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Extending the recovery window: Effects of trait rumination on subsequent evening cortisol following a laboratory performance stressor



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Summary Mental rehearsal of past stressors through rumination may extend the physiological stress response and exposure to stress-related physiological mediators, such as cortisol. If repeated over time, this prolonged activation may contribute to a number of chronic health conditions. Findings from the emerging literature on the tendency to ruminate and its association with cortisol have been somewhat mixed. In the present study, we tested whether trait rumination predicted elevated cortisol concentrations in response to a performance stressor, and whether this association varied by the social-evaluative context of the stressor and gender. We also examined whether associations persisted into the evening of the stressor. Participants (50% female; mean age = 19.83, $SD = 1.62$) were randomly assigned to complete a laboratory speech stressor either in a social-evaluative (SET; $n = 86$) or non-evaluative context (non-SET; $n = 58$). Salivary cortisol concentrations were measured throughout the laboratory visit and later that evening. There was a main effect of trait rumination on greater total cortisol exposure into the evening of the stressor. In addition, trait rumination interacted with stressor context to predict cortisol declines: on the night of the SET stressor, high trait ruminators did not exhibit typical declines in cortisol. Different cortisol patterns emerged for men and women with tendencies to ruminate: women with higher rumination scores had flatter cortisol slopes with greater evening cortisol, whereas men with higher trait rumination scores had greater initial cortisol reactivity to the stressor. Together, these findings suggest that the relationship between the tendency to

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ruminate and cortisol concentrations is qualified by individual differences (gender) and stressor characteristics (social-evaluative threat).

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1. Introduction

Persistent or excessive exposure to stressors and consequent physiological changes have a range of negative health implications (for review, see [Juster et al., 2010](#)). One route to persistent activation of the stress response is through the mental rehearsal of stressful experiences, or rumination. In other words, cognitive representations of past stressful events may perpetuate stress-related physiological changes or reactivate stress responses. This conceptual model is outlined by the Perseverative Cognition Hypothesis, and there is growing empirical support for the premise that cognitive processes such as rumination can amplify, maintain, or reactivate physiological stress responses and predict greater somatic symptoms and physical health complaints ([Brosschot et al., 2006](#)). Thus far, the majority of the evidence for the association between rumination and extended related physiological activation has come from the cardiovascular domain. A smaller, but increasing body of work has begun to test this model within the context of the hypothalamic–pituitary–adrenal (HPA) axis (for review, see [Zoccola and Dickerson, 2012](#)). However, findings from the emerging literature on rumination and cortisol have been somewhat mixed. These discrepancies may stem in part from situational factors and individual differences, such as gender and the social-evaluative nature of the stressor.

Rumination has been defined in multiple ways across contexts, but there is general consensus that rumination can be described as repetitive, past-oriented thoughts about negatively-valenced content ([Segerstrom et al., 2003](#); [Smith and Alloy, 2009](#)). Rumination is a relatively stable emotion regulation strategy and certain individuals are more prone to perseverate than others. For example, individuals who display a ruminative cognitive style of responding to sad or depressive mood consistently report this tendency over a range of time intervals (e.g., [Just and Alloy, 1997](#); [Nolen-Hoeksema and Davis, 1999](#)). In addition, gender differences are frequently noted, with women tending to report more rumination than men ([Zlomke and Hahn, 2010](#)). Thus, to better understand whether and how rumination prolongs cortisol elevations, it may be useful to examine individual differences in the tendency for rumination and test for gender differences.

Conditions in which central goals are threatened or blocked are also important for shaping cortisol responses and ruminative thought. This is well illustrated with social self-preservation theory ([Dickerson et al., 2004](#)). According to this model, social-evaluative threat (SET) occurs when a key component of one's self-identity is actually or potentially judged in a negative way by others (e.g., rejection). In response to SET, one may engage in negative *self* evaluation and consequently, may experience shame and other self-conscious emotions, which in turn coordinate a specific set of physiological and behavioral responses, including

HPA axis activation ([Dickerson et al., 2004](#)). Meta-analytic and empirical studies indicate that SET preferentially elicits the HPA axis and subsequent increases in salivary cortisol ([Dickerson et al., 2008](#); [Dickerson and Kemeny, 2004](#)). In contrast, mood inductions or difficult distressing tasks that lack a social-evaluative component have been less reliable methods for eliciting increases in cortisol ([Dickerson and Kemeny, 2004](#)). Therefore, individuals who tend to ruminate on stressors may be more likely to experience prolonged cortisol exposure in response to cortisol-eliciting stressors, or SET contexts, compared to non-SET situations.

Rumination may also be more likely to occur in response to a SET context. In evaluative and potentially rejecting situations, the fundamental needs of an individual are threatened (e.g., need for social belonging). Such a failure in goal attainment is expected to elicit rumination ([Martin and Tesser, 1996](#)). Consistent with this premise, recent work provides support for the notion that SET can lead to both shame and rumination ([Zoccola et al., 2012, 2008](#)). Specifically, participants assigned to perform a SET stressor reported greater post-task ruminative thoughts than those who performed identical tasks without the evaluative component ([Zoccola et al., 2008](#)). A follow-up study confirmed these results, and demonstrated that SET effects on state rumination can persist at least 3–5 days post-stressor ([Zoccola et al., 2012](#)). Together these results suggest that stressors characterized by SET may be particularly likely to induce ruminative thought and prolong stress-related cortisol activation.

Past studies that have utilized SET stressors in the laboratory to test the relationship between rumination and cortisol have generally showed that rumination predicts greater cortisol reactivity or delayed recovery (e.g., [Puterman et al., 2011](#); [Zoccola et al., 2010, 2014](#)), although there have also been null ([Young and Nolen-Hoeksema, 2001](#)) and mixed findings depending on the rumination measure ([Gianferante et al., 2014](#); [Zoccola et al., 2008](#)). Results from studies testing the link between rumination and cortisol following *non-evaluative* stressor tasks (e.g., anger provocation; challenging model construction) indicate that cortisol concentrations may sometimes be greater in ruminative conditions relative to comparison tasks, such as distraction ([Denson et al., 2009](#); [Rudolph et al., 2011](#)). However, these studies have not demonstrated net increases in cortisol relative to pre-stressor resting values. Furthermore, the SET studies that demonstrated rumination-related elevations in cortisol during the recovery period had somewhat limited post-stressor follow-up periods (up to 75 min post-stressor). Although cortisol concentrations typically return to resting levels within the first hour following a social-evaluative psychosocial stressor ([Dickerson and Kemeny, 2004](#)), for individuals with a tendency to ruminate, cortisol recovery may take longer ([Puterman et al., 2011](#); [Zoccola et al., 2010](#)). Deviations from the normative diurnal decline in cortisol

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