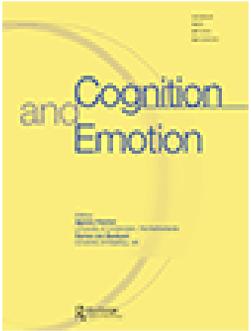

Cognition and Emotion

ISSN: 0269-9931 (Print) 1464-0600 (Online) Journal homepage: http://www.tandfonline.com/loi/pcem20

Emotion regulation and biological stress responding: associations with worry, rumination, and reappraisal

Elizabeth J. Lewis, K. Lira Yoon & Jutta Joormann

To cite this article: Elizabeth J. Lewis, K. Lira Yoon & Jutta Joormann (2017): Emotion regulation and biological stress responding: associations with worry, rumination, and reappraisal, Cognition and Emotion, DOI: 10.1080/02699931.2017.1310088

To link to this article: https://doi.org/10.1080/02699931.2017.1310088

Published online: 11 Apr 2017.

Submit your article to this journal

Article views: 323

View Crossmark data

Citing articles: 2 View citing articles

Full Terms & Conditions of access and use can be found at
http://www.tandfonline.com/action/journalInformation?journalCode=pcem20

COGNITION AND EMOTION, 2017
http://dx.doi.org/10.1080/02699931.2017.1310088

Routledge
Taylor & Francis Group

Check for updates

Emotion regulation and biological stress responding: associations with worry, rumination, and reappraisal

Elizabeth J. Lewis[a], K. Lira Yoon[b] and Jutta Joormann[a]

[a]Department of Psychology, Yale University, New Haven, CT, USA; [b]Department of Psychology, University of Notre Dame, Notre Dame, IN, USA

ABSTRACT

Individual differences in the habitual use of emotion regulation strategies may play a critical role in understanding psychological and biological stress reactivity and recovery in depression and anxiety. This study investigated the relation between the habitual use of different emotion regulation strategies and cortisol reactivity and recovery in healthy control individuals (CTL; $n = 33$) and in individuals diagnosed with social anxiety disorder (SAD; $n = 41$). The tendency to worry was associated with increased cortisol reactivity to a stressor across the full sample. Rumination was not associated with cortisol reactivity, despite its oft-reported similarities to worry. Worry and rumination, however, were associated with increased cortisol during recovery from the stressor. The only difference between CTL and SAD participants was observed for reappraisal. In the CTL but not in the SAD group, reappraisal predicted recovery, such that an increased tendency to reappraise was associated with greater cortisol recovery. These results suggest an important role of the habitual use of emotion regulation strategies in understanding biological stress reactivity and recovery.

ARTICLE HISTORY
Received 15 May 2016
Revised 11 March 2017
Accepted 13 March 2017

KEYWORDS
Cortisol; psychosocial stressor; worry; rumination; reappraisal

Experiencing stressful events in life is impossible to avoid. Given that exposure to these events increases the risk for the onset of depression and anxiety disorders, it is important to identify factors that aid or hinder stress recovery. Indeed, the way in which individuals anticipate and regulate their affective responses to stressors may have greater implications for psychological and physical health outcomes than the stress response during the event itself (Brosschot, Gerin, & Thayer, 2006). The ways in which individuals respond to and try to regulate their affect is frequently referred to as emotion regulation (Gross, 2013). Research on emotion regulation has shown that there are important individual differences in how people respond to stressful events and to the ensuing affect surrounding these events (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Gross, 2002). In addition, individual differences in the use of emotion regulation strategies have been linked to risk for experiencing symptoms of anxiety and depression (Aldao & Nolen-Hoeksema, 2012).

Much of this research, however, has focused on self-reported stress responding, which, while important, only provides limited insight. Investigating the connection between emotion regulation strategies and biological indices of stress responding such as cortisol reactivity and recovery may help to understand the link between emotion regulation and psychopathology. The current study focuses on three frequently employed emotion regulation strategies that have been linked to depression and anxiety (worry, rumination, and reappraisal), and examines associations with cortisol responding.

Worry, the "definitional" feature of generalised anxiety disorder (GAD), is a form of repetitive negative thinking about future events during which individuals feel as if they are anticipating and preparing for future threats (Borkovec, Alcaine, & Behar, 2004). In contrast,

CONTACT Elizabeth J. Lewis elizabeth.lewis@yale.edu
© 2017 Informa UK Limited, trading as Taylor & Francis Group

rumination is a form of repetitive negative thinking about the causes and consequences of one's sad mood, and is generally focused on past events (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Rumination prolongs negative affect and has been found to predict the onset and maintenance of depression (Just & Alloy, 1997; Nolen-Hoeksema, 2000; Nolen-Hoeksema et al., 2008). Rumination may contain a more adaptive component (reflective pondering) and a more maladaptive component (brooding; Joormann, Dkane, & Gotlib, 2006; Treynor, Gonzalez, & Nolen-Hoeksema, 2003). Despite these important differences between worry and rumination, studies have shown that they are correlated and frequently co-occur within individuals (Hughes, Alloy, & Cogswell, 2008; Watkins, 2004).

In contrast to rumination, worry is theorised to blunt affect via the avoidance of threatening imagery (Borkovec & Inz, 1990). The avoidance theory of worry (Borkovec et al., 2004) conceptualises worry as an attempted emotion regulation strategy that "serves to avoid the threatening emotional core of anxiety" (Barlow, 2004, p. 99), and therefore blunts the experience of emotion (Barlow, 2004; Blair & Blair, 2012; Borkovec, Lyonfields, Wiser, & Deihl, 1993). Consistent with this, worry in GAD has been associated with decreased self-report of anxious affect during trauma recall (Behar, Zuellig, & Borkovec, 2005) and with blunted psychophysiological reactivity (Borkovec et al., 1993; Fisher & Newman, 2013).

In addition to worry and rumination, many studies have examined reappraisal, which is frequently considered an adaptive emotion regulation strategy (Gross, 2013; Ochsner & Gross, 2005). Reappraisal involves viewing a situation in a way that alters the situation's meaning and therefore its emotional impact (Gross, 2013). This strategy is effective at modulating one's emotional experience as indicated by decreased self-report of negative affect in response to negative stimuli and decreased emotional expressivity as indexed by lower corrugator activity when viewing negative stimuli (e.g., Urry, 2010). However, the effect of reappraisal on physiological indicators of stress response is mixed (Aldao & Mennin, 2012; Gross, 1998; Urry, 2010).

The current study focused on the activation of the hypothalamic–pituitary–adrenal (HPA) axis, which regulates cortisol release (Dickerson & Kemeny, 2004). This hormone is associated with an organism's "fight or flight" response, which in response to an acute stressor is beneficial to the organism to mobilise resources when threatened; however, chronic activation of the HPA axis is linked to disorders of stress, such as cardiovascular disease and various psychiatric disorders (Brosschot et al., 2006; Danese & McEwen, 2012). While the HPA axis is sensitive to physiological stressors, it is additionally sensitive to psychological and psychosocial stressors (Dickerson & Kemeny, 2004; Kirschbaum, Pirke, & Hellhammer, 1993). This has often been tested in laboratory settings by measuring cortisol levels before and after a stressor such as the Trier Social Stress Test (Kirschbaum et al., 1993).

Many studies examining HPA-axis functioning have subsumed worry and rumination under the construct of perseverative cognition (Brosschot, Pieper, & Thayer, 2005; Brosschot et al., 2006; Zoccola, Dickerson, & Yim, 2011) thereby clouding potentially important differences between these strategies. These studies have generally found perseverative cognition to be associated with increases in cortisol output. For example, one study found that perseverative cognition during the previous day prospectively predicted increases in cortisol output the next morning (Zoccola et al., 2011). One study that specifically assessed worry in the moment found it to be associated with increased plasma cortisol during sexual arousal in males, though it is unclear whether this would generalise to other situations (Rowland et al., 1987).

Studies have observed increased cortisol reactivity or delayed recovery associated with trait rumination (Young & Nolen-Hoeksema, 2001; Zoccola & Dickerson, 2012; Zoccola, Dickerson, & Zaldivar, 2008). Specifically, trait rumination has been associated with delayed cortisol recovery from a social stressor task (Stewart, Mazurka, Bond, Wynne-Edwards, & Harkness, 2013). Interestingly, very few studies have differentiated between patterns of cortisol reactivity and recovery associated with the brooding and pondering subtypes of rumination. One study found that induced brooding rumination was associated with greater cortisol over the course of the experiment (Denson, Spanovic, & Miller, 2009), though no studies to date have investigated the relationship between cortisol and reflective pondering.

Finally, the trait tendency to reappraise has been found to be associated with exaggerated cortisol reactivity to a speech task (Lam, Dickerson, Zoccola, & Zaldivar, 2009). Another study, however, found that trait reappraisal was inversely correlated with cortisol as well as heart rate during a first-time skydiving

experience (Carlson, Dikecligil, Greenberg, & Mujica-Parodi, 2012). Importantly, few studies have examined the relation between the use of these regulation strategies and cortisol responding in healthy compared to clinical participants.

The current study investigates how individual differences in the habitual use of worry, rumination, and reappraisal are associated with cortisol reactivity and recovery in healthy control and clinical participants. Very few studies have assessed different emotion regulation tendencies in the same study without collapsing across these strategies, preventing any discussion of the specificity of relationships between emotion regulation strategies and cortisol reactivity and recovery. Additionally, the current study extends previous work by investigating both brooding and reflective subtypes of rumination, and investigating correlations with both cortisol reactivity and recovery. Healthy control individuals and participants with social anxiety disorder (SAD) were recruited for participation in the study, as SAD is characterised by increased rates of repetitive negative thinking (Morrison & Heimberg, 2013; Nolen-Hoeksema et al., 2008).

Indeed, there is evidence that cortisol reactivity to social stressors is altered in SAD, though the directionality is inconsistent across studies (Phan & Klumpp, 2010). While current evidence suggests that SAD is not characterised by differing basal levels of cortisol, most studies investigating cortisol reactivity and recovery to a psychosocial stressor find altered stress responding in SAD, whether increased or decreased (Phan & Klumpp, 2010). For example, several studies have found increased cortisol responding associated with psychosocial stressor inductions (Condren, O'Neill, Ryan, Barrett, & Thakore, 2002; Roelofs et al., 2009; van West, Claes, Sulon, & Deboutte, 2008), while several studies have found cortisol to be decreased (Beaton et al., 2006; Shirotsuki et al., 2009). Finally, some have not found differences between patients and control participants (Klumbies, Braeuer, Hoyer, & Kirschbaum, 2014; Krämer et al., 2012; Martel et al., 1999). One study found a little over a third of the SAD sample to be "responders" to the psychosocial stressor, in that cortisol was increased to a psychosocial stressor, while the rest were deemed "nonresponders" (Furlan, DeMartinis, Schweizer, Rickels, & Lucki, 2001). A recent meta-analysis concluded that individual differences such as gender might be critically important, reporting increased cortisol reactivity in males with SAD but not females (Zorn et al., 2016). These inconsistencies prompt the need for investigating other individual difference variables that might be critically important for biological stress responding (Elzinga, Spinhoven, Berretty, de Jong, & Roelofs, 2010; Phan & Klumpp, 2010), including one's tendency to engage in perseverative cognition.

Given the proposed physiologically blunting nature of worry (e.g., Borkovec et al., 1993), it was hypothesised that trait worry is associated with decreased cortisol reactivity to an acute stressor and reduced recovery. In contrast, it was hypothesised that trait rumination is associated with increased reactivity due to the emotionally prolonging nature of rumination (e.g., Nolen-Hoeksema et al., 2008). Additionally, decreased recovery was expected, such that increased trait rumination is associated with smaller decreases in cortisol over the recovery period. It was finally hypothesised that trait reappraisal is associated with greater decreases in cortisol over the recovery period. It was expected that these effects would be moderated by group.

Methods

Participants

Participants were recruited through Internet bulletin boards as well as a local newspaper. All participants underwent the Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 1996) to confirm absence or presence of psychopathology. Exclusion criteria for the control participants included the existence of any past or current Axis I disorder, head trauma or losses of consciousness, acute or chronic illnesses that are known to interfere with HPA activity (see McEwen, 1998), or use of medication that interferes with HPA activity except for psychiatric medication or oral contraceptives (see Kudielka, Hellhammer, & Wüst, 2009). Exclusion criteria for the clinical group differed in that current and past comorbid disorders were allowed except for a diagnosis of bipolar disorder, any psychotic symptoms, or alcohol or substance abuse within the past six months. There were 33 healthy control (CTL) participants and 41 participants with a primary diagnosis of SAD (24 of whom were also diagnosed with major depressive disorder [MDD]). A subset of the data investigating group differences in cortisol reactivity is reported in Yoon and Joormann (2012). The current study, however, examined a larger sample and focused on the relation between emotion regulation and cortisol reactivity/recovery and whether this is moderated by clinical status.

Measures

Emotion Regulation Questionnaires

Worry and Reappraisal were assessed by the worry and reappraisal subscales of the Thought Control Questionnaire (TCQ; Wells & Davies, 1994). The TCQ is a 30-item self-report measure wherein participants rate on a scale of 1–4 ("never" – "almost always") how often they use these strategies to control their thoughts. The TCQ has adequate to good internal consistency as well as adequate to very good test–retest reliability. Rumination was assessed with Reflective Pondering and Brooding subscales of the Ruminative Responses Scale of the Response Style Questionnaire (RRS; Nolen-Hoeksema & Morrow, 1991, which is a 22-item self-report measure. This scale assesses participants' responses to possible causes and consequences of their depressed mood on a scale of 1–4 ("almost never – almost always").

Symptom measures

Participants additionally completed the Beck Depression Inventory – II in order to assess the severity of depressive symptoms (BDI; Beck, Steer, & Brown, 1996). The BDI-II is a 21-item self-report assessment of depressive symptoms experienced over the past two weeks. The BDI-II has adequate to good construct validity and test–retest reliability (Dozois, Dobson, & Ahnberg, 1998). The Liebowitz Social Anxiety Scale was also included, which assesses fear and avoidance across 24 social situations on a scale from 0 to 3 (LSAS; Liebowitz, 1987). The LSAS has good test–retest reliability and internal consistency, as well as convergent and discriminant validity.

Affect ratings

Participants completed mood ratings seven times over the course of the study session. Participants rated on a 10-point Likert-type scale ranging from 0 ("not at all") to 9 ("extremely") how "nervous" they were feeling at the time they were prompted.

Psychosocial stressor task

The psychosocial stress task comprised a social-evaluative task as well as a cognitive task, and is a variant of the Trier Social Stress Test (Kirschbaum et al., 1993). There is meta-analytic evidence that the combination of these two tasks is the best elicitor of an acute cortisol response (Dickerson & Kemeny, 2004).

The task started with a 10-minute waiting period, during which participants gave a saliva sample/affect rating at the midpoint (5 minutes in, Baseline 1) and after the full waiting period (10 minutes in, Baseline 2). Participants were then told that they would have to make a speech, and cortisol was collected (with mood rating) after this five-minute anticipation phase (Anticipation). After participants performed their five-minute speech, participants completed two subtests of the Wechsler Adult Intelligence Scale (WAIS; Wechsler, 2008), after five minutes of which cortisol/affect were again assessed (Post-stress). Finally, three recovery samples were collected every 10 minutes during the 30-minute recovery period, during which participants watched a nature video (Recovery 1–3).

Cortisol assay

Salivette swabs were used to collect cortisol samples from saliva (Sarsedt, Nümbrecht, Germany). The samples were kept frozen at a temperature of −20°C until shipment to a cortisol assay laboratory. Samples were centrifuged at 3000 rpm for 5 minutes before analysis to produce a clear supernatant with low viscosity. For cortisol analysis, 50 µL were subsequently removed using a commercially available immunoassay with chemoluminescence detection, which has a lower detection limit of 0.43 nmol/L. Inter- and intra-assay coefficients of variation were below 8% for both low (3 nmol/L) and high (25 nmol/L) cortisol levels.

Procedure

Participants came in for two study sessions. In the first session they gave informed consent, completed the clinical interview, and completed the questionnaires. If participants were invited to participate in the second study session (as per exclusion criteria described above), participants were provided with written and verbal instructions to refrain from eating, drinking (anything other than water), smoking tobacco, or exercising two hours before the study session. All second study sessions were completed between 2:00 PM and 4:00 PM to minimise the effects of diurnal cortisol fluctuation. In session two, participants completed the psychosocial stressor task, including the speech and cognitive stressors, as well as the recovery during which participants watched a soothing nature video. Participants were then debriefed and compensated for their time.

Data analysis strategy

In the following sections, manipulation checks using repeated measures analyses of variance (ANOVAs) were performed in order to assess whether the psychosocial stressor task was effective in inducing anxiety and a change in cortisol over the course of the paradigm. The cortisol indices were then averaged during the baseline timepoints as well as the recovery timepoints due to high correlations between these indices, and difference scores were created in order to index cortisol reactivity and recovery. Self-report indices were then correlated with the measures of cortisol reactivity and recovery, and hierarchical linear regressions were then completed to follow up on significant correlations.

Results

Participant characteristics

The clinical and control groups differed significantly on age, $t(71) = 2.64$, $p = .010$, though groups did not differ on gender composition, $\chi^2(1) = .82$, $p = .365$. Consequently, age was included in all analyses that looked at group differences but was not a significant predictor. The clinical group had both higher LSAS, $t(69) = 14.30$, $p < .001$, and BDI scores, $t(69) = 10.37$, $p < .001$, corroborating our diagnoses. The SAD and CTL samples also differed on most of the trait emotion regulation measures. The clinical group had higher total RRS scores, $t(70) = 11.73$, $p < .001$, as well as higher RRS Brooding subscale scores, $t(70) = 13.41$, $p < .001$, and higher RRS Reflective Pondering subscale scores, $t(70) = 8.40$, $p < .001$. The clinical compared to the control group also exhibited greater TCQ-Worry scores, $t(69) = 7.22$, $p < .001$. Interestingly, controls and the clinical group did not differ in TCQ-Reappraisal scores, $t(69) = -0.64$, $p = .526$. Participant characteristics are reported in full in Table 1.

Table 1. Participant characteristics.

Characteristic	CTL ($N = 33$)	SAD ($N = 41$)
Age (years)	37.73 (10.67)**	32.90 (11.24)**
Sex (female:male)	13:20	20:20
BDI	3.88 (5.53)***	29.45 (13.01)***
LSAS	17.80 (13.47)***	91.47 (26.45)***
RRS total	31.15 (9.71)***	65.46 (14.23)***
RRS brooding	7.06 (2.40)***	16.33 (3.30)***
RRS ref. pondering	7.15 (2.68)***	13.08 (3.21)***
TCQ-Worry	7.56 (1.61)***	12.74 (3.78)***
TCQ-Reappraisal	13.41 (3.88)	13.95 (3.30)
Nervousness rating 1	0.50 (1.14)***	2.67 (3.04)***
Nervousness rating 2	0.48 (1.06)***	2.58 (2.89)***
Nervousness rating 3	1.65 (2.18)***	4.72 (3.56)***
Nervousness rating 4	1.03 (1.65)***	3.66 (3.49)***
Nervousness rating 5	0.13 (0.42)**	1.74 (2.75)**
Nervousness rating 6	0.10 (0.41)**	1.59 (2.74)**
Nervousness rating 7	0.10 (0.40)**	1.71 (2.92)**
Cortisol sample 1	5.99 (4.47)	6.59 (3.63)
Cortisol sample 2	5.75 (3.47)	6.72 (4.01)
Cortisol sample 3	5.30 (2.84)	6.54 (3.88)
Cortisol sample 4	5.24 (2.61)	6.98 (5.18)
Cortisol sample 5	5.24 (2.99)	6.56 (4.62)
Cortisol sample 6	4.99 (2.61)	5.74 (3.81)
Cortisol sample 7	5.04 (4.04)	5.06 (3.06)

Notes: *$p < .05$; **$p < .01$; and ***$p < .001$ (2-tailed). RRS Ref. Pondering = RRS Reflective Pondering.

Relations among emotion regulation strategies

Several of the trait emotion regulation measures investigated were correlated with each other in the full sample and in the subgroups (see Table 2). In the full sample (collapsing across groups), worry was significantly correlated with brooding ($r[71] = .64$, $p < .001$), reflective pondering ($r[71] = .66$, $p < .001$), and reappraisal ($r[71] = .31$, $p = .008$). Reflective pondering was additionally significantly correlated with brooding ($r[71] = .89$, $p < .001$) and reappraisal ($r[71] = .30$, $p = .011$).

In the clinical group, there was a correlation between worry and reflective pondering ($r[39] = .47$, $p = .003$) and a correlation between worry and reappraisal ($r[39] = .50$, $p = .001$). There was additionally a correlation between reflective pondering and both

Table 2. Zero-order correlations between emotion regulation strategies.

	Full *sample* ($N = 74$)			
	1	2	3	4
1. Worry	–			
2. Ref. pond.	.66**	–		
3. Brooding	.64**	.89**	–	
4. Reappraisal	.31**	.30*	.20	–
	SAD ($N = 41$)			
	1	2	3	4
1. Worry	–			
2. Ref. pond.	.47**	–		
3. Brooding	.24	.73**	–	
4. Reappraisal	.50**	.38*	.21	–
	CTL ($N = 33$)			
	1	2	3	4
1. Worry	–			
2. Ref. pond.	.06	–		
3. Brooding	.08	.89**	–	
4. Reappraisal	.11	.33	.31	–

Notes: *$p < .05$; **$p < .01$ (both 2-tailed). Ref. Pond. = Reflective Pondering.

brooding ($r[39] = .73$, $p < .001$) and reappraisal ($r[39] = .38$, $p = .018$) in the clinical group. In control participants, there was a correlation between reflective pondering and brooding ($r[33] = .89$, $p < .001$).

Manipulation check

In order to assess the ability of our psychosocial stressor task to induce anxiety, ratings of nervousness that were taken across the study were analysed with a repeated measures ANOVA. The time (7) × group (2) ANOVA revealed a main effect of time, $F(6, 342) = 21.95$, $p < .001$, $\eta_p^2 = .28$, which was qualified by a significant interaction of group × time, $F(6, 342) = 3.50$, $p = .002$, $\eta_p^2 = .06$ (Table 1). To follow up on this significant interaction, two within-group analyses were completed. The main effect of time was significant for both groups ($F[6, 138] = 7.15$, $p < .001$, $\eta_p^2 = .24$ for CTL[1]; $F[6, 204] = 19.43$, $p < .001$, $\eta_p^2 = .37$ for SAD[2]). Most importantly, both groups showed a significant increase in nervousness from the end of the baseline period (nervousness rating 2) to the time of the speech (i.e. speech anticipation, nervousness rating 3), $F(1, 29) = 11.52$, $p = .002$, $\eta_p^2 = .28$ for CTL, $F(1, 38) = 37.86$, $p < .001$, $\eta_p^2 = .50$ for SAD. Therefore, our stressor was effective in inducing feelings of nervousness in both the SAD and CTL groups.

In order to assess whether the stressor affected cortisol levels over the course of the paradigm, a repeated measures ANOVA was conducted for all time points of cortisol collection (7) × group (2). This revealed both a main effect of time, $F(6, 426) = 8.31$, $p < .001$, $\eta_p^2 = .11$, and an interaction between group and time, $F(6, 426) = 3.18$, $p = .005$, $\eta_p^2 = .04$ (Table 1). To follow up on this significant interaction, a repeated measures ANOVA was completed for each group. This revealed that control individuals demonstrated a significant change in cortisol over the course of the paradigm, $F(6, 186) = 2.64$, $p = .018$, $\eta_p^2 = .08$. The clinical group also demonstrated a significant change in cortisol over the course of the paradigm, $F(6, 240) = 9.59$, $p < .001$, $\eta_p^2 = .19$.

Relations of emotion regulation strategies to cortisol

Emotion regulation strategies were correlated with cortisol indices of reactivity and recovery. Given the high correlation between the two baseline measures of cortisol, we collapsed across these two indices to form one measure of baseline cortisol ($r[74] = .94$, $p < .001$; $\alpha = .97$). Likewise, due to the high correlations between the three recovery measures of cortisol, we collapsed across them to form one measure of recovery ($\alpha = .96$). Cortisol reactivity was computed by subtracting cortisol at baseline from cortisol at post-stress. Likewise, cortisol recovery was computed by subtracting cortisol during recovery from cortisol at post-stress.

In the full sample, cortisol reactivity (post-stress cortisol – baseline cortisol) was correlated positively with both worry ($r[71] = .34$, $p = .003$) and reappraisal ($r[71] = .25$, $p = .040$; see Table 3). In individuals with SAD, cortisol reactivity was associated with worry ($r[41] = .39$, $p = .014$). In CTL individuals, there were no significant correlations between cortisol reactivity and emotion regulation strategies (all p's > .05).

In all participants, cortisol recovery (post-stress – recovery cortisol) was correlated with worry ($r[71] = .37$, $p = .002$), brooding ($r[71] = .29$, $p = .015$), and reflective pondering ($r[71] = .34$, $p = .004$). In individuals with SAD, there were no significant correlations of the trait emotion regulation measures with cortisol recovery (all p's > .05). In CTL participants, there was a significant correlation between cortisol recovery and reappraisal ($r[32] = .46$, $p = .007$). No other correlations reached significance.

Association between emotion regulation and cortisol reactivity and recovery: moderation by group

Finally, using a moderation analysis, group differences in the relation between cortisol reactivity/recovery and emotion regulation indices were investigated. Because significant correlations were found between cortisol reactivity and worry and reappraisal, a linear regression was completed investigating the effects of worry, reappraisal, and group on cortisol reactivity (post-stress cortisol – baseline).[3] This revealed a main effect of worry, $\beta = .33$, $t(67) = 2.05$, $p = .044$, $R^2 = .14$ (see Table 4). While the ΔR^2 for the second step of the model was significant, indicating that the model was improved by adding in the interaction terms between worry and reappraisal and group, the interaction between worry and group did not reach significance, $\beta = .47$, $t(65) = 1.33$, $p = .187$, $\Delta R^2 = .03$.[4]

To follow up on the significant correlations for cortisol recovery, linear regressions were conducted for emotion regulation strategies and cortisol recovery (post-stress cortisol – recovery cortisol). In investigating the association between reflective pondering, brooding, worry, reappraisal, and group on cortisol

Table 3. Zero-order correlations between emotion regulation strategies and cortisol reactivity and recovery.

	Full Sample ($N = 74$)		SAD ($N = 41$)		CTL ($N = 33$)	
Strategy	React.	Recov.	React.	Recov.	React.	Recov.
Worry	.34**	.37**	.39*	.28	−.04	.07
Ref. pond.	.17	.34**	.05	.14	.01	.29
Brooding	.15	.29*	−.07	−.02	.04	.24
Reappraisal	.25*	.19	.20	.01	.32	.46**
Mean	− 0.10	0.73	0.33	1.20	− 0.63	0.15
SD	2.34	1.73	2.61	1.92	1.85	1.28

Notes: *$p < .05$; **$p < .01$ (both 2-tailed). Reactivity = post-stress – baseline; recovery = post-stress – recovery; Ref. Pond. = Reflective Pondering. Salivary cortisol difference scores are in nmol/L.

recovery, an interaction between reappraisal and group was found, $\beta = -.35$, $t(61) = -2.00$, $p = .050$, $\Delta R^2 = .06$ (see Table 4). This indicated that the second step of the model accounted for an additional 6% of the variance.

In order to decompose this interaction, a moderation analysis was performed (Hayes, 2013). This revealed that the effect of reappraisal on cortisol recovery was being driven by CTL individuals, $t(31) = 2.21$, $p = .030$, while reappraisal did not have an effect on cortisol recovery in the SAD group, $t(38) = -1.45$, $p = .153$ (Figure 1).

Table 4. Hierarchical regression with worry, reappraisal, and group status predicting Reactivity (post-stress – baseline) cortisol. Hierarchical regression with reflective pondering, brooding, worry, reappraisal, and group status predicting Recovery (post-stress – recovery) cortisol.

DV: Reactivity	β	R^2	Δ R^2
Step 1		.14*	
Group	−.05		
Worry	.33*		
Reappraisal	.15		
Step 2		.17*	.03*
Group	.09		
Worry	−.15		
Reappraisal	.23		
Group × worry	.47		
Group × reap.	−.16		
DV: Recovery	β	R^2	Δ R^2
Step 1		.17*	
Group	.22		
Ref. P .	.33		
Brooding	−.34		
Worry	.20		
Reappraisal	.08		
Step 2		.23*	.06*
Group	.21		
Ref. P .	.27		
Brooding	−.17		
Worry	.03		
Reappraisal	.28		
Group × Ref. P.	.03		
Group × brood.	−.14		
Group × worry	.26		
Group × Reap.	−.35*		

Notes: *$p < .05$; **< (2-tailed). Reap. = Reappraisal; Ref. P. = Reflective Pondering; Brood. = Brooding.

Discussion

The current study sought to investigate the association between biological stress reactivity and recovery and habitual use of emotion regulation strategies. A relation between cortisol reactivity and tendency to worry was observed, such that an increased tendency to worry predicted increased cortisol reactivity, contrary to expectations that worry would be associated with blunted cortisol reactivity. This result is, however, in line with previous work suggesting that worry might be associated with increased cortisol reactivity, though much of this work collapsed across worry and rumination (Brosschot et al., 2006). However, the differences in findings between worry being blunting or physiologically activating may be due to the distinction between investigating trait worry versus state inductions of worry. Many of the findings of worry being physiologically blunting have been observed during state inductions of worry (Borkovec et al., 1993). Chronic worry may indeed prolong stress responding, though it may not be associated with acute spikes in physiological stress responding. This prolonged stress responding may be the mechanism by which perseverative cognition is related to chronic disorders of stress responding such as cardiovascular disease (Brosschot et al., 2006).

In addition, cortisol reactivity was not associated with rumination, in contrast to the hypothesis that rumination would be associated with increased reactivity. This is additionally in conflict with previous work showing increased cortisol reactivity being associated with rumination, although findings are mixed depending on the type of rumination studied (Zoccola & Dickerson, 2012). This may mean that worry is driving the effects of increased cortisol reactivity in studies that collapse across worry and

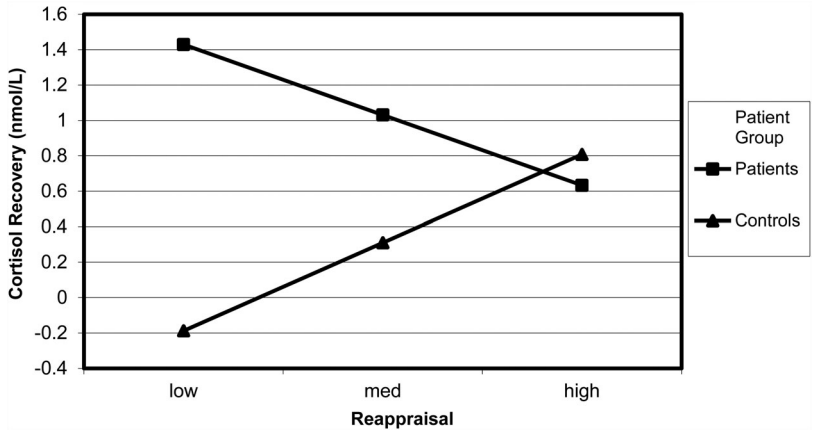

Figure 1. Interaction between reappraisal and patient group on cortisol recovery.

rumination, though future research is necessary to replicate this finding (Brosschot et al., 2006). It may be important for future research to elaborate theoretically on how worry might work to maintain physiological activation in a way that rumination might not. Indeed, potential future avenues for research might be to parse out physiological consequences previously thought to be attributable to perseverative cognition as a whole that might solely be traceable to worry.

Interestingly, tendency towards reappraisal was associated with cortisol recovery in control participants only; as trait reappraisal increased, healthy control individuals showed greater cortisol recovery, while individuals with SAD did not show this benefit. This is in line with the hypothesis that reappraisal is associated with greater recovery, and that this effect would be moderated by group. This finding is particularly interesting because individuals with SAD and healthy control individuals did not differ in their overall reported tendency towards the use of reappraisal. Perhaps this indicates a difficulty engaging in reappraisal in an effective way in this disorder. Importantly, the current findings do not lend support for the theory that reappraisal is an effortful process that increases cortisol reactivity and delays recovery; this finding may be specific to reappraisal inductions, especially if individuals are not used to or practiced at reappraisal (Denson, Creswell, Terides, & Blundell, 2014). These authors conclude that reappraisal is an effortful emotion regulation strategy that, though it fosters a sense of self-efficacy and is effective at decreasing negative affect, potentiates cortisol reactivity (Denson et al., 2014; Jamieson, Mendes, & Nock, 2013). However, future work is needed to investigate whether the increase in cortisol reactivity and delayed recovery associated with induced reappraisal is moderated by one's tendency to reappraise. It is possible that individuals with SAD are not as effective at using this strategy as control participants, or that there is a skill deficit that precludes the effective use of reappraisal in a way that is associated with benefits that are reflected in biological stress reactivity. Given that reappraisal is a key skill focused on in cognitive-behavioural therapy, it may be that one would see changes in stress reactivity over the course of treatment due to practicing the effective use of reappraisal. Training in reappraisal, as is often done in cognitive-behavioural therapy, is theorised to potentially aid in decreasing chronic activation of the HPA axis in those with psychological disorders (Denson et al., 2009; John & Gross, 2004). Indeed, one study found that training in reappraisal is associated with decreases in cortisol reactivity (Gaab et al., 2003), though in this study the intervention also involved relaxation training, so the specific effects of reappraisal training cannot be ascertained. Therefore, longitudinal studies assessing trait and state reappraisal and stress reactivity pre- and post-treatment may be necessary to resolve this question.

Upon investigating the relations between emotion regulation strategies and cortisol recovery (post-stress – recovery cortisol), recovery from the stressor was found to be correlated with worry as well as brooding and reflective pondering. This was contrary to our hypotheses that worry and rumination would be associated with blunted recovery for different reasons; worry for its blunting of reactivity and rumination for its perpetuating reactivity throughout the

recovery period. Therefore, higher levels of worry and rumination were both expected to correlate with blunted recovery. It is interesting that worry is associated both with reactivity and recovery, suggesting that high worriers have greater reactivity to the stressor, which would mean both that worry is not an effective strategy for blunting reactivity and that it may be an "inefficient" strategy for emotion regulation (Andreescu et al., 2015). Strangely, this greater drop in cortisol during the recovery is unexplained by this theory, though it could reflect having a greater magnitude to fall due to the greater reactivity. It is therefore even more interesting that an oft-cited similar strategy, rumination, is not associated with greater increases during the stressor, but is associated in this study with greater decreases during recovery (McEvoy, Mahoney, & Moulds, 2010).

It may also be that worry is associated with physiological blunting when worry is overused as a strategy for emotion regulation among those with pathological or uncontrollable worry, such as in GAD (Borkovec et al., 1993). Though SAD and GAD are highly comorbid, uncontrollable worry is a central feature of GAD (Blair & Blair, 2012). Individuals with uncontrollable worry may differ from those with high trait worry in terms of stress reactivity, such that worry is associated with blunting only in those with uncontrollable worry. It is possible that individuals with SAD do not necessarily struggle with uncontrollable worry, and perhaps the correlation with worry in cortisol reactivity is reflecting anticipatory anxiety in SAD, which may not in fact blunt physiological responding but might amplify it. Indeed, in socially anxious individuals, anticipatory processing before a speech task versus distraction was found to increase self-reported anxiety as well as skin-conductance (Wong & Moulds, 2011). Future studies may want to investigate this question in a group of individuals with chronic and uncontrollable worry to investigate this potential distinction.

The current study has several limitations. Although individuals with SAD report similar levels of repetitive negative thinking and generally show increased cortisol reactivity to psychosocial stressor tasks (Condren et al., 2002; Morrison & Heimberg, 2013), future work may want to extend this investigation to other patient groups that might specifically experience uncontrollable worry and rumination, such as GAD and MDD (Borkovec & Inz, 1990; Nolen-Hoeksema et al., 2008). Additionally, given differences observed in previous work between induced emotion regulation strategies and trait tendency towards use of emotion regulation strategies, future work may want to induce emotion regulation strategies and see whether the interaction between state and trait emotion regulation strategies can better predict biological stress responding. Despite this, the current study is the first to investigate several emotion regulation strategies in the same study in order to assess their specific contributions to cortisol reactivity and recovery.

Conclusion

In sum, this study found several associations between emotion regulation strategies and cortisol reactivity and recovery using a variant of the Trier Social Stress Task. The habitual use of worry was found to be related to cortisol reactivity regardless of group, suggesting that high trait levels of worry increase biological stress responding to an acute stressor regardless of the presence or absence of psychopathology. Interestingly, the relation between trait reappraisal and cortisol recovery was found to be moderated by group, such that for control participants who use reappraisal more frequently, cortisol recovery from the stressor was greater. Individuals with SAD who were high on trait reappraisal did not reap these benefits. This may indicate that while cognitive-behavioural therapy focuses on teaching skills such as reappraisal, individuals with SAD may additionally need help in implementing these strategies in a way that aids in diminishing physiological stress responding. Since in this study healthy controls and individuals with SAD did not differ in terms of tendency to reappraise, the way in which individuals engage in reappraisal may be crucial. The current study points to the complex relations between various emotion regulation strategies and biological stress responding. Further inquiry is necessary to investigate the manner in which individuals with clinical disorders engage in these strategies and how this is manifested in stress reactivity and recovery.

Notes

1. Due to missing data, this affect rating is only available for 24 CTL individuals.
2. Due to missing data, this affect rating is only available for 35 individuals with SAD.
3. We additionally examined moderation in the emotion regulation strategies that did not show significant zero-order correlations with cortisol reactivity (brooding and

reflective pondering). Neither subtype of rumination had a main effect or interacted with group to predict cortisol reactivity (brooding interaction: $\beta = -.12$, $t(68) = -.43$, $p = .665$, $\Delta R^2 = .00$; reflective pondering interaction: $\beta = .05$, $t(68) = 0.20$, $p = .839$, $\Delta R^2 = .00$).

4. When separating out worry from reappraisal in two separate regressions, the interaction terms are not significant even though the worry model shows a significant increase in model fit, $\beta = .42$, $t(67) = 1.22$, $p = .229$, $\Delta R^2 = .02$. In contrast, the reappraisal model does not have a significant interaction term nor does it have a significant increase in model fit when including the interaction term, $\beta = .01$, $t(67) = 0.08$, $p = .935$, $\Delta R^2 = .00$.

Disclosure statement

No potential conflict of interest was reported by the authors.

References

Aldao, A., & Mennin, D. S. (2012). Paradoxical cardiovascular effects of implementing adaptive emotion regulation strategies in generalized anxiety disorder. *Behaviour Research and Therapy*, *50*(2), 122–130. doi:10.1016/j.brat.2011.12.004

Aldao, A., & Nolen-Hoeksema, S. (2012). When are adaptive strategies most predictive of psychopathology? *Journal of Abnormal Psychology*, *121*(1), 276–281. doi:10.1037/a0023598

Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: A meta-analytic review. *Clinical Psychology Review*, *30*(2), 217–237. doi:10.1016/j.cpr.2009.11.004

Andreescu, C., Sheu, L. K., Tudorascu, D., Gross, J. J., Walker, S., Banihashemi, L., & Aizenstein, H. (2015). Emotion reactivity and regulation in late-life generalized anxiety disorder: Functional connectivity at baseline and post-treatment. *The American Journal of Geriatric Psychiatry: Official Journal of the American Association for Geriatric Psychiatry*, *23*(2), 200–214. doi:10.1016/j.jagp.2014.05.003

Barlow, D. H. (2004). *Anxiety and its disorders: The nature and treatment of anxiety and panic*. New York, NY: Guilford Press.

Beaton, E. A., Schmidt, L. A., Ashbaugh, A. R., Santesso, D. L., Antony, M. M., McCabe, R. E., … Schulkin, J. (2006). Low salivary cortisol levels among socially anxious young adults: Preliminary evidence from a selected and a non-selected sample. *Personality and Individual Differences*, *41*(7), 1217–1228. doi:10.1016/j.paid.2006.02.020

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *BDI-II manual for the Beck Depression Inventory-II*. San Antonio, TX: The Psychological Corporation.

Behar, E., Zuellig, A., & Borkovec, T. (2005). Thought and imaginal activity during worry and trauma recall. *Behavior Therapy*, *36*(2), 157–168.

Blair, K. S., & Blair, R. J. R. (2012). A cognitive neuroscience approach to generalized anxiety disorder and social phobia. *Emotion Review*, *4*(2), 133–138. doi:10.1177/1754073911430251

Borkovec, T. D., Alcaine, O., & Behar, E. (2004). Avoidance theory of worry and generalized anxiety disorder. Generalized anxiety disorder. In R. G. Heimberg, C. L. Turk, & D. S. Mennin (Eds.), *Advances in research and practice* (pp. 77–108). New York: Guilford Press.

Borkovec, T. D., & Inz, J. (1990). The nature of worry in generalized anxiety disorder: A predominance of thought activity. *Behaviour Research and Therapy*, *28*(2), 153–158.

Borkovec, T. D., Lyonfields, J. D., Wiser, S. L., & Deihl, L. (1993). The role of worrisome thinking in the suppression of cardiovascular response to phobic imagery. *Behaviour Research and Therapy*, *31*(3), 321–324.

Brosschot, J. F., Gerin, W., & Thayer, J. F. (2006). The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health. *Journal of Psychosomatic Research*, *60*(2), 113–124. doi:10.1016/j.jpsychores.2005.06.074

Brosschot, J. F., Pieper, S., & Thayer, J. F. (2005). Expanding stress theory: Prolonged activation and perseverative cognition. *Psychoneuroendocrinology*, *30*(10), 1043–1049. doi:10.1016/j.psyneuen.2005.04.008

Carlson, J. M., Dikecligil, G. N., Greenberg, T., & Mujica-Parodi, L. R. (2012). Trait reappraisal is associated with resilience to acute psychological stress. *Journal of Research in Personality*, *46*(5), 609–613. doi:10.1016/j.jrp.2012.05.003

Condren, R. M., O'Neill, A., Ryan, M. C. M., Barrett, P., & Thakore, J. H. (2002). Hpa axis response to a psychological stressor in generalised social phobia. *Psychoneuroendocrinology*, *27*(6), 693–703.

Danese, A., & McEwen, B. S. (2012). Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiology & Behavior*, *106*(1), 29–39. doi:10.1016/j.physbeh.2011.08.019

Denson, T. F., Creswell, J. D., Terides, M. D., & Blundell, K. (2014). Cognitive reappraisal increases neuroendocrine reactivity to acute social stress and physical pain. *Psychoneuroendocrinology*, *49*, 69–78. doi:10.1016/j.psyneuen.2014.07.003

Denson, T. F., Spanovic, M., & Miller, N. (2009). Cognitive appraisals and emotions predict cortisol and immune responses: A meta-analysis of acute laboratory social stressors and emotion inductions. *Psychological Bulletin*, *135*(6), 823–853. doi:10.1037/a0016909

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, *130*(3), 355–391. doi:10.1037/0033-2909.130.3.355

Dozois, D. J. A., Dobson, K. S., & Ahnberg, J. L. (1998). A psychometric evaluation of the Beck Depression Inventory – II. *Psychological Assessment*, *10*(2), 83–89. doi:10.1037/1040-3590.10.2.83

Elzinga, B. M., Spinhoven, P., Berretty, E., de Jong, P., & Roelofs, K. (2010). The role of childhood abuse in HPA-axis reactivity in social anxiety disorder: A pilot study. *Biological Psychology*, *83*(1), 1–6.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. (1996). *Structured clinical interview for DSM-IV*. Washington, DC: American Psychiatric Association.

Fisher, A. J., & Newman, M. G. (2013). Heart rate and autonomic response to stress after experimental induction of worry versus relaxation in healthy, high-worry, and generalized anxiety disorder individuals. *Biological Psychology*, *93*(1), 65–74. doi:10.1016/j.biopsycho.2013.01.012

Furlan, P. M., DeMartinis, N., Schweizer, E., Rickels, K., & Lucki, I. (2001). Abnormal salivary cortisol levels in social phobic patients in response to acute psychological but not physical stress. *Biological Psychiatry*, *50*(4), 254–259.

Gaab, J., Blättler, N., Menzi, T., Pabst, B., Stoyer, S., & Ehlert, U. (2003). Randomized controlled evaluation of the effects of cognitive-behavioral stress management on cortisol responses to acute stress in healthy subjects. *Psychoneuroendocrinology*, *28*(6), 767–779.

Gross, J. J. (1998). Antecedent- and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology*, *74*(1), 224–237. doi:10.1037/0022-3514.74.1.224

Gross, J. J. (2002). Emotion regulation: Affective, cognitive, and social consequences. *Psychophysiology*, *39*, 281–291.

J. J. Gross (Ed.). (2013). *Handbook of emotion regulation*. New York, NY: Guilford.

Hayes, A. F. (2013). *Introduction to mediation, moderation, and conditional process analysis: A regression-based approach*. New York, NY: Guilford Press.

Hughes, M. E., Alloy, L. B., & Cogswell, A. (2008). Repetitive thought in psychopathology: The relation of rumination and worry to depression and anxiety symptoms. *Journal of Cognitive Psychotherapy*, *22*(3), 271–288. doi:10.1891/0889-8391.22.3.271

Jamieson, J. P., Mendes, W. B., & Nock, M. K. (2013). Improving acute stress responses the power of reappraisal. *Current Directions in Psychological Science*, *22*(1), 51–56. doi:10.1177/0963721412461500

John, O. P., & Gross, J. J. (2004). Healthy and unhealthy emotion regulation: Personality processes, individual differences, and life span development. *Journal of Personality*, *72*(6), 1301–1334. doi:10.1111/j.1467-6494.2004.00298.x

Joormann, J., Dkane, M., & Gotlib, I. (2006). Adaptive and maladaptive components of rumination? Diagnostic specificity and relation to depressive biases. *Behavior Therapy*, *37*(3), 269–280. doi:10.1016/j.beth.2006.01.002

Just, N., & Alloy, L. B. (1997). The response styles theory of depression: Tests and an extension of the theory. *Journal of Abnormal Psychology*, *106*(2), 221–229.

Kirschbaum, C., Pirke, K. M., & Hellhammer, D. H. (1993). The "Trier Social Stress Test" – a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology*, *28*(1–2), 76–81.

Klumbies, E., Braeuer, D., Hoyer, J., & Kirschbaum, C. (2014). The reaction to social stress in social phobia: Discordance between physiological and subjective parameters. *PloS One*, *9*(8), e105670. doi:10.1371/journal.pone.0105670

Krämer, M., Seefeldt, W. L., Heinrichs, N., Tuschen-Caffier, B., Schmitz, J., Wolf, O. T., & Blechert, J. (2012). Subjective, autonomic, and endocrine reactivity during social stress in children with social phobia. *Journal of Abnormal Child Psychology*, *40*(1), 95–104. doi:10.1007/s10802-011-9548-9

Kudielka, B. M., Hellhammer, D. H., & Wüst, S. (2009). Why do we respond so differently? Reviewing determinants of human salivary cortisol responses to challenge. *Psychoneuroendocrinology*, *34*(1), 2–18. doi:10.1016/j.psyneuen.2008.10.004

Lam, S., Dickerson, S. S., Zoccola, P. M., & Zaldivar, F. (2009). Emotion regulation and cortisol reactivity to a social-evaluative speech task. *Psychoneuroendocrinology*, *34*(9), 1355–1362. doi:10.1016/j.psyneuen.2009.04.006

Liebowitz, M. R. (1987). Social phobia. *Modern Problems in Pharmacopsychiatry*, *22*, 141–173.

Martel, F. L., Hayward, C., Lyons, D. M., Sanborn, K., Varady, S., & Schatzberg, A. F. (1999). Salivary cortisol levels in socially phobic adolescent girls. *Depression and Anxiety*, *10*(1), 25–27.

McEvoy, P. M., Mahoney, A. E. J., & Moulds, M. L. (2010). Are worry, rumination, and post-event processing one and the same? Development of the repetitive thinking questionnaire. *Journal of Anxiety Disorders*, *24*(5), 509–519. doi:10.1016/j.janxdis.2010.03.008

McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine*, *338*(3), 171–179.

Morrison, A. S., & Heimberg, R. G. (2013). Social anxiety and social anxiety disorder. *Annual Review of Clinical Psychology*, *9*(1), 249–274. doi:10.1146/annurev-clinpsy-050212-185631

Nolen-Hoeksema, S. (2000). The role of rumination in depressive disorders and mixed anxiety/depressive symptoms. *Journal of Abnormal Psychology*, *109*(3), 504–511.

Nolen-Hoeksema, S., & Morrow, J. (1991). A prospective study of depression and posttraumatic stress symptoms after a natural disaster: The 1989 Loma Prieta Earthquake. *Journal of Personality and Social Psychology*, *61*(1), 115.

Nolen-Hoeksema, S., Wisco, B., & Lyubomirsky, S. (2008). Rethinking rumination. *Perspectives on Psychological Science*, *3*(5), 400–424.

Ochsner, K., & Gross, J. (2005). The cognitive control of emotion. *Trends in Cognitive Sciences*, *9*(5), 242–249.

Phan, K. L., & Klumpp, H. (2010). Neuroendocrinology and neuroimaging studies of social anxiety disorder. *Social Anxiety: Clinical, Developmental, and Social Perspectives*, *2*, 273–312.

Roelofs, K., van Peer, J., Berretty, E., de Jong, P., Spinhoven, P., & Elzinga, B. M. (2009). Hypothalamus–pituitary–adrenal axis hyperresponsiveness is associated with increased social avoidance behavior in social phobia. *Biological Psychiatry*, *65*(4), 336–343. doi:10.1016/j.biopsych.2008.08.022

Rowland, D. L., Heiman, J. R., Gladue, B. A., Hatch, J. P., Doering, C. H., & Weiler, S. J. (1987). Endocrine, psychological and genital response to sexual arousal in men. *Psychoneuroendocrinology*, *12*(2), 149–158.

Shirotsuki, K., Izawa, S., Sugaya, N., Yamada, K. C., Ogawa, N., Ouchi, Y., … Nomura, S. (2009). Salivary cortisol and DHEA reactivity to psychosocial stress in socially anxious males. *International Journal of Psychophysiology: Official Journal of the International Organization of Psychophysiology*, *72*(2), 198–203. doi:10.1016/j.ijpsycho.2008.12.010

Stewart, J. G., Mazurka, R., Bond, L., Wynne-Edwards, K. E., & Harkness, K. L. (2013). Rumination and impaired cortisol recovery following a social stressor in adolescent depression. *Journal of Abnormal Child Psychology*, *41*(7), 1015–1026. doi:10.1007/s10802-013-9740-1

Treynor, W., Gonzalez, R., & Nolen-Hoeksema, S. (2003). Rumination reconsidered: A psychometric analysis. *Cognitive Therapy and Research*, *27*(3), 247–259.

Urry, H. L. (2010). Seeing, thinking, and feeling: Emotion-regulating effects of gaze-directed cognitive reappraisal. *Emotion*, *10*(1), 125–135. doi:10.1037/a0017434

van West, D., Claes, S., Sulon, J., & Deboutte, D. (2008). Hypothalamic-pituitary-adrenal reactivity in prepubertal

children with social phobia. *Journal of Affective Disorders*, *111* (2-3), 281–290. doi:10.1016/j.jad.2008.03.006

Watkins, E. (2004). Appraisals and strategies associated with rumination and worry. *Personality and Individual Differences*, *37*(4), 679–694. doi:10.1016/j.paid.2003.10.002

Wechsler, D. (2008). *Wechsler Adult Intelligence Scale* (4th ed.). San Antonio, TX: Pearson Assessment.

Wells, A., & Davies, M. I. (1994). The thought control questionnaire: A measure of individual differences in the control of unwanted thoughts. *Behaviour Research and Therapy*, *32*(8), 871–878. doi:10.1016/0005-7967(94)90168-6

Wong, Q. J. J., & Moulds, M. L. (2011). Impact of anticipatory processing versus distraction on multiple indices of anxiety in socially anxious individuals. *Behaviour Research and Therapy*, *49*(10), 700–706. doi:10.1016/j.brat.2011.07.007

Yoon, K. L., & Joormann, J. (2012). Stress reactivity in social anxiety disorder with and without comorbid depression. *Journal of Abnormal Psychology*, *121*, 250–255.

Young, E. A., & Nolen-Hoeksema, S. (2001). Effect of ruminations on the saliva cortisol response to a social stressor. *Psychoneuroendocrinology*, *26*(3), 319–329. doi:10.1016/S0306-4530(00)00059-7

Zoccola, P. M., & Dickerson, S. S. (2012). Assessing the relationship between rumination and cortisol: A review. *Journal of Psychosomatic Research*, *73*(1), 1–9. doi:10.1016/j.jpsychores.2012.03.007

Zoccola, P. M., Dickerson, S. S., & Yim, I. S. (2011). Trait and state perseverative cognition and the cortisol awakening response. *Psychoneuroendocrinology*, *36*(4), 592–595. doi:10.1016/j.psyneuen.2010.10.004

Zoccola, P. M., Dickerson, S. S., & Zaldivar, F. P. (2008). Rumination and cortisol responses to laboratory stressors. *Psychosomatic Medicine*, *70*(6), 661–667. doi:10.1097/PSY.0b013e31817bbc77

Zorn, J. V., Schür, R. R., Boks, M. P., Kahn, R. S., Joëls, M., & Vinkers, C. H. (2016). Cortisol stress reactivity across psychiatric disorders: A systematic review and meta-analysis. *Psychoneuroendocrinology*, *77*, 25–36. doi:10.1016/j.psyneuen.2016.11.036