BRIEF REPORT

Brooding rumination and cardiovascular reactivity to a laboratory-based interpersonal stressor

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Abstract

There is a well-known link between stress and depression, but diathesis-stress models suggest that not all individuals are equally susceptible to stress. The current study examined if brooding rumination, a known risk factor for depression, influences cardiovascular reactivity to a laboratory-based interpersonal stressor. Sixty-five women watched a baseline video and were exposed to an interpersonal stressor while high frequency heart rate variability (HRV) was collected. We found that women who endorsed higher levels of brooding rumination exhibited greater HRV withdrawal from baseline to stressor, an effect that was maintained when we controlled for levels of depression. This physiological vulnerability, when combined with high levels of stress, may be one mechanism underlying how brooding rumination increases depression risk.

Descriptors: High frequency heart rate variability (HF-HRV), Brooding rumination, Interpersonal stress, Diathesis-stress, Depression

Psychological stressors can lead to distinct physiological responses involving multiple biological systems (e.g., autonomic nervous system, hypothalamic-pituitary-adrenal [HPA] axis; Chida & Hamer, 2008). Repeated physiological reactivity in response to psychological stress has been associated with a variety of negative health consequences, including risk for major depressive disorder (MDD; cf. McEwen, 2003). Corroborating these findings, there is evidence from psychosocial studies showing a robust link between stressful life events and risk for depression (for a review, see Monroe, Slavich, & Georgiades, 2014). However, evidence from diathesis-stress models of depression suggests that not all individuals are equally susceptible to stress. These models propose that individuals with certain vulnerabilities are more likely to experience heightened reactivity following stressful life events, placing them at increased risk for later depression (e.g., Ingram & Price, 2001). This suggests there may be stable, individual characteristics that predict who is at greatest risk for depression.

Among risk factors for depression, rumination is a particularly well-studied vulnerability that also has strong links to stress reactivity (for a review, see Nolen-Hoeksema, Wisco, & Lyobomirsky, 2008). According to the response styles theory (Nolen-Hoeksema et al., 2008), the tendency to ruminate, or passively contemplate the causes and consequences of one's negative mood, contributes to the development and maintenance of depression. Supporting this theory, there is evidence that rumination predicts new MDD onsets in the context of high life stress (e.g., Abela & Hankin, 2011). This suggests that rumination may moderate the link between stress and depression, such that individuals high in trait rumination, compared to those low in trait rumination, are more likely to have heightened responses to stress, which then places them at greater risk for future depression. However, less is known about why ruminators are at such increased risk for depression following stressful events.

One hypothesis is that rumination increases biological reactivity to stress, which may put greater wear and tear on physiological systems integral for healthy emotional functioning (McEwen, 2003). Some studies have shown that high levels of rumination are associated with increased cortisol reactivity to stress (Roger & Najarian, 1998; Zoccola, Quas, & Yim, 2010). However, other studies show rumination is associated with blunted cortisol reactivity (Zoccola, Dickerson, & Zaldivar, 2008) or find no relation between rumination and cortisol reactivity (Kuehner, Huffziger, & Liebsch, 2008; Young & Nolen-Hoeksema, 2001). One potential explanation for these mixed findings is methodological differences in the measurement of cortisol. For example, measures of cortisol output depend on the time of day in which cortisol is measured, the type of stressor, and the proximity of the stressor (Kudielka, Gierens, Hellhammer, Wust, & Schlotz, 2012). Given these concerns, future research is needed to better understand the biological mechanisms underlying rumination and stress response.

Studies utilizing cardiovascular measures of stress reactivity may help elucidate the link between rumination and heightened biological reactivity. Phasic changes in heart rate variability (HRV)

may be well suited to examine the link between rumination and stress reactivity. Decreases in HRV are thought to reflect autonomic response to stress as cardiac vagal control is withdrawn and the sympathetic nervous system is activated. Research suggests that greater HRV withdrawal (i.e., reduction of high frequency HRV) reflects greater stress reactivity (Brindle, Ginty, Phillips, & Carroll, 2014). Providing initial evidence for a link between rumination and heightened cardiovascular reactivity, one study showed higher levels of rumination were associated with greater HRV withdrawal in a task where children read vignettes designed to elicit sadness (Borelli, Hilt, West, Weekes, & Gonzalez, 2014). Although this emerging evidence linking rumination to increased cardiovascular stress reactivity is promising, additional research is needed to examine whether rumination is also associated with greater cardiovascular reactivity in adults.

Therefore, the goal of the current study was to examine the link between trait rumination, specifically brooding rumination (cf. Treynor, Gonzalez, & Nolen-Hoeksema, 2003), and cardiovascular reactivity to a laboratory-based interpersonal stressor in a sample of adult women. Given the significant gender differences in rumination and depression (e.g., Nolen-Hoeksema et al., 2008), we chose to focus exclusively on women. We predicted that women who endorsed higher levels of brooding rumination would exhibit greater HRV withdrawal to the stressor. Additionally, we sought to ensure that the results were not driven by the presence of current depression. Therefore, we predicted that these relations would be maintained even after statistically controlling for the influence of both baseline depressive symptom levels and mood reactivity to the stressor.

Method

Participants

Sixty-five female undergraduates participated in exchange for partial course credit. Participants ranged in age from 18 to 25 years old, and 66% were Caucasian.

Measures

Questionnaires. Participants completed the Ruminative Response Scale (RRS; Treynor et al., 2003), a self-report questionnaire assessing the frequency with which ruminative thoughts or behaviors occur when the person is feeling sad, down, or depressed. For the current study, we focused on the 5-item brooding subscale ($\alpha = .85$; $M = 9.83$, $SD = 3.60$). To measure depressive symptoms, participants completed the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996), a 21-item questionnaire that assesses the severity of current depressive symptoms in the past 2 weeks ($\alpha = .92$; $M = 11.06$, $SD = 8.91$).

Interpersonal stressor. Participants underwent a social rejection induction adapted from previous studies (Robins, 1988). In this induction, participants were asked to listen to a series of described events and to imagine themselves in that situation. An audio recording described four rejection scenarios that happen over the course of one day: (1) having another woman answer their significant other's phone, (2) seeing their significant other in public with another woman, (3) overhearing two friends criticizing the participant's appearance and personality, and (4) overhearing these friends speculating that the participant's significant other is unfaithful. Two versions of the audiotape were used, depending on the reported sexual orientation of the participant (i.e., the signifi-

cant other was described as a male for heterosexual participants and as a female for homosexual participants). This social rejection induction has induced significant increases in negative affect and physiological reactivity in previous research (Dixon-Gordon, Chapman, Lovasz, & Walters, 2011; Robins, 1988).

State mood ratings. A visual analog scale (VAS) was used to measure state sadness at two time points: following a 5-min baseline nature video and immediately after the interpersonal stressor. Participants marked how they were feeling between *very happy* to *very sad* on a scale measuring 100 mm, with higher scores indicating greater state sadness. In prior studies, the VAS has demonstrated excellent reliability and validity (Killgore, 1999).

Heart rate variability. Electrocardiogram (ECG) data were obtained using a Biopac MP150 wireless system and recorded with Acqknowledge v4.2 software (Biopac Systems, Inc., Santa Barbara, CA). ECG was recorded via a standard three-electrode (lead II) setup and sampled at 1000 Hz. MindWare HRV 3.0.12 was used to inspect, transform, and analyze the ECG signal. Data were visually inspected for artifacts (e.g., an unusual R-R interval), and the experimenter corrected artifacts manually. After inspection, a power spectral analysis was done sequentially with a fast Fourier transform. Finally, the power density in the high frequency (HF; .15–.50 Hz) band of HRV was calculated for each 60-s section, consistent with recommendations by the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (1996). HRV was calculated by averaging the 60-s sections from each task, resulting in separate HRV averages for the 5-min baseline nature video and the 3-min interpersonal stressor.

Procedure

Upon arrival at the laboratory, participants were asked to provide informed consent. Next, participants completed a series of questionnaires. Finally, participants watched a 5-min baseline nature video featuring landscape scenes from Olympic National Park and completed the 3-min interpersonal stressor.

Results

First, paired samples *t* tests revealed that participants' HRV significantly decreased from baseline ($M = 6.90$, $SD = 1.29$) to the interpersonal stressor ($M = 6.74$, $SD = 1.16$), $t(64) = -2.75$, $p = .01$. Similarly, participants exhibited a significant increase in sadness from baseline ($M = 31.34$, $SD = 17.77$) to the interpersonal stressor ($M = 44.60$, $SD = 19.98$), $t(64) = 6.44$, $p < .001$. Next, linear regression was used to test the hypothesis that brooding rumination would predict greater cardiovascular reactivity to the interpersonal stressor. Using linear regression analyses with average HRV during the stressor as the dependent variable, average baseline HRV and brooding rumination were entered as independent variables. Results indicated that individuals with higher levels of brooding exhibited significantly greater reductions in HRV in response to the interpersonal stressor, $t(62) = -2.34$, $p = .02$, $r_{part} = -.29$.[1] Impor-

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1. To confirm these findings using the methodology of some prior literature, we reconducted our analyses using a change score (i.e., HRV stressor—HRV baseline). Consistent with the findings using linear regression, there was a significant correlation between brooding and the HRV change score ($r = -.30$, $p = .02$).

tantly, these results were maintained when statistically controlling for baseline depressive symptoms and sadness ratings during the baseline and stressor period, $t(59) = -2.40$, $p = .02$, $r_{part} = -.30$.

Discussion

The primary goal of this study was to examine the association between brooding rumination and cardiovascular reactivity to an interpersonal stressor. Consistent with our hypothesis, we found that women with higher levels of brooding rumination exhibited greater HRV withdrawal when exposed to an interpersonal stressor. These results were maintained even when we statistically controlled for women's current depressive symptoms and mood reactivity to the stressor, suggesting these results were at least partially independent of women's current levels of depression. This is important given the strong correlation between rumination and depression (e.g., Nolen-Hoeksema et al., 2008). The current results, therefore, suggest that brooding rumination may affect stress reactivity whether or not the person is currently depressed. This physiological vulnerability, when combined with high levels of stressful life events, may be a mechanism placing ruminators at greater risk for depressive disorders.

These findings have interesting implications for prior research examining altered stress reactivity among depressed and at-risk individuals. For example, there is evidence for blunted HRV withdrawal to stress in currently depressed individuals but not in individuals with remitted depression (Bylsma, Salomon, Taylor-Clift, Morris, & Rottenberg, 2014). In contrast, this study and others suggest that brooding rumination, a risk factor for depression, is associated with increased HRV withdrawal to stress (e.g., Borelli et al., 2014). These findings complement prior research showing that rumination is linked to lower resting HRV (Woody, McGeary, & Gibb, 2014) and prolonged cardiovascular activation following stress (Key, Campbell, Bacon, & Gerin, 2008). Given this mixed literature, future research is needed to determine if rumination is a potential moderator of HRV withdrawal among depressed or remitted individuals.

The results of this study also have potentially important implications for the types of laboratory-based stressors used to test the link between rumination and stress reactivity. For example, prior studies examining rumination and heightened biological stress reactivity have evaluated rumination for its association to performance-related stressors (e.g., Trier Social Stress Test) or negative mood inductions. Although these tasks often have an interpersonal component, they may not be tapping into the type of interpersonal stress most salient for high ruminators. Specifically, research suggests that ruminators are more likely to seek out social support after stress but report more social friction and less social support due to the negative interpersonal consequences of rumination (e.g., Nolen-Hoeksema et al., 2008). The current study provides initial support for the link between high levels of brooding rumination and exacerbated cardiovascular reactivity to a specific type of interpersonal stress, social rejection. Building upon these initial findings, future research on rumination and stress reactivity would benefit from the use of laboratory-based interpersonal stressors that tap into feelings of social isolation, loneliness, and rejection, as these may capture a more ecologically valid stress response.

The current study exhibited several strengths, including the focus on a distinct type of interpersonal stress using a social rejection induction and the examination of brooding rumination and stress reactivity controlling for state depression. However, there were some limitations that highlight areas for future research. First, the current study focused on women, and future research is needed to determine the generalizability of the results to men. Second, the study did not assess for past or current MDD and, therefore, cannot make any generalizations about the link between brooding rumination and cardiovascular reactivity in the context of a history of MDD. Finally, future research is needed to determine whether this heightened biological reactivity mediates the link between rumination and depression onset in the context of high life stress.

In summary, the current results provide important information about the association between brooding rumination and cardiovascular reactivity to interpersonal stress. Importantly, this study adds to the growing literature that suggests that higher levels of rumination are linked to greater biological reactivity to stress (Borelli et al., 2014; Roger & Najarian, 1998; Zoccola et al., 2010) and that this reactivity can be elicited by a social rejection induction. If replicated in clinical samples and examined longitudinally, these results could contribute to a better understanding of the mechanisms by which rumination increases risk for the development of depression. If this risk is inherent in heightened biological reactivity to stress, this knowledge could contribute to the development of intervention and prevention programs that seek to improve physiological functioning and regulation through biofeedback, stress reduction skills, and improvements in diet and exercise (Thayer & Lane, 2009).

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