Examining the Effects of Induced Rumination on HPA-Axis Regulation

By

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619 thesis submitted in partial fulfillment

of the requirements for the degree of

Masters of Science

(Clinical Psychology)

University of Michigan

2015

619 Readers:

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Abstract

Rumination impacts a persons' ability to cope with stress and may prolong or intensify the HPA-axis stress response. Prior studies have shown that ruminative tendencies are associated with greater HPA-axis response to laboratory stressors. However, most past studies have been correlational or have manipulated rumination before the stress task, making it difficult to tease apart stress-task induced rumination tendencies from individual differences in trait rumination. Therefore, the current study used an experimental protocol that manipulated rumination *after* a psychosocial stressor, while simultaneously assessing individual differences in trait rumination. This study examined 70 participants (35 males; age 18-21) who underwent a psychosocial stress task, and the Trier Social Stress Task (TSST). After the TSST, participants were randomly assigned to a rumination task (answering questions designed to elicit rumination about their performance) or a distraction task (completing non-evocative questions about their performance). We also assessed trait rumination prior to the TSST. A total of 10 saliva cortisol samples were obtained throughout the protocol. While salivary cortisol levels did not differ between the rumination or distraction condition, there was an interaction between trait rumination and experimental condition. Higher trait rumination was associated with prolonged duration of HPA-axis activation, but not higher intensity, only in the induced rumination condition. Trait rumination was not associated with HPA response in the distraction condition. Our findings suggest that one's individual tendency to ruminate prolonged the activation of the HPA-axis but this effect is mitigated if a person engages in distraction after a stress task. This suggests that trait aspects interact with contextual aspects (e.g., social context fosters rumination vs. distraction) in shaping

HPA-axis reactivity to psychosocial stress. The implications of these findings are discussed.

Key words: Psychopathology, Rumination, HPA-axis, Stress

Examining the Effects of Induced Rumination on HPA-Axis Regulation

Atypical Hypothalamic-Pituitary-Adrenal (HPA) axis, a key neuroendocrine system implicated in stress regulation, is associated with mood disorders, including depression (Lopez-Duran, Kovacs & George, 2009). Specifically, major depression is associated with atypical (delayed) recovery of the HPA-axis after stress in both adults (Burke, Davis, Otte & Mohr, 2005) and youth (Lopez-Duran, Kovacs & George, 2009). Yet, the underlying mechanism of this association is still poorly understood. It may reflect endogenous dysregulation of the HPA-axis in depressed individuals (e.g., hypersensitivity of the adrenal cortex, impaired negative feedback capacity) or result from sustained cortical signaling due to cognitive biases, such as ruminative tendencies (Kuehner, Huffziger, & Liebsch, 2009). Previous studies that have linked rumination to HPA axis reactivity have either been correlational or have manipulated rumination prior to the stress task (Young & Nolen-Hoeksema, 2001; Stewart, Mazurka, Bond, Wynne-Edwards & Harkness, 2013). This is problematic because neither approach dissects stress-task induced rumination tendencies from individual differences in trait rumination. Thus it is unknown whether ruminating about a stressor can result in the type of atypical HPA-axis response observed in depression. In this study, we manipulated rumination *after* a psychosocial stressor in order to examine its effects on HPA axis reactivity, particularly HPA axis delayed onset of recovery, while simultaneously assessing individual differences in trait aspects of rumination. The results of this study will advance our understanding of the independent and combined effects of induced stress and cognitive biases on HPA-axis reactivity.

The primary purpose of the HPA-axis is to maintain homeostasis and promote successful adaptation to the environment through hormonal regulation (Aguilera, 2012). For example, when a stressor is perceived and recognized by the limbic system, the paraventricular nucleus (PVN) of the hypothalamus secretes corticotropin-releasing hormone (CRH) and vasopressin (AVP) to the pituitary gland (Stratakis & Chrousos 1995). In response, the pituitary then secretes adrenocorticotropin hormone (ACTH) to the adrenal gland which increases production and release of glucocorticoids (cortisol in humans) as well as epinephrine (Gunnar & Vazquez, 2006). This physiological stress response redistributes energy to the brain and the muscles to promote survival and regulation in response to stress (Gunnar & Quevedo, 2007). There is a growing realization that cognitive process can impact how the HPA-axis responds to stress (e.g., Mayer, Abelson, & Lopez-Duran, 2014). This raises the possibility that atypical HPA-axis functioning observed in psychopathology is not due to endogenous problems with the axis itself but to atypical cognitive functioning. For example, cognitive biases such as ruminative thoughts could be responsible for prolonged signaling into the axis resulting in a strong or more prolonged cortisol response to stress. Specifically, rumination may result in continuous excitatory input into hypothalamus prolonging cortisol release from the adrenal and/or delaying the down-regulation of the cortisol response.

Ruminative thought can be described as focusing one's attention on his or her negative emotional state and its consequences, which inhibits any actions that might distract the individual from his or her negative mood (Nolen-Hoeksema, 1991). A number of studies have linked rumination tendencies to increased HPA responses to laboratory stress (e.g., Stewart et al., 2013). However, previous studies linking

rumination and the HPA response have been correlational (Young & Nolen-Hoeksema, 2001; Zoccola, Dickerson & Zaldivar, 2008; Stewart, Mazurka, Bond, Wynne-Edwards & Harkness, 2013), which provides us little information on whether the actual act of rumination negatively impacts stress regulation. In addition, the few studies that have actually manipulated rumination have done so before the stress task (Zoccola, Dickerson & Zaldivar, 2008; Stewart, Mazurka, Bond, Wynne-Edwards & Harkness, 2013). This is problematic because participants are asked to ruminate about something unrelated to the stress task, making it difficult to examine HPA responses to stress induced rumination. The two studies that have used a post-task rumination manipulation failed to induce a cortisol response (Kuehner, Huffziger, & Liebsch, 2009), (LeMoult & Joorman, 2014). While previous studies provide some evidence that rumination affects HPA responses to psychosocial stress (Kuehner, Huffziger, & Liebsch, 2009; Zoccola, Quas & Yim, 2010; LeMoult & Joorman, 2014), this direct link has yet to be tested experimentally. By inducing rumination after the stressor, we elicit comparable HPA-axis responses in all participants (controlling for individual differences in trait rumination) that allow us to test for the unique moderating effects of rumination on HPA responses to stress.

Therefore, the overall objective of this study is to examine whether rumination after a psychosocial stressor impacts HPA axis regulation using a randomized experimental protocol. It is hypothesized that participants who engage in the ruminative task, compared to a distraction task, will display delay in the recovery of the HPA-axis to the stressor.

Method

Participants

This study examined 70 university students (35 males; age rage 18-21; $M = 18.58$,

SD = 0.95), who participated in this study for course credit. Participants racially

identified as White (58.82%), Asian (29.41%), African American (5.88%), and Hispanic

(5.88%).

Design

Participants (n = 70) were exposed to social evaluative threat — using the Trier

Social Stress Test (TSST) — in a single laboratory visit. They were randomly assigned to

receive either rumination or distraction instructions following the TSST. Trait rumination

tendencies as well as depressive symptoms were also assessed prior to the TSST.

Procedure

This study consisted of a single laboratory visit lasting approximately 2 hours (see

figure 1). Participants reported to the laboratory between 12:30 to 2pm. They were

consented and accommodated to the research setting for about 30 minutes (adaptation

phase). During this time, participants completed demographic information and self-report

questionnaires. Participants then moved to a second room for a 15-minute social-

evaluative stress task, the Trier Social Stress Test (TSST; see below). After the TSST,

participants were randomly assigned to complete a 10-minute rumination or distraction

task, followed by a 35-minute regulation phase that took place in a different

accommodation room. Participants were debriefed at the end of the laboratory visit.

Materials

Demographic information and the following questionnaires were completed

during the adaption phase of the laboratory visit:

Ruminative Response Scale (RRS). The RRS is a self-reported measure of

rumination tendencies. Participants were asked how often they engage in ruminative

thoughts or behaviors when they feel sad or upset (Roelofs et al., 2006). The RRS is a 22-item questionnaire asking participants to provide ratings on a 1-4 scale (e.g., please indicate what you generally do… "think about a recent situation, wishing it had gone better") and is highly reliable and valid (Treynor, Gonzalez, and Nolen-Hoeksema, 2003).

Experimental Design and Stress Conditions

Laboratory stress task Protocol. In order to produce an endocrine stress response, we used a modified version of the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993). The TSST is a well-established psychosocial stress task designed to elicit a cortisol response in children and adults. In this paradigm, participants were asked to prepare a 5-minute speech, give the 5-minute speech and do 5 minutes of mental arithmetic in front of a panel of judges. They were told that the panel of judges consisted of University of Michigan's Peer Advisors, who evaluated and compared the quality of speech given to that of their peers.

Rumination vs. Distraction Task. Immediately following the TSST, participants were randomly assigned to either a rumination or distraction condition, which consisted of a revised version of the commonly used emotion regulation induction procedure developed by Nolen-Hoeksema and Morrow (1993) (Nolen-Hoeksema & Morrow, 1993; LeMoult & Joormann, 2014). This 10-minute task took place in the same room as the TSST. The rumination and distraction condition required participants to think and write about a series of questions related to the TSST. A total of 4 questions, one at a time, were displayed on a computer screen in front of the participants. Participants were then asked to think and write about each question online using an online data capturing system

(Qualtrics). In the rumination condition, participants were asked to focus on the negative self-evaluative aspects of the TSST experience and its potential consequences. Specifically, the participants answered the following questions 1) "Think about aspects of your performance that did not go as well. How do you think this impacted your chances of being selected?", 2) "Think about your physical stress symptoms during the last task. How do you think the judges interpreted them?", 3) "What kind of negative nonverbal feedback did you receive from the judges and how did it affect your performance?", and 4) "What does doing poorly on this task say/mean about your abilities as a student at the University of Michigan?" The distraction condition asked participants to focus on factual, non-performance related aspects of the TSST such as specific details of the TSST setting. Specifically, the participants answered the following questions: 1) "Please describe the physical characteristics of the judges (hair, eye color, gender, age, etc)", 2) "Please describe in a few sentences what you were asked to do in the previous task", 3) "Please describe in as much detail as you can the layout of the room you are in currently", and 4) "Thinking back on your speech, can you provide some details about the organization you had in mind about your speech?"

Cortisol Sampling

Salivary cortisol was collected during the laboratory stress protocol. A total of 10 saliva samples per participant were obtained. The first sample was collected after the participant consented to the study, (-30 minutes pre TSST). The second sample was obtained after the 30-minute adaptation period at the beginning of the speech preparation period (-5 before TSST). The third and fourth samples were taken approximately 5 minutes later, (0 minutes before TSST), and again immediately following the completion

of the math task, (10 minutes post TSST), respectively. The rest of the samples were obtained at 25, 30, 35, 40, 45 and 60 minutes after the start of the TSST.

Cortisol Analysis

All saliva samples were stored in a freezer in a secured room in preparation for analysis. Saliva samples were then analyzed for cortisol at the Core Assay Facility of the University of Michigan's Department of Psychology. Samples were stored at -20° C until assayed using an enzyme immunoassay kit (Salimetrics™). All samples from the same participant were assayed in the same batch. Duplicates varying more than 6% were re-assayed. The inter-assay and intra-assay coefficients of variability were 5% and 9% respectively.

Statistical Analysis Plan

We assessed HPA functioning by collecting salivary cortisol samples and using growth curve modeling (GCM) with landmark registration (GCM-LR; Lopez-Duran, Mayer, & Abelson, 2014) to control for variability in the timing of the stress response. This allows us to control for individual and group differences in salivary cortisol stress reactivity that might be obscured by time-to-peak variability (Lopez-Duran, Mayer, & Abelson, 2014). It simultaneously examines cortisol changes before and after the peak while controlling for baseline and response time differences, providing us with additional information that allows us to elucidate group differences in the underlying biological processes (e.g., intensity and duration of response, regulatory capacity) (Lopez-Duran, Mayer & Abelson, 2014).

<div align="center">

Results

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Sample Characteristics

No group differences were found in the distribution of ethnicity X^2 (3, N = 70) = 3.9, p = 0.27, age t(58) = 0.02, p = 0.98 for rumination condition M = 18.586, SD = 0.945 or distraction condition M = 18.580, SD = 0.992, or trait rumination t(69) = 0.43, p = 0.67 in the rumination condition M = 41.0, SD = 15.058 or distraction condition M = 39.685, SD = 9.878. No impact of gender was found on cortisol response, trait rumination or by condition so we did not include gender as a covariate. Gender was also equally distributed across groups 35 Males, M = 18.58, SD = 0.95, X^2 (1, N = 70) = 0.0, p = 1.000.

Cortisol Response

Salivary cortisol levels increase significantly from the start of the task, Activation Slope b = 0.02, SE = 0.003, p < .001, and then declined significantly after reaching peak, Recovery Slope b = -0.01, SE = 0.002, p < .001. This indicates that the TSST was effective in eliciting a cortisol response in this sample.

Impact of trait rumination, induced rumination, and distraction on cortisol response

In a model without interactions, trait rumination did not impact peak, b = .004, SE = .004, p = .304, Activation Slope b = .0004, SE = .0003, p = .149), or Recovery Slope (b =-0.0002, SE = .0003, p = .556). Condition (induced rumination vs. distraction) did not impact peak, b = .083, SE = .098, p = .395, Activation Slope b = -0.003, SE = .008, p = .686), or Recovery Slope (b =-0.006, SE = .007, p = .387).

We conducted a second conditional linear model to examine the interaction between trait rumination and condition predicting activation, peak and recovery (see figure 2a and 2b). Trait rumination and condition significantly interacted in predicting

peak levels, $F(1, 401) = 5.86, p = .016$ with no effect on activation, $F(1,264) = 0.08, p = .78$, and recovery slope, $F(1,273) = .25, p = .62$. Specifically, in the rumination condition, participants with higher trait rumination had higher peak levels ($b = -0.022$, SE $= .007, p = .0017$) with no effect on activation ($b = 0.0005$, SE $= .0005, p = .313$) or recovery slope ($b = -0004$, SE $= .0005, p = .458$). Differences in peak levels with no differences on activation or recovery slopes, while simultaneously controlling for baseline levels, indicate differences in timing such that those with higher trait rumination activate for longer.

Discussion

The aim of the study was to examine whether induced rumination after a psychosocial stressor impacted HPA-axis regulation. By manipulating rumination after the stressor, we were able to examine its effects on HPA-axis reactivity, while simultaneously assessing individual differences in trait rumination. Our findings suggest that an individual's tendency to ruminate prolonged activation of the HPA-axis only when asked to ruminate after the stressor. Our findings help to elucidate the impact of stress and cognitive biases on HPA-axis regulation.

Results indicated no main effects of trait rumination or condition on cortisol responses to the stressor. This is inconsistent with past literature that suggests rumination impacts HPA functioning (Kuehner, Huffziger, & Liebsch, 2009; LeMoult & Joorman, 2014). Lemoult and Joorman found that induced rumination prior to a stressor impacted the HPA-axis after the stressor. However, their stressor failed to elicit a HPA-axis response. Instead, rumination impacted a downward slope of cortisol that likely reflected recovery to the stress of coming to a laboratory. Their sample also included individuals

with current or past major depression and thus inducing rumination may have been easier compared to the healthy sample included in our study. Likewise, although Kuehner et al., (2009) also failed to activate the HPA-axis using a mood induction paradigm, they found a link between induced rumination and less decline of the HPA-axis during the laboratory visit but only among those with high depression symptoms. It is then possible that the impact of rumination on the HPA-axis is notable only among those who have elevated levels of depression symptoms. Alternatively, it is possible that inducing rumination is not easy if a person does not already "know" how to ruminate. Since rumination and depression are highly related (Treynor, Gonzalez, and Nolen-Hoeksema, 2003), past findings suggest that rumination can impact the HPA-axis only among those who already have rumination tendencies. Finally, our lack of main effect linking trait rumination and the HPA-axis is inconsistent with studies that found such a link (Stewart et al., 2013). One key difference between our study and past research is our use of the post stress condition. One half of our sample, and consequently, one half of trait ruminators were asked to engage in a distraction tasks, which, as we further discussed below, may have disrupted the normal association between trait rumination and HPA-axis activation.

Intriguingly, our results yielded an interaction of trait rumination and our rumination condition in shaping the HPA response to stress. Findings showed that participants who scored higher on trait rumination (higher RRS) and were in the rumination condition had higher peak levels with similar activation and recovery slopes compared to those lower in trait rumination. Differences in cortisol peak levels can be due to three factors: differences in intensity (slope) of activation, differences in baseline levels, or differences in the duration of activation. Since we found no differences in

activation slopes and we controlled for baseline levels, the observed differences in peak

levels can be attributed to differences in activation duration (see Lopez-Duran, Mayer, &

Abelson, 2014). This means that when asked to ruminate after the stressor, participants

with higher trait rumination activated for longer, reaching higher peak levels compared to

those lower in trait rumination. In contrast, participants who had higher trait rumination

but were assigned to the distraction condition did not have prolonged activation of the

HPA-axis and were comparable to those with lower trait rumination. These results are

consistent with past literature suggesting that cognitive processes can prolong activation

of the HPA-axis response to stress (Mayer, Abelson, & Lopez-Duran, 2014), and that

rumination does affect HPA responses to stress (Kuehner, Huffziger, & Liebsch, 2009;

Zoccola, Quas & Yim, 2010; LeMoult & Joorman, 2014). Thus, our finding that induced

rumination impacted HPA functioning for trait ruminators provides causal evidence that

rumination impacts HPA-axis regulation. However, our findings also suggest that

distraction is an effective strategy to mitigate the impact of trait rumination on HPA-axis

responses. This adds complexity to the body of research already understood about the

impact rumination has on HPA-axis functioning.

Finally, this study has important implications regarding the HPA mechanism by

which rumination impacts mood disorders. Specifically, previous research has found

depression to be associated with atypical recovery of the HPA-axis in both adults (Burke,

Davis, Otte & Mohr, 2005) and youth (Lopez-Duran, Kovacs & George, 2009). There is

a debate as to what this reflects. It may reflect endogenous dysregulation of the HPA-axis

in depressed individuals (e.g., hypersensitivity of the adrenal, impaired regulatory

capacity) that contributes to poor stress regulation among affected individuals. However,

it's possible that the HPA-axis is actually *intact* despite the elevated cortisol response to stress, and instead the axis is responding adaptably to more intense or more sustained cortical signals. Thus, cognitive biases could be responsible for such prolonged signaling, such as when individuals continue to think about the negative event after the event has concluded. Thus, cognitive functioning, such as trait tendencies can be one potential mechanism that affects HPA-axis functioning and explain HPA dysregulation observed in depressed populations.

There are several limitations within the current study. First, our sample consisted of a non-depressed population with relatively low demographic risk factors. This restricts generalizability of our findings to depressed populations. Specifically, future studies would benefit from including more diverse socio-economic and clinical populations. Additionally, the sample size of the current study was relatively small and thus replication with larger samples are needed to further confirm our interaction effect of trait rumination and rumination condition on hormonal levels. A larger sample size would allow for further analysis regarding the impact of gender and reveal potential main effects of trait rumination and conditions on cortisol peak levels.

Our findings suggest that one's individual tendency to ruminate prolonged activation of the HPA-axis but this effect is mitigated if a person engages in distraction after a stress task. This suggests that trait aspects interact with contextual aspects (e.g., social context fosters rumination vs. distraction) in shaping HPA-axis reactivity to psychosocial stress. This has important implications for treatment. Encouraging distraction as a coping strategy for those with a high tendency to ruminate could buffer stress-induced HPA responses. Interestingly, the observed pattern in the rumination

condition closely resembled HPA-axis responses to stress in mood disorders, particularly major depressive disorder. Future studies should further investigate the impact of rumination in the link between stress and atypical HPA axis reactivity.

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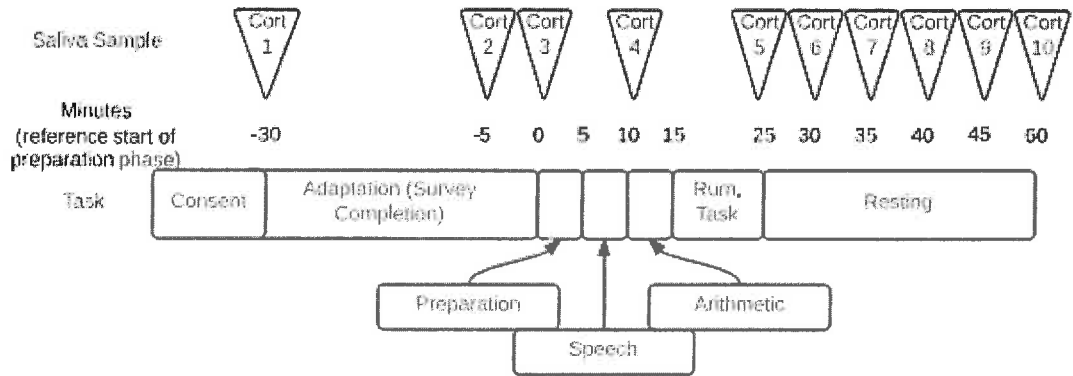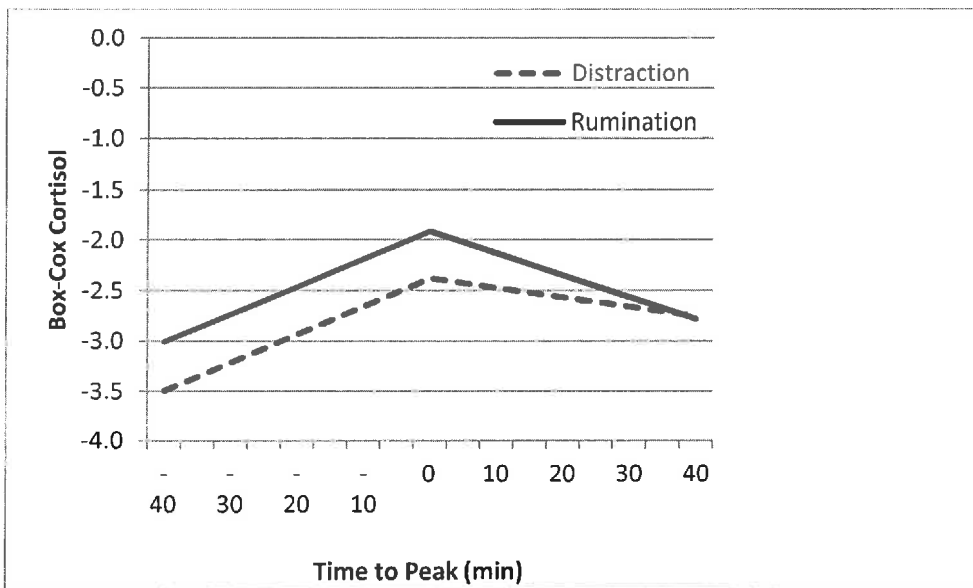
Figure 1.

Description of study protocol

Figure 2.

Graphs represent peak responses by trait rumination levels. (A) Low trait rumination differences in peak after accounting for differences in baseline did not significantly differ by condition. (B) High trait rumination displayed significant differences in peak after accounting for differences in baseline.

A)

B)