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# The Relationship between Rumination and Affective, Cognitive, and Physiological Responses to Stress in Adolescents

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# Abstract

Although previous studies have established that rumination influences responses to stressful life events, the mechanisms underlying this relationship remain inadequately understood. The current study examines the relationship between trait rumination and affective, cognitive, and physiological responses to a standardized laboratory-based stressor in adolescents. A community-based sample of adolescents (N = 157) aged 13–17 completed the Trier Social Stress Test (TSST). Affective, cognitive, and physiological responses were obtained before, during, and after the TSST. Adolescents who engaged in habitual rumination experienced greater negative affect and more negative cognitive appraisals in response to the TSST than adolescents with lower levels of rumination. Rumination was unrelated to heart rate reactivity, but predicted slower heart rate recovery from the TSST, indicating that rumination might be specifically associated with physiological responses following stressors, suggesting potential mechanisms through which it might increase risk for psychopathology.

# Keywords

rumination; stress; stressful life events; emotion regulation; physiological reactivity; recovery

# Introduction

More than two decades ago, Susan Nolen-Hoeksema (1991) conceptualized rumination as the process of responding to distress by repetitively and passively focusing on one's symptoms and their possible causes and consequences. Since that time, hundreds of investigations have provided evidence underscoring the detrimental effects of this form of repetitive thinking. Most notably, rumination has been associated with the development and maintenance of a wide range of mental disorders, including depression, anxiety, eating disorders, and substance abuse problems (Aldao, Nolen-Hoeksema, & Schweizer, 2010;

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Nolen-Hoeksema & Watkins, 2011; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Given its central role in the onset and maintenance of multiple forms of psychopathology, rumination has become the direct target of numerous psychosocial interventions, as exemplified by Watkins' recent rumination-focused intervention (Watkins et al., 2011; Watkins et al., 2007).

Having established rumination as a robust, transdiagnostic risk factor for psychopathology, researchers have begun to explore the determinants of a ruminative response style. One particular focus of this line of work has been exposure to stress. Theoretical conceptualizations of rumination highlight the role of stressful life events in the developmental origin of repetitive, negative thinking (Nolen-Hoeksema & Watkins, 2011), and evidence from community-based studies suggests that engagement in rumination is a mechanism linking stress exposure to increases in negative affect and internalizing psychopathology (Michl, McLaughlin, Shepherd, & Nolen-Hoeksema, 2013; Moberly & Watkins, 2008). There is also emerging evidence that the relationship between stress and rumination is bi-directional, such that self-reported exposure to stressful life events is associated prospectively with increases in rumination (Michl, et al., 2013), but individuals who engage in high levels of rumination are also more likely to generate interpersonal stressors in their lives (McLaughlin & Nolen-Hoeksema, 2012). However, relatively little attention has been devoted to understanding the ways in which the habitual use of rumination may be associated with responses to stress in laboratory settings. In this investigation, we address this question by examining the relationship between trait levels of rumination and responses (affective, cognitive, and physiological) to a standardized laboratory-based stressor in adolescents.

Stress is defined as changes in social and environmental circumstances that require psychological and physiological adaptation by the organism (Monroe, 2008). The stress process involves a dynamic interaction over time between an individual and the environment that changes in response to social and environmental challenges, perceptions of those challenges, and the coping resources available to respond (Monroe, 2008). Exposure to a wide range of stressful life events has been consistently associated with the onset and maintenance of mental disorders in childhood, adolescence, and adulthood (Dohrenwend et al., 2006; Kendler, Hettema, Butera, Gardner, & Prescott, 2003; Kendler, Karkowski, & Prescott, 1999; Keyes, Hatzenbuehler, & Hasin, 2011; McLaughlin, Green, et al., 2010; McLaughlin et al., 2012), and engagement in rumination might influence responses to such events. Indeed, several lines of evidence suggest that rumination might mediate the relationship between stressors and psychopathology. Early work on rumination by Nolen-Hoeksema documented that individuals who engaged in high levels of rumination were more likely to experience symptoms of depression and major depressive episodes following stressful life events, including natural disasters (Nolen-Hoeksema & Morrow, 1991) and bereavement (Nolen-Hoeksema, Parker, & Larson, 1994). Other studies have found that high engagement in stress-reactive rumination, or self-referential thoughts focused specifically on a recent stressor, is associated with risk for future onset of depressive episodes (Robinson & Alloy, 2008).

Building on the evidence suggesting that rumination might influence responses to stress, several lines of work have begun to explore mechanisms that could explain this relationship. For example, individuals who habitually ruminate might experience stronger emotional reactions in response to stressors and/or might exhibit negative affect that is maintained for longer periods of time than those who do not ruminate (e.g., Nolen-Hoeksema et al., 2008). Because of the role that rumination plays in sustaining amygdala activation to negative emotional information (Ray et al., 2005; Siegle, Steinhauer, Thase, Stenger, & Carter, 2002) and prolonging depressed mood (Morrow & Nolen-Hoeksema, 1990; Nolen-Hoeksema & Morrow, 1993; Nolen-Hoeksema, Morrow, & Fredrickson, 1993), it is likely that rumination is associated with longer periods of negative affect following stressors. In addition to being associated with emotional responses to stress, rumination may be linked to cognitive responses to stressors. Given that rumination is associated with poor inhibitory control in response to emotional information (Joorman, 2006), individuals who ruminate might have difficulty re-directing attention away from thoughts about the stressor and its affective consequences. Additionally, rumination could engender more negative interpretations of stressful life events and maintenance of negative thinking following stressors. Indeed, evidence suggests that experimentally-induced rumination is associated with higher levels of cognitive distortions, more negative autobiographical memories, and lower perceptions of self-worth as compared to induced distraction (Rimes & Watkins, 2005; Watkins & Teasdale, 2001).

Recent evidence suggests that rumination might also be associated with physiological responses to stressors. Germane to this investigation, rumination has been associated with delayed cortisol recovery in response to stress (Stewart, Mazurka, Bond, Wynne-Edwards, & Harkness, 2013; Zoccola, Dickerson, & Zaldivar, 2008; Zoccola, Quas, & Yim, 2010). In addition, the literature on perseverative thinking and physiological reactivity suggests rumination and worry interfere with cardiovascular recovery (e.g., heart rate, heart rate variability, blood pressure) following a stressor, leading to prolonged physiological activation in response to stress (Brosschot, Gerin, & Thayer, 2006). Importantly, however, the vast majority of existing research on rumination and physiological responses to stress has focused on angry rumination, and consequently, much less is known about the cardiovascular concomitants of depressive rumination (see Siegle & Thayer, 2003). Indeed, the review by Brosschott et al. (2006) included only one study examining depressive rumination and ANS responses, and this study found no association between experimentally-induced rumination and autonomic nervous system [ANS] activity as compared to induced distraction (Vickers & Vogeltanz-Holm, 2003). Moreover, the primary focus of this line of inquiry has been on state-level rumination and, consequently, much remains to be understood about the relationship between habitual (i.e., trait) use of rumination and cardiovascular reactivity and recovery. Examining these relationships is particularly important because it can help us understand how individual differences in the use of rumination might place people at greater risk for developing psychopathology upon encountering stressors (e.g., Nolen-Hoeksema & Watkins, 2011).

We extend research on the relationship between rumination and stress in several ways. First, there is a dearth of previous studies examining individual differences in depressive rumination as a predictor of ANS reactivity and recovery following a stressor. Thus, this

investigation provides a novel extension of previous work by examining the relationship between depressive rumination and responses to stress across affective, cognitive, and physiological domains, focusing specifically on patterns of ANS activity.

Second, most laboratory-based studies of rumination and responses to stress have been conducted in samples of adults (c.f., Stewart et al., 2013). Adolescence is a period associated with substantial development in physiological stress response systems. Adolescents exhibit greater reactivity to social and performance stressors (Gunnar, Wewerka, Frenn, Long, & Griggs, 2009; Stroud et al., 2009), and are more likely to experience negative affect in response to stressors as compared to children at earlier periods of development (Larson & Ham, 1993; Larson, Moneta, Richards, & Wilson, 2002). Thus, adolescence is an important developmental period in which to examine the influence of emotion regulation strategies, including rumination, on stress reactivity.

Third, with rare exception (Stewart, et al., 2013; Zoccola, et al., 2010, 2008), existing evidence on rumination and responses to stress is based largely on studies that rely on self-reported exposure to stressful life events (Michl, et al., 2013) or that examine self-reported responses to stress, without directly measuring exposure to stressors (Robinson & Alloy, 2008). A central limitation in studies examining self-reported stressful life events is that individuals vary widely in the degree to which they experience negative affect in response to stressors (Hammen, 1991; Rudolph & Hammen, 1999), and those who are at risk of depression or experiencing low mood due to high engagement in rumination might be particularly likely to perceive events to be stressful or negative. Consequently, examining the association between rumination and stress responses in study designs that experimentally manipulate stressors is important.

In the current study, we examined the associations between rumination and responses to a standardized laboratory-based social/evaluative stressor, the Trier Social Stress Test (TSST) (Kirschbaum, Pirke, & Hellhammer, 1993), in a community-based sample of adolescents. Specifically, we examined whether rumination was associated with affective, cognitive, and physiological responses to the TSST. We hypothesized that higher levels of trait rumination would be associated with: (1) greater increases in negative affect following the TSST, (2) more negative cognitive appraisals of the task, and (3) slower physiological recovery following the task. We extend previous research on rumination and physiological reactivity and recovery by examining the association of rumination with cardiovascular responses to a laboratory-based stressor. In addition, given substantial evidence for gender differences in rumination (Nolen-Hoeksema, 2001; Nolen-Hoeksema & Twenge, 2002), we examined whether the associations of rumination with stress responses varied according to gender. Although females engage in higher levels of rumination than males, existing evidence suggests that the processes associated with rumination may, at times, be similar for males and females (Michl, et al., 2013; Nolen-Hoeksema & Aldao, 2011). Thus, we expected that the associations between rumination and stress responses would be similar for males and females.

## Methods

#### **Participants**

A community-based sample of 172 adolescents aged 13-17 was recruited for participation at schools, after-school programs, medical clinics, and the general community in Boston and Cambridge, MA. Recruitment efforts were targeted at obtaining a sample with high diversity with respect to race/ethnicity and socio-economic status (SES) and variability in exposure to childhood adversity experiences, including maltreatment. To do so, we recruited heavily from low-SES neighborhoods and from clinics that served a predominantly low-SES catchment area. A total of 4 adolescents were taking medications known to influence cardiovascular functioning (e.g., beta blockers), so we did not run them through the study procedures. The resulting sample of 168 adolescents was 56.0% female (n = 94) and had a mean age of 14.9 years (SD = 1.36). All females were post-menarchal. Racial/ethnic composition of the sample was as follows: 40.8% White (n = 69), 18.34% Black (n=31), 17.8% Hispanic (n = 30), 7.7% Asian (n = 13), and 14.8% Biracial or Other (n = 25). Over one-third of the sample (38.1%, n = 64) was from single-parent households. Equipment malfunctions resulted in loss of physiological data from 8 participants. We excluded participants from analysis due to presence of a heart murmur (n = 1), severe cognitive impairment (n = 1), and a diagnosis of a pervasive developmental disorder (n = 1). The final sample included 157 participants.

#### Measures

**Rumination**—We administered an abbreviated form of the Children's Response Styles Questionnaire (CRSQ; Abela, Brozina, & Haigh, 2002), a 25-item scale that assesses the extent to which children respond to sad feelings with rumination. The measure is modeled after the Response Styles Questionnaire (Nolen-Hoeksema & Morrow, 1991) that was developed for adults. For each item, youth are asked to rate how often they respond in that way when they feel sad on a 4-point Likert scale ranging from almost never (1) to almost always (4). The rumination subscale of our abbreviated measure included 6 items that were summed. Sample items include: "*Think about a recent situation wishing it had gone better*" and "*Think why can't I handle things better?*"The reliability and validity of the CRSQ have been demonstrated in samples of early adolescents (Abela et al., 2002). The abbreviated CRSQ rumination scale demonstrated good reliability in this study ( $\alpha = 0.81$ ), which is nearly identical to that one generated by studies utilizing the full CRSQ measure (Abela et al., 2002,  $\alpha$  .76,  $\alpha$  .84; McLaughlin & Nolen-Hoeksema, 2011,  $\alpha = 86$ ).

**Depression**—We administered the Children's Depression Inventory (CDI; Kovacs, 1992), which is a widely used self-report measure of depressive symptoms in children and adolescents. It includes 27 items consisting of three statements (e.g., "I am sad once in a while," "I am sad many times," and "I am sad all the time") representing different levels of severity of a specific symptom of depression. The CDI has sound psychometric properties, including internal consistency, test–retest reliability, and discriminant validity (Kovacs, 1992; Reynolds, 1994). The item pertaining to suicidal ideation was removed from the measure at the request of school officials and the human subjects committee. The 26

remaining items were summed to create a total score ranging from 0 to 52. The CDI demonstrated excellent reliability in this sample ( $\alpha = .89$ ).

Affect—Participants completed the Positive and Negative Affect Scale (PANAS: Watson, Clark, & Tellegen, 1988) before the baseline period and immediately after the stress procedure. The PANAS is a 20-item measure that assesses positive and negative affect. We administered the state version of the PANAS, which asks participants to "indicate the extent to which you feel this way right now, that is, how you feel right at this moment." Participants respond on a 5-point Likert scale ranging from 1 very slightly or not at all (1) to extremely (5). The PANAS has excellent internal consistency (Watson et al., 1988) and has demonstrated convergent, discriminant, and predictive validity in a number of investigations (Waikar & Craske, 1997; Watson & Walker, 1996). In this study, we present the findings pertaining to negative affect. The internal reliability of this scale was good ( $\alpha = 0.84$ ).

**Cognitive Appraisals**—Participants completed appraisals of the degree of perceived demands and personal resources they anticipated and experienced before the baseline period and immediately after the stressor by using a measure utilized in studies of the biopsychosocial model of challenge and threat (Jamieson, Nock, & Mendes, 2012; Mendes, Gray, Mendoza-Denton, Major, & Epel, 2007), a theory that is used to differentiate adaptive from maladaptive responses to stress (Blascovich, 2008; Mendes, Major, McCoy, & Blascovich, 2008). Within this model, appraisal of greater demands than resources associated with a stressor is related to a pattern of affective and physiological responding characteristic of *threat*, whereas appraisal of greater resources to meet the demands of a challenging situation are associated with greater approach-related affect and physiological responses characteristic of challenge (Blascovich & Mendes, 2000; Mendes, et al., 2007; Mendes, et al., 2008). Each item was rated on a one- to seven-point scale. Items representing situational demands (e.g., "The upcoming task is difficult") and personal resources (e.g., "I have the abilities to perform the upcoming task successfully") were summed separately. The internal reliability of the demand and resource appraisal scales was good ( $\alpha = 0.77$  and 0.81, respectively).

**Physiological measures**—Electrocardiogram (ECG) and impedance cardiography recordings were acquired to generate measures of heart rate, pre-ejection period (PEP), a measure of sympathetic nervous system activity, and respiratory sinus arrhythmia (RSA), a measure of parasympathetic nervous system activity. ECG recordings were obtained with a Biopac ECG amplifier (Goleta, CA) using a modified Lead II configuration (right clavicle, left lower torso, and right leg ground). Cardiac impedance recordings were obtained with a Bio-Impedance Technology model HIC-2500 impedance cardiograph (Chapel Hill, NC). One pair of mylar tapes completely encircled the neck and another pair encircled the torso. A continuous 500  $\mu$ A AC 95 kHz current was passed through the two outer electrodes, and basal thoracic impedance (z0) and the first derivative of basal impedance (dz/dt) was measured from the inner electrodes. A Biopac MP150 integrated the ECG and impedance cardiography signals, sampled at 1.0 kHz, using Acqknowledge software.

ECG and impedance cardiograph data were scored by raters blind to participant status on rumination and psychopathology. Signals were averaged into one-minute epochs using

Mindware Software (Mindware Technologies, Gahanna, OH). Minutes with significant artifact in the ECG and dz/dt signal were not scored to prevent bias in heart rate, PEP, and RSA estimates (<3% of minutes recorded were discarded). RSA was calculated from the inter-beat interval time series using spectral analysis implemented in Mindware HRV Software. RSA was calculated for the frequency band 0.12 - 0.40 Hz. Based on evidence suggesting that controlling for respiration rate is necessary in order for RSA to represent a measure of purely parasympathetic cardiac control (Grossman, Karemaker, & Wieling, 1991; Grossman & Taylor, 2007), we controlled for respiration rate in all analysis. Respiration rate was derived from the basal cardiac impedance signal. Impedance cardiography was used to calculate PEP, a measure of sympathetic nervous system activation representing the amount of time that elapses from the beginning of ventricular depolarization to the moment the aortic valve opens and blood begins leaving the left ventricle (electrical systole). Because accurate scoring of impedance cardiography data requires manual placement of the B point (opening of the aortic valve) (Blascovich, Mendes, Vanman, & Dickerson, 2011), these data were scored by two independent raters. PEP differences of more than 5% were reviewed and adjudicated by author KM. Reactivity values were calculated using the first minute of each task (preparation, speech, math), which is standard practice (Jamieson, Mendes, Blackstock, & Schmader, 2010; Mendes, et al., 2008).

**Procedure**—Baseline physiological data were collected during a five-minute period in which participants were asked to sit quietly. Adolescents then completed questionnaire and interview measures. Informed consent was obtained from the parent/guardian who attended the session, and assent was provided by adolescents.

Participants completed the Trier Social Stress Test (TSST) (Kirschbaum, et al., 1993), a widely-used stress induction procedure that has been used with children and adolescents (Buske-Kirschbaum et al., 1997; Buske-Kirschbaum et al., 2003; Stroud, et al., 2009). The TSST involves three periods (speech preparation, speech, and math). After being told that they would be delivering a speech in front of trained evaluators who would judge their performance, participants were given five minutes to prepare their speech. In the current study, participants were asked to talk about the qualities of a good friend and which of those characteristics they did and did not possess. Next, participants delivered a five-minute speech in front of two evaluators. Evaluators were trained to provide neutral and mildly negative feedback (e.g., appearing bored) during the speech. Finally, participants completed a mental subtraction task out loud in front of the evaluators for five-minutes. Specifically, participants were asked to count backwards in steps of seven from a three-digit number and were stopped and asked to start again each time they made a mistake. ECG and cardiac impedance recordings were measured continuously across each period; blood pressure recordings were sampled during the first and fourth minutes of each period. Two participants declined to participate in the TSST, resulting in a total of 155 participants who completed the procedure.

Following the TSST, participants were asked to sit quietly for another five-minute period, and their physiological activity was monitored, allowing examination of physiological recovery following the TSST.

#### **Statistical Analysis**

We examined the association of rumination with affect, cognitive appraisals, and cardiovascular reactivity and recovery related to the TSST using linear regression. We first examined whether trait rumination predicted negative affect ascertained after participants were told they would be delivering a speech but before the TSST preparation period began and immediately following the TSST, controlling for baseline affect. Second, we tested whether rumination was associated with cognitive appraisals (i.e., demand and resource appraisals). To do so, we examined appraisals before the TSST preparation period began (but after participants learned they would be delivering a speech), and appraisals of the speech and math portions of the TSST ascertained immediately following the task. In addition, we examined whether rumination predicted *changes* (i.e., difference scores) in perceived demands and resources from the assessment prior to the TSST to the post-TSST assessment. Third, we examined whether rumination predicted changes in heart rate, PEP, and RSA during each component of the TSST, controlling for baseline values.

We then tested whether trait rumination was associated with cardiovascular recovery following the TSST by testing the association with heart rate, PEP, and RSA during the entire recovery period relative to baseline and relative to the end of the TSST (i.e., including baseline and/or the end of the TSST as covariates in the regression models). To obtain a more nuanced profile of patterns of cardiovascular recovery for physiological markers where a significant difference was observed, we also tested whether rumination was associated with heart rate, PEP, and RSA at each of the 5 minutes of the recovery period, including baseline activity as a covariate. In all analyses, we controlled for age and gender. Given the strong association of rumination with depressive symptoms, we conducted a sensitivity analysis by additionally controlling for CDI scores in each of our models. Finally, to determine whether these associations were moderated by gender, we added an interaction term between gender and rumination to each of the models.<sup>1</sup>

# Results

#### **Descriptive Statistics**

Table 1 provides the mean and standard deviation of rumination, affect, and cognitive appraisals before and after the TSST. Table 2 provides the mean and standard deviation of heart rate, PEP, and RSA at baseline, during each of the three periods of the TSST, and during the recovery period. Importantly, the TSST elicited significant decreases in PEP (i.e., increases in sympathetic activation), F(3,450) = 107.98, p < .001, and significant increases in heart rate, F(3, 459) = 231.94, p < .001. Conversely, it did not produce changes in subjective negative affect, F(1,156) = 2.09, p = .15.

#### **Rumination and Affect**

Rumination was positively associated with higher levels of negative affect prior to the TSST,  $\beta = 0.23$ , p < .004, and greater increases in negative affect following the TSST (controlling

 $<sup>^{1}</sup>$ In addition, because the sample was recruited to include adolescents with exposure to child maltreatment, we re-estimated all models after adding an interaction term between rumination and 1) child maltreatment exposure, and 2) child maltreatment severity. None of these interactions were significant. As such, we only present analysis on the entire sample.

J Exp Psychopathol. Author manuscript; available in PMC 2016 April 28.

for pre-TSST negative affect),  $\beta = 0.26$ , p < .001. These associations were not moderated by gender,  $\beta = .01 \ p = .87$ ,  $\beta = -0.02$ , p = .82, respectively. Importantly, each of these associations remained significant after controlling for depressive symptoms,  $\beta = 0.23$ , p < .010, and  $\beta = 0.25$ , p < .001, respectively.

#### **Rumination and Cognitive Appraisals**

Trait rumination was not associated with either demand appraisals,  $\beta = 0.06$ , p = .46, or with resource appraisals,  $\beta = 0.01$ , p = .87, prior to the TSST. Following the TSST, rumination was associated with demand appraisals related to both the speech,  $\beta = 0.20$ , p = .012, and math,  $\beta = 0.18$ , p = .027, such that higher rumination was associated with greater perceived threat related to these tasks. After controlling for depressive symptoms, however, rumination became marginally associated with post-speech threat perception,  $\beta = .17$ , p = .060 and non-significantly associated with math threat perception,  $\beta = .09$ , p = .339. Rumination was unrelated to resource appraisals regarding the speech,  $\beta = -0.06$ , p = .46, or the math,  $\beta = -0.08$ , p = .324, prior to the TSST.

Rumination was also related to *changes* in cognitive appraisal from baseline. Higher trait rumination was associated with greater change in perceived demands related to the speech,  $\beta = 0.14$ , p = .029, and to the math,  $\beta = 0.18$ , p = .027, such that, higher trait rumination was associated with greater increases in perceived demands related to the stress tasks. When we controlled for depressive symptoms, rumination was not significantly associated with changes in threat perception related to the speech,  $\beta = .10$ , p = .182, but it remained significantly associated with changes in threat perception related to math,  $\beta = 0.25$ , p = .005. No associations were observed between rumination and changes in resource appraisals ( $\beta = .$ 10 and -.08, p = .16 and .33). We found no evidence for an interaction between rumination and gender in predicting demand and resource appraisals at any point in the study ( $\beta$ s range from -0.24 to -.01 and p-values range from .21 to .95).

#### **Rumination and Cardiovascular Reactivity**

Rumination was unrelated to baseline physiological characteristics or to heart rate reactivity, PEP reactivity, or RSA reactivity during the speech preparation, speech, and math portions of the TSST (all *p*-values range from .15 to .97; see Table 3).

#### **Rumination and Cardiovascular Recovery**

In contrast to the lack of associations between rumination and cardiovascular reactivity, rumination was associated with slower heart rate recovery following the TSST. Specifically, rumination was associated with higher heart rate during the recovery period relative to baseline,  $\beta = 0.06$ , p = .050, and marginally higher heart rate relative to the speech,  $\beta = 0.11$ , p = .061, and math,  $\beta = 0.18$ , p = .072, portions of the TSST. Importantly, when controlling for depressive symptoms, rumination remained a significant predictor of heart rate during recovery relative to baseline,  $\beta = .07$ , p = .026. Associations with heart rate recovery relative to the speech,  $\beta = 0.04$ , p = .482, portions of the TSST were no longer significant.

We next examined the relationship between rumination and recovery at each minute during the recovery period relative to baseline. Here, rumination was associated with greater heart rate relative to baseline during the second,  $\beta = 0.07$ , p = .031, and third,  $\beta = 0.07$ , p = .038, minutes of the baseline period and marginally during the first minute,  $\beta = 0.07$ , p = .068. Gender did not interact with rumination in predicting heart rate recovery during the entire recovery period,  $\beta = -0.02$ , p = .773, or during any minute of the recovery period ( $\beta$  range from -0.05 - 0.04, *p*-values range from .53-.79). Importantly, when adding depression scores as a covariate, the association between rumination and heart rate recovery became stronger. Specifically, rumination continued to be significantly associated with greater heart rate relative to baseline during the second,  $\beta = 0.08$ , p = .033, and third,  $\beta = 0.08$ , p = .025, minutes of the baseline period, and marginally during the first minute,  $\beta = 0.07$ , p = .087. In addition, it became marginally associated with heart rate relative to baseline during the fourth,  $\beta = 0.07$ , p = .059, and fifth periods,  $\beta = 0.06$ , p = .090, which were previously non-significant.

Rumination did not predict RSA or PEP during the recovery period relative to baseline or to either portion of the TSST, and no gender interactions were observed in these associations.

# Discussion

A variety of affective, cognitive, and interpersonal mechanisms underlie the relationship between trait rumination and anxiety and mood pathology (see Nolen-Hoeksema et al., 2008 for a review). We provide novel evidence for an additional pathway through which rumination might influence risk for psychopathology. Our results indicate that rumination is associated with heightened affective and cognitive reactions to psychosocial stress as well as impaired ANS recovery. Specifically, we found that adolescents who scored high on a trait rumination experienced greater negative affect in response to a standardized laboratorybased stressor, more negative appraisals of the task, and slower heart rate recovery once the stressor ended as compared to adolescents who reported lower habitual use of rumination. In contrast, rumination was not associated with patterns of ANS reactivity during the stress procedure, suggesting that rumination might be specifically associated with physiological *recovery* from the effects of stress. Importantly, these patterns of results were largely replicated when controlling for the overlap between rumination and depression.

Our findings indicate that rumination is associated with negative affective, cognitive, and physiological responses following a stressor. One interpretation of this pattern is that for people who habitually ruminate, stress can result in a cascade of negative feelings, thoughts, and physiological responses that culminate in prolonged physiological activation once the stressor has ended. With respect to affective responses, experimental studies have shown that rumination generates greater levels of negative affect than other types of thinking, including distraction, and is associated with prolonged negative emotional states (McLaughlin, Borkovec, & Sibrava, 2007; Morrow & Nolen-Hoeksema, 1990; Nolen-Hoeksema & Morrow, 1993). In addition, neuroimaging research suggests that individuals who ruminate experienced prolonged amygdala activation in response to emotional stimuli (Ray, et al., 2005; Siegle, et al., 2002). These patterns suggest that rumination is also likely to influence

the duration of negative affect experienced following a stressful event, something that was not measured directly in the current study.

Although rumination was unrelated to appraisals of the degree of demands associated with the public speaking task before it began, higher habitual use of rumination was associated with more negative appraisals of how stressful and threatening the task was once it had ended, as well as with a greater increase from baseline in negative appraisals of the task. This pattern is consistent with previous evidence suggesting that rumination is associated with patterns of maladaptive, negative thinking, including negative attributions about social situations, greater recall of negative memories, and intrusive negative thoughts during tasks requiring concentration (Lyubomirsky, Caldwell, & Nolen-Hoeksema, 1998; Lyubomirsky, Kasri, & Zehm, 2003; Lyubomirsky & Nolen-Hoeksema, 1995; Watkins, 2008).

It has been argued that perseverative thinking, by maintaining the cognitive representation of stressful events for prolonged periods of time, may result in slower cardiovascular recovery (Brosschot, et al., 2006). Our findings support this notion. Specifically, high levels of habitual rumination were associated with a slower rate of heart rate recovery. Importantly, however, we found no associations between rumination and either PEP or RSA, which are measures of sympathetic and parasympathetic nervous system activation, respectively. This pattern is consistent with meta-analytic findings, which suggest that psychosocial factors rarely predict PEP and RSA recovery after laboratory-based stressors (Burke, Davis, Otte, & Mohr, 2005). Because heart rate is influence by both sympathetic and parasympathetic influences, however, it is possible that rumination has small and non-significant negative associations with PEP (i.e., greater sympathetic activation) and RSA (i.e., reduced parasympathetic activation) that combine to influence heart rate.

The relationship between rumination and affective, cognitive, and physiological response to stress is likely to have additional detrimental downstream effects on social behavior and mental health. Prior research suggests that the type of negative cognitive appraisals observed here among adolescents with high levels of rumination are associated with poor decisionmaking in emotional situations (Kassam, Koslov, & Mendes, 2009) and low engagement in approach-related social behavior (Koslov, Mendes, Pajtas, & Pizzagalli, 2011), which might reduce the likelihood that individuals with high levels of rumination seek social support following exposure to stressors. Given that rumination is associated with other types of interpersonal problems (Nolen-Hoeksema & Davis, 1999; Schwartz & McCombs, 1995), negative responses to stress may further impede efforts to obtain social support that could mitigate the negative mental health consequences of stress exposure, a possibility that awaits further study. The patterns of emotional and physiological reactivity associated with rumination may also contribute to the onset of psychopathology. For example, prospective research suggests that elevated emotional reactivity to stress is associated with the onset of mood and anxiety disorders (McLaughlin, Kubzansky, et al., 2010). In addition, metaanalytic findings suggest that delayed or incomplete physiological recovery following a stressor is associated with both major depression and anxiety (Burke, et al., 2005). Overall, our findings suggest that heightened cognitive and emotional reactivity to stress and poor physiological recovery are additional pathways through which rumination may operate to increase risk for psychopathology. This is somewhat consistent with theoretical

conceptualization of rumination as a transdiagnostic factor linking distal stressful life events to psychopathology risk (Nolen-Hoeksema & Watkins, 2011). However, Nolen-Hoeksema and Watkins (2011) stipulated that distal stress might lead to rumination through dysregulated stress response, which in turn might result in psychopathology. In this respect, it will be essential for future work to determine whether rumination precedes dysregulated stress responses, or whether changes in stress reactivity precede later increases in rumination. In addition, it will be important to identify whether the directionality of this relationship might vary as a function of characteristics of the person and/or the situation.

In the current study, we assessed trait levels of depressive rumination. This represents an important extension on rumination and stress literature because most prior studies have focused on angry rumination (Schwartz, Gerin, Davidson, & Christenfield, 2000; Suchday, Carter, Ewart, Larkin, & Desiderato, 2004). Importantly, our finding that depressive rumination is associated with slowed heart rate recovery following a psychosocial stressor is consistent with findings on angry rumination, which also suggest prolonged physiological activation among those with high levels of angry rumination (Brosschot, et al., 2006). Other studies have focused on stress-reactive rumination, which has been shown to be more strongly associated with depression following stressful life events than rumination focused on sad mood or dysphoria (Robinson & Alloy, 2008). Consequently, it will be of great importance for future work examining the relationship between rumination and stressful events to focus on the assessment of stress-reactive rumination, for example, by administering the stress-reactive rumination scale (e.g., Alloy et al. 2000). This is particularly noteworthy in light of the small effect sizes found between habitual rumination and heart rate recovery and the null findings pertaining to RSA/PEEP recovery and physiological reactivity. More broadly, as we have recently suggested (Aldao, Mennin, & McLaughlin, 2013), it will be essential to examine the relationship between various forms of rumination and physiological reactivity and recovery. We also propose that by assessing fluctuations in state-level rumination in the context of laboratory stressors, we might be able to develop a more nuanced understanding of the bidirectional relationship between rumination and affective, cognitive, and physiological reactivity and recovery (Aldao, 2013).

The findings from this investigation suggest additional avenues for future work. First, this study focused specifically on adolescence, a period associated with substantial development in physiological stress response systems (e.g., Gunnar et al., 2009; Stroud et al., 2009). Consequently, these results require replication in studies of adults. Second, we focused on a particular kind of stressor—namely, a social/evaluative stressor known to elicit strong cardiovascular responses. Rumination has been examined in the context of several other types of life stressors, including bereavement and natural disasters (Nolen-Hoeksema et al., 1994; Nolen-Hoeksema & Morrow, 1991). However, the degree to which rumination influences recovery from other types of stressors, such as traumatic events, remains to be determined. Moreover, as we mentioned above, it will be important to examine various forms of rumination, in particular those that directly assess responses to stress (e.g., stress reactive rumination scale; Alloy et al., 2000).

Third, we conducted a relatively large number of analyses, particularly when testing physiological reactivity and recovery, which could have resulted in an inflated type I error

rate. However, adopting a liberal approach to hypothesis testing was necessary given that 1) the examination of rumination in relation to physiological reactivity/recovery is a relatively new area of inquiry and 2) there are multiple ways of assessing recovery. We hope that the growing interest in studying the associations between rumination and physiological processes will lead to the identification of which physiological measures are most robustly associated with rumination as well as which are the ideal time frames for capturing reactivity and recovery. This growing body of literature will, in turn, provide investigators with the necessary information to adopt a more rigorous approach to hypothesis testing. Fourth, it is important to note that the task elicited a relatively small increase in negative affect (p = .15), despite being associated with large increases in sympathetic nervous system activation (p < . 001). This might have been due to the relatively low prevalence of mental disorders in our sample, an idea that is further supported by the fact that participants who ruminated habitually experienced greater increases in their negative affect. Consequently, it will be essential for future work to examine the relationship between rumination and stress responses in a clinical population.

Perhaps one of the most notable limitations of this investigation is that we focused on trait rumination, which prevented us from testing the temporal covariation between state rumination and reactivity and recovery. In this respect, it will be essential for future investigations to assess and manipulate rumination during laboratory stressors. As we mentioned earlier, this would provide investigators with the opportunity to examine how rumination impacts stress, and how, in turn, stress might impact subsequent rumination. Additionally, our sample included a high proportion of adolescents who had experienced child maltreatment. Although we found no evidence for interactions between maltreatment and rumination in predicting responses to stress, these findings nonetheless warrant replication in samples with lower levels of exposure to adversity.

Rumination is associated with increased risk of multiple forms of psychopathology. A variety of mechanisms have been shown to underlie this relationship. In the current study, we provide evidence highlighting another potential mechanism through which rumination exerts detrimental effects on mental health. Specifically, high levels of rumination were associated with a variety of maladaptive responses to a standardized laboratory-based stressor, including heightened negative affect, negative cognitive appraisals of the task, and slow physiological recovery. Future research is needed to determine whether interventions targeting rumination have beneficial downstream effects on these dimensions of stress reactivity and recovery.

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## Table 1

Descriptive statistics of rumination, affect, cognition, and psychopathology variables (N = 157)

	Mean	SD
Trait Rumination	5.95	(4.20)
Negative Affect		
Baseline	2.01	0.65
Post-TSST	2.06	0.77
Demand Appraisals		
Pre-TSST	3.70	1.09
Speech	4.50	3.15
Math	4.21	1.27
Challenge Appraisals		
Pre-TSST	4.52	1.05
Speech	3.82	1.50
Math	4.41	1.71

# Table 2

Descriptive statistics of physiological variables at baseline, during the TSST, and during the recovery period (N = 157)

	Mean	SD
Heart Rate		
Baseline	75.23	12.18
Preparation	86.32	16.57
Speech	92.63	17.70
Math	88.96	15.64
Recovery	77.23	13.18
RSA		
Baseline	6.65	1.08
Preparation	6.55	1.12
Speech	6.45	1.09
Math	6.54	1.21
Recovery	6.58	1.12
PEP		
Baseline	104.93	7.77
Preparation	93.86	18.59
Speech	89.98	18.42
Math	92.94	17.21
Recovery	104.26	14.39

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#### Table 3

Associations between trait rumination and physiological activity at baseline and physiological reactivity during the TSST and recovery period (N = 157)

	β	(SE)	p-value
Heart Rate	-		
Baseline	0.12	0.24	.150
Preparation	-0.02	0.22	. 681
Speech	-0.03	0.27	.686
Math	-0.02	0.24	.745
Recovery	0.06*	0.10	.050
RSA			
Baseline	-0.06	0.02	.446
Preparation	-0.02	0.03	.850
Speech	0.05	0.03	.582
Math	0.00	0.03	.966
Recovery	-0.06	0.21	.446
PEP			
Baseline	0.02	0.18	.725
Preparation	0.00	0.27	. 968
Speech	0.01	0.29	.852
Math	0.05	0.25	.382
Recovery	0.03	0.15	.415

\* p < .05, 2-sided test