avoidance	−0.011	0.147	MDD, Com > Con	−0.014	0.159	MDD, GAD, Com > Con
Cognitive avoidance	0.196	0.150	MDD, GAD > Con	0.078	0.196	MDD > Con

Note. MDD = major depressive disorder; GAD = generalized anxiety disorder; Com = comorbid MDD and GAD; Con = control. T_E and T1 outcomes were assessed at the same sampling occasion. Models predicting level of outcome include age and sex as covariates. Models predicting change in outcome include age, sex, and prior level of the outcome variable (at signal T0) as covariates.

* $p < .05$.

** $p < .01$.

***$p < .001$.