Trait rumination and response to negative evaluative lab-induced stress: neuroendocrine, affective, and cognitive outcomes

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Abstract:

Theoretical models of depression posit that, under stress, elevated trait rumination predicts more pronounced or prolonged negative affective and neuroendocrine responses, and that trait rumination hampers removing irrelevant negative information from working memory. We examined several gaps regarding these models in the context of lab-induced stress. Non-depressed undergraduates completed a rumination questionnaire and either a negative-evaluative Trier Social Stress Test (n = 55) or a non-evaluative control condition (n = 69), followed by a modified Sternberg affective working memory task assessing the extent to which irrelevant negative information can be emptied from working memory. We measured shame, negative and positive affect, and salivary cortisol four times. Multilevel growth curve models showed rumination and stress interactively predicted cortisol reactivity; however, opposite predictions, greater rumination was associated with blunted cortisol reactivity to stress. Elevated trait rumination interacted with stress to predict augmented shame reactivity. Rumination and stress did not significantly interact to predict working memory performance, but under control conditions, rumination predicted greater difficulty updating working memory. Results support a vulnerability-stress model of trait rumination with heightened shame reactivity and cortisol dysregulation rather than hyper-reactivity in non-depressed emerging adults, but we cannot provide evidence that working memory processes are critical immediately following acute stress.

Keywords: rumination | lab-induced stress | cortisol | shame | working memory | multilevel growth curve models

Article:

Life stress precipitates depression (e.g. Hammen, Citation2005; Monroe, Citation2008); however, individuals differ in stress vulnerability. One particular vulnerability that has received attention is trait rumination, repetitively focusing on the causes and consequences of negative affect (e.g. Nolen-Hoeksema, Wisco, & Lyubomirsky, Citation2008). Trait rumination, a form of perseverative cognition, is considered a transdiagnostic risk factor for internalising disorders (i.e. depression and anxiety disorders; McLaughlin & Nolen-Hoeksema, Citation2011; Michl,

McLaughlin, Shepherd, & Nolen-Hoeksema, Citation2013), and some theorise that trait rumination, "could provide a psychological mechanism for amplifying a stressor," (Young & Nolen-Hoeksema, Citation2001, p. 320). Trait rumination is elevated in both actively depressed and remitted depressed individuals (D'Avanzato, Joormann, Siemer, & Gotlib, Citation2013), predicts future depression among currently non-clinical youth (Rood, Roelofs, Bögels, Nolen-Hoeksema, & Schouten, Citation2009), and has substantial test-retest stability over periods of several months to a year (e.g. .47 to .80; Just & Alloy, Citation1997; Nolen-Hoeksema, Parker, & Larson, Citation1994), suggesting that it is an important and relatively stable marker of risk. Thus, trait rumination may interact with stressful circumstances to predict internalising-related outcomes; studying its effects on an array of aspects of stress responding may inform mechanistic models. Further, we and others have emphasised that the nature of the stressor examined is critical in depression etiology research (Vrshek-Schallhorn et al., Citation2014, Citation2015); in lab-induced stress research, explicitly negative evaluative inductions may present advantages as a model of riskier naturalistic stressors for depression. The present study examines whether trait rumination interacts with an acute, explicitly negative evaluative stressor to predict aberrant stress responding across multiple units of analysis: cortisol reactivity, shame reactivity, and updating negative information in working memory.

Rumination and cortisol reactivity

The balance of extant evidence from lab-based stress inductions suggests that trait rumination is associated with augmented cortisol reactivity, though findings are more consistent for state than trait rumination (for a review, see Zoccola & Dickerson, Citation2012). Similarly, a meta-analysis supported that perseverative cognition broadly was linked with greater cortisol excretion (Ottaviani et al., Citation2016); however, none of the six experimental cortisol studies' effect sizes pertained to trait rumination (instead, state rumination), and only one of eight naturalistic studies' effect sizes pertained to trait rumination in lab-induced stress (i.e. Gianferante et al., Citation2014). Among papers examining this latter question (only one of which excluded depressed individuals), several reported positive associations between trait stress rumination and cortisol reactivity (Zoccola, Quas, & Yim, Citation2010), including one which found a positive association on a second but not first stressor administration in currently non-depressed adults (Gianferante et al., Citation2014), and a third in which high trait ruminators showed greater cortisol reactivity when asked to ruminate but not when asked to self-distract (Shull et al., Citation2016). Two exceptions to this augmented reactivity pattern found no significant relationship between trait rumination and cortisol reactivity (Hilt, Aldao, & Fischer, Citation2015; Young & Nolen-Hoeksema, Citation2001), and an additional exception reported a significant negative association between trait rumination and cortisol reactivity (Zoccola, Dickerson, & Zaldivar, Citation2008).

To examine the relationship between trait rumination and cortisol reactivity, the present study utilised an explicitly negative evaluative adaptation (Way & Taylor, Citation2010) of the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, Citation1993). Stressor characteristics are critical across multiple areas of stress and depression research. Among interview-assessed naturalistic stressful life events, major but not minor severity stressors heighten depressive episode onset risk (for a review, see Monroe, Citation2008), and interpersonal stressors are more impactful than non-interpersonal stressors (Vrshek-Schallhorn et al., Citation2015), Similarly, a large meta-analysis showed that uncontrollable and social evaluative lab-based stressors produced the largest cortisol responses (Dickerson & Kemeny, Citation2004). Although

observers in most TSST studies respond in a neutral fashion (Kudielka, Hellhammer, & Kirschbaum, Citation2007) which nevertheless may be interpreted as negative or evaluative, because of the relevance of overtly interpersonal stress and rejection for depression (Slavich, O'Donovan, Epel, & Kemeny, Citation2010; Vrshek-Schallhorn et al., Citation2015), the present study employed an explicitly negative evaluative manipulation.

Overall, the balance of evidence favours a hypothesis that trait rumination is associated with greater cortisol reactivity to stress. However, no studies yet examine this under explicitly negative-evaluative stress, and few studies report employing even an implicit or ambiguously negative evaluative audience (Zoccola et al., Citation2008), which may be particularly salient to depression risk.

Trait rumination and shame

Theory suggests trait rumination will correspond to greater and/or prolonged negative affective stress-responses (e.g. Joormann, Citation2005), a qualitative review linked trait rumination with increased negative affect (Kirkegaard Thomsen, Citation2006), and trait rumination interacted with daily hassles to predict negative affect (Moberly & Watkins, Citation2008). Within negative affect, shame – self-conscious negative affect – has received particular attention related to depression risk and trait rumination. Shame correlated higher with depression than did general negative affect in a meta-analysis (Kim, Thibodeau, & Jorgensen, Citation2011), and shame and rumination share a focus on the self that general negative affect does not (Smith & Alloy, Citation2009). Moreover, trait rumination mediated the association between shame and depressive symptoms in parents experiencing marital separation (Orth, Berking, & Burkhardt, Citation2006). Taken together, these findings suggest that rumination will interact with stress to predict reactivity in both negative affect and shame, but that its link with shame might be relatively stronger. No work examines whether trait rumination predicts shame reactivity under negative-evaluative stress, yet shame appears key in characterising those at risk for internalising pathology.

Trait rumination and updating working memory

Cognition/emotion depression theories suggest that stress enhances risk via information processing deficits, particularly problems disengaging from irrelevant negative information; trait rumination is considered crucial to this process (Gotlib & Joormann, Citation2010). Trait rumination is associated with impaired working memory in empirical studies. Under basal conditions, trait rumination was associated with difficulty inhibiting irrelevant emotional information (Joormann, Citation2006). Further, depressed outpatients showed poorer performance than controls updating irrelevant negative information in working memory, and performance correlated negatively with trait rumination (Joormann & Gotlib, Citation2008). Last, declining n-back working memory performance from basal to stressful conditions was correlated with depression symptoms more robustly for those high in brooding trait rumination (Quinn & Joormann, Citation2015). However, no work examines trait rumination and affective working memory relationships as a function of stress, which is vital to evaluating relevant models.

The present study

To address the gaps described above, in the present study, 124 non-depressed emerging adults provided affect measures and salivary cortisol repeatedly in the context of an explicitly negative evaluative or a control variant of the TSST protocol, and completed a computerised test of affective working memory updating. We focused on trait rumination rather than state rumination because of our group's emphasis on relatively stable trait-like individual differences in the pathway to affective disorders and the potential to utilise traits in early detection and prevention efforts. We hypothesised that trait rumination would interact with stress to predict greater cortisol reactivity, greater shame reactivity (with a relatively stronger effect than on negative affect, with no hypothesis regarding effects on positive affect), and deficits in updating negative information in working memory.

Method

Participants

Introductory psychology students at a private university were enrolled after completing mass testing session questionnaires assessing eligibility. Eligible participants were native English-speakers 18+ years old, with normal or corrected-to-normal hearing and vision. We excluded individuals endorsing chronic health conditions, head trauma history, learning disabilities, colour-blindness, or use of nicotine, psychotropic or corticosteroid medications, or hormonal birth control. Because current depression predicted blunted cortisol responses to stress in a meta-analysis (Burke, Davis, Otte, & Mohr, Citation2005), participants were eligible only if we could rule out a current major depressive episode (MDE) using a depression screener at mass testing.

N = 127 individuals provided informed consent; two stress condition participants withdrew consent after receiving the stress protocol instructions, and one control session was interrupted by a fire evacuation. The remaining 124 participants (45 female; 69 controls, 55 stress) provided signed permission to use their data after deception debriefing. Ages ranged from 18 to 23 ($M = 18.70$, $SD = .89$); 33 participants (26.6%) were minority race/ethnicity. Four control participants did not complete the working memory task due to computer difficulties.

Materials

Questionnaires

At mass testing, participants completed the Diagnostic Inventory for Depression symptom portion (DID; Zimmerman, Sheeran, & Young, Citation2004), containing 17 items assessing eight MDE symptoms from DSM-IV, after excluding two items addressing the symptom of suicidality. Due to excluding items regarding the symptom of suicidality, we assigned DID-based diagnoses to participants who endorsed four+ of the eight remaining symptoms (instead of the usual five+ of nine symptoms) at the recommended level of 2+ (0–4 scale), including at least one or both of the essential symptoms depressed mood or anhedonia. We also excluded respondents who answered insufficient items to rule out current depression. Total scores also captured subclinical depression symptom severity in enrolled participants ($\alpha = .67$).

At the testing session, participants completed the Ruminative Response Scale (RRS; Treynor, Gonzalez, & Nolen-Hoeksema, Citation2003), a 22-item questionnaire of depressive trait rumination. Ratings range from 1 (almost never) to 4 (almost always), indicating how often individuals engage in ruminative thinking; item mean scores were used in analyses to facilitate interpretation on the 1–4 response scale ($\alpha = .94$). Missing data were rare (1 item across 124 respondents). We used the full scale following Zoccola et al. (Citation2008) but we also report results for the brooding subscale in a footnote.

Participants completed the Positive and Negative Affect Scales (PANAS; Watson, Clark, & Tellegen, Citation1988) plus the Guilt subscale from the PANAS-Expanded Form, which indicates shame, (PANAS-X; Watson & Clark, Citation1999). Two 10-item scales capture positive affect (PA; e.g. interested, excited, $\alpha = .88$ to .93 across repeated administrations) and negative affect (NA; e.g. nervous, distressed, $\alpha = .75$ to .86). The PANAS-X Guilt subscale includes six items assessing negative self-conscious affect relating to shame (e.g. blameworthy, disgusted with self; $\alpha = .86$ to .90). Items are rated on a five-point Likert scale (1 – very slightly or not at all, 5 – extremely). Two Guilt items overlap with PANAS NA ("guilty," "ashamed") and were scored only in Guilt leaving 8 NA items.

Immediately following the TSST, participants completed three manipulation check questions, including "You completed a challenging experience protocol. To what extent did you perceive that you were evaluated by others during this protocol?" Response options ranged from 0 ("I was not evaluated at all") to 3 ("I was substantially evaluated"). Two similar items also ranging from 0 to 3 assessed the extent to which perceived evaluation was positive or negative if the participant endorsed feeling evaluated; the instructions indicated not to answer extent of positive or negative evaluation if they had denied feeling evaluated at all on the first item.

Salivary cortisol

Participants provided saliva samples by passive drool. Samples were frozen at −20°C within 20 min of testing completion. They were later shipped on dry ice to Trier, Germany, where they were assayed in duplicate using time-resolved fluorescent-detection immunoassay (DELFIA; Dressendörfer, Kirschbaum, Rohde, Stahl, & Strasburger, Citation1992). Intra-assay variation ranged from 4.0% to 6.7%; inter-assay variation ranged from 7.1% to 9.0%. Data were inspected for outliers; one Control's levels were consistently elevated > 3 standard deviations above the mean, leading to exclusion from cortisol analyses only. Data were logarithmically transformed to correct skew, but are displayed untransformed to aid interpretation.

Clinical interviews

To screen for past depression and rule out that a current Major Depressive Episode (MDE) had onset between screening and the laboratory session, participants completed the MDE section of the Structured Clinical Interview for DSM-IV, non-patient edition (SCID-I/NP; First, Spitzer, Gibbon, & Williams, Citation2001). SCIDs were administered by undergraduate RAs who completed extensive administration training, matched internal gold standard ratings, and demonstrated proficiency in practice with the PI (SVS), a licensed doctoral-level clinical psychologist. All cases were presented during group supervision to assign consensus diagnoses. No current MDE cases were diagnosed. RAs blind to initial diagnoses rerated 51 SCID interview audio recordings (kappa for past MDD = 0.74).

Stimuli

Working memory task stimuli came from the Affective Norms for English Words list (ANEW; Bradley & Lang, Citation1999), which provides valence and arousal norms. Valence ranges from 1 (most negative) to 9 (most positive), where 5 is neutral; arousal ranges from 1 (least arousing) to 9 (most arousing). Positive and negative nouns balanced for valence extremeness, arousal level, word length, and Kucera-Francis written frequency were chosen. We required that negative nouns have a valence ≤ 3.5, and that positive nouns have a valence ≥ 6.5. In the final group, 140 negative nouns ranged from valence 1.39–3.48 ($M = 2.61$, $SD = 0.53$) with arousal 3.41–8.17 ($M = 5.55$, $SD = 1.01$), and 140 positive nouns ranged from valence 6.54–8.72 ($M = 7.40$, $SD = 0.57$) with arousal 2.97–8.10, ($M = 5.52$, $SD = 0.96$). Lists did not differ significantly in arousal, frequency, word length, or valence extremeness, $Fs(1,239) \leq 1.047$, $ps \geq .307$.

Procedures

Timeline

Participants completed informed consent, followed by the SCID MDE section, questionnaires, the initial PANAS and cortisol sample. Additional PANAS and cortisol samples occurred immediately following the stress induction (approximately +20 min from the baseline sample), the working memory task and an additional cognitive task not presented here together lasting 25 min (+45 min from baseline), deception debriefing and brief rest totalling 15 min (+60 min from baseline). Testing occurred in the afternoon to prevent morning cortisol levels from obscuring reactivity (Dickerson & Kemeny, Citation2004).

Negative evaluative stress induction

Participants completed an explicitly negative evaluative Trier Social Stress Test (“Stress” condition) or a no-audience, non-evaluative control protocol (“Control” condition), both adapted from Way and Taylor (Citation2010), and both variants of the original TSST (Kirschbaum et al., Citation1993). Condition assignment was pseudo-random: Participants enrolled blind to the pre-scheduled experimental condition. All sessions occurred on weekdays; we made an effort to offer sessions of each condition balanced on two possible starting times (i.e. 1300 or 1530 h). All participants received instructions, followed by five minutes each of: speech preparation time, giving a speech, and mental arithmetic aloud (serially subtracting 13 from 2017, with prompts to start again following errors). Participants drew a speech topic, but there was only one topic per condition. Videotaping was actually sham recording.

The conditions differed substantially. Controls spoke about health tips others could follow, a non-evaluative topic. The experimenter sat out of the participant’s sight line, there was no other audience, and the participant was told that his or her performance would not be evaluated. The experimenter was silent, pretending to prepare paperwork for future testing, except for gentle requests to continue speaking or to re-start the mental arithmetic following errors. By contrast, to promote evaluative threat, Stress condition participants spoke about why their peers should select them for a leadership position, and they had an audience (one male and one female) who gave negative non-verbal feedback during the task per a behavioural script. Non-verbal negative behaviours included furrowed brow, a sigh of fatigue, exchanging a dissatisfied glance with the

other confederate, rubbing the bridge of one's nose, and making a conspicuous "X" on papers. To convey that the confederate's negative reactions were associated with participant performance, confederates were instructed to begin with a mildly pleasant facial expression and neutral to interested body language, and to transition to dissatisfied and bored facial expressions from the behavioural script immediately after the speech began. Confederates introduced themselves as "lab volunteers" trained in non-verbal public speaking analysis; they stated they would complete a task performance evaluation. Participants were also told that public speaking experts would evaluate the videotape for voice frequency analysis and non-verbal behaviours. The experimenter stayed out of the participant's sight line; confederates provided instructions and feedback. At the debriefing after the study, participants were shown the confederates' behavioural script in order to alleviate feelings of being negatively evaluated.

Working memory task

Working memory updating was assessed using a modified Sternberg task adapted from Joormann and Gotlib (Citation2008) administered beginning approximately three minutes on average after the TSST. Each trial included three phases. In a learning phase, two 3-word lists were simultaneously displayed in white for 7800 msec, one in a red rectangle background and the other in a blue background, followed by a blank black screen for 800 msec. Next, a cue (a red or blue frame) presented for 1000 msec indicated which list was relevant for the recognition task. Last, a probe screen displayed a single word within the blue or red frame, which remained until participants indicated whether this word came from the relevant list (1 = yes, 2 = no). The words lists displayed were positive or negative. The probe displayed was similarly either positive or negative, and could be new or come from the lists presented. Thus, there were eight trial types (summarised in Table 1), half each with irrelevant positive and negative lists, with probes in four conditions: a word from the relevant list, an intrusion from the irrelevant list (our focus), a new positive word, or a new negative word. Participants completed 80 trials across two blocks, with a short break. The eight trail types were each presented ten times, balanced for presentation location (top/bottom) and background colour (blue/red). Words were sampled from their lists randomly, and were not re-used within block. Responses and latencies were recorded. Task instructions, a step-by-step demonstration trial, and five neutral practice trials promoted understanding.

Statistical approach

Cortisol and affect hypotheses used multilevel regression models employing SAS 9.3 PROC MIXED with maximum likelihood estimation to conduct growth curve analyses to examine whether changes in the repeated measure were accounted for by different time functions – namely linear and quadratic time trends (e.g. Hedeker & Gibbons, Citation2006). A linear time trend models simple increases or decreases over time, whereas a quadratic time trend time models curvilinear change. Such multilevel models permit examination of nested data, such as moments (level 1) measured repeatedly within individuals (level 2). The time variables utilised orthogonal coefficients to achieve uncorrelated terms for linear and quadratic effects of time, and trait rumination was grand mean centred for analyses. Models utilised unstructured covariance matrices given that no other covariance pattern was theoretically indicated (e.g. Hedeker & Gibbons, Citation2006). Intraclass correlations (ICCs) examined preliminarily indicate the extent to which variance is attributable to moments within people (level 1) versus between people (level 2) and

Table 1. Sternberg working memory performance by stress and trial condition.

Trial condition	Relevant valence	Probe valence	Probe type	Controls		Negative evaluative TSST	
				Reaction time, M(SD)	Accuracy, M(SD)	Reaction time, M(SD)	Accuracy, M(SD)
1	Positive	Positive	Relevant	1025.09 (283.97)	9.14 (1.16)	927.05 (231.27)	9.16 (1.08)
2*	Positive	Negative	Intrusion from original list	1228.21 (343.83)	9.08 (1.24)	1089.87 (282.33)	9.24 (0.9)
3	Positive	Negative	New positive	945.32 (341.77)	9.40 (1.34)	853.91 (256.39)	9.64 (0.68)
4*	Positive	Negative	New negative	935.51 (336.36)	9.74 (0.87)	819.48 (259.19)	9.75 (0.67)
5	Negative	Negative	Relevant	1044.62 (326.11)	9.02 (1.12)	907.98 (265.94)	9.20 (0.99)
6	Negative	Positive	Intrusion from original list	1183.10 (372.78)	9.06 (1.12)	1090.05 (340.58)	9.15 (1.13)
7	Negative	Positive	New positive	930.28 (312.93)	9.65 (1.04)	826.05 (242.40)	9.73 (0.68)
8	Negative	Positive	New negative	912.44 (303.38)	9.37 (1.61)	833.27 (265.04)	9.76 (0.58)

Note: * = Analyses focused on these trial conditions: irrelevant negative intrusions from the original list versus new negative words.

provide justification for examining multilevel models. To test our hypotheses of greater reactivity (in cortisol, shame, and negative affect) under stress as a function of trait rumination level, we examined three-way interactions of Trait Rumination × Stress × Quadratic Time. To test whether effects of rumination on shame reactivity were greater than its effects on negative affect reactivity, we first calculated Area Under the Curve with respect to Increase (AUCI), an indicator of reactivity over time that produces a single metric (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, Citation2003), for shame and negative affect, respectively. We then examined the zero-order correlation of rumination with each AUCI score in the negative evaluative condition, and finally conducted Steiger's z-test of the difference in dependent correlations (Lee & Preacher, Citation2013).

Statistical approach

Cortisol and affect hypotheses used multilevel regression models employing SAS 9.3 PROC MIXED with maximum likelihood estimation to conduct growth curve analyses to examine whether changes in the repeated measure were accounted for by different time functions – namely linear and quadratic time trends (e.g. Hedeker & Gibbons, Citation2006). A linear time trend models simple increases or decreases over time, whereas a quadratic time trend time models curvilinear change. Such multilevel models permit examination of nested data, such as moments (level 1) measured repeatedly within individuals (level 2). The time variables utilised orthogonal coefficients to achieve uncorrelated terms for linear and quadratic effects of time, and trait rumination was grand mean centred for analyses. Models utilised unstructured covariance matrices given that no other covariance pattern was theoretically indicated (e.g. Hedeker & Gibbons, Citation2006). Intraclass correlations (ICCs) examined preliminarily indicate the extent to which variance is attributable to moments within people (level 1) versus between people (level 2) and provide justification for examining multilevel models. To test our hypotheses of greater reactivity (in cortisol, shame, and negative affect) under stress as a function of trait rumination level, we examined three-way interactions of Trait Rumination × Stress × Quadratic Time. To test whether effects of rumination on shame reactivity were greater than its effects on negative affect reactivity, we first calculated Area Under the Curve with respect to Increase (AUCI), an indicator of reactivity over time that produces a single metric (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, Citation2003), for shame and negative affect, respectively. We then examined the zero-order correlation of rumination with each AUCI score in the negative evaluative condition, and finally conducted Steiger's z-test of the difference in dependent correlations (Lee & Preacher, Citation2013).

Hypotheses regarding working memory performance were examined using the General Linear Model framework in SPSS 21.0 to conduct a custom RM ANOVA permitting interaction with a dimensional covariate (trait rumination). Our hypotheses focused on the difference between two trial types, negative intrusions versus new negative trials. Trial type (irrelevant negative vs. new negative word) was a within-subject variable; experimental condition, standardised trait rumination, and their interaction effect were entered as between-subjects variables. Within the two trial types examined, we expected that: stress would heighten the difficulty of responding to the task broadly (Stress main effect), stress would magnify the cost of dismissing the more difficult negative intrusion trials (a Stress × Trial Type interaction), and that trait rumination would heighten difficulty removing negative information (a Trait Rumination × Stress × Trial Type interaction).

Due to skew, reaction times were natural log transformed for analyses, but are depicted untransformed in graphs for interpretability.

Trait rumination was analyzed dimensionally throughout all analyses, but is displayed categorically in graphs to aid visualisation. All analyses employed two-tailed statistical tests. The targeted minimum sample size (N = 120) is somewhat larger than typical for studies of the TSST (meta-analytic mean N = 29.58; Dickerson & Kemeny, Citation2004); however, this level was selected to achieve greater power for genetic analyses not reported here, which we assumed would have the lowest power (Avery & Vrshek-Schallhorn, Citation2016), and to achieve similar size as other stress-induction studies involving genetics published when this study was designed (e.g. N = 118, Way & Taylor, Citation2010). The final N reflects all that could be collected in the time available to the team.

Results

Randomisation and manipulation checks

Comparisons support that pseudo-randomisation was effective in balancing the groups. Stress and Control groups did not differ significantly in study start time (1300 or 1530 h),Footnote1 gender, minority status, or MDE history (all $\chi 2(1) < 2.277$, ps ≥ 0.131; Table 2), nor in body mass index, DID score, trait rumination, baseline cortisol, negative affect, positive affect, or Shame (all Fs(1,119–123) ≤ 2.662, all ps $\geq .105$; see Table 2 for descriptive statistics). Three items completed following the induction support its effectiveness. Stress participants reported feeling more evaluated than Controls, $F(1,122) = 58.331$, $p < .001$, and indicated that evaluation was more negative, $F(1,122) = 92.567$, $p < .001$, and less positive, $F(1,122) = 34.136$, $p < .001$, than Controls (Table 2 presents descriptive statistics). Correlations among reactivity indices appear in supplemental materials, Table S1; in brief, there were few significant correlations across levels of analysis (neuroendocrine, affective, cognitive).

Preliminary analyses

Preliminary analyses indicated that subclinical depression symptoms were significantly correlated with rumination in the full sample ($r = .336$, $p < .001$). To examine relationships to cortisol and affect variables, a summary indicator of reactivity, area under the curve with respect to increase (AUCI; Pruessner et al., Citation2003) was calculated, and Control and Stress conditions were examined separately. Subclinical depression symptoms approached significance correlating with Controls' shame ($r = .215$, $p < .083$), negative affect ($r = .217$, $p < .081$), and positive affect ($r = .205$, $p < .098$) reactivity, but not with those of the Stress condition (rs = −.188 to −.046, ps $\geq .188$). Subclinical depression symptoms were not significantly related to cortisol AUCI in either group. To conservatively estimate trait rumination's influence – and not variance shared with subclinical depression symptoms – we covaried subclinical depression symptoms (DID total scores) in line with similar work (Hilt et al., Citation2015) in all growth curve analyses, but we present models without these covariates in supplemental materials (Table S2). All additional interaction terms necessary to partial it out of the hypothesised effect (Trait Rumination × Stress × Quadratic Time) in growth curve analyses were used.

Table 2. Group equivalence and manipulation checks: descriptive values.

	Challenge, n = 55	**Control, n = 69**	**p-value**
Gender	M = 34, F = 21	M = 45, F = 24	NS
Minority status	Yes = 17, No = 38	Yes = 16, No = 53	NS
Study start time (A: 1300 or B: 1530 h)	A = 22, B = 33	A = 37, B = 32	NS
Past MDD	Yes = 2, No = 53	Yes = 7, No = 62	NS
	M (SD)	M (SD)	
Age	18.89 (1.048)	18.55 (0.718)	NS
Body Mass Index	22.493 (3.566)	22.628 (3.495)	NS
Subclinical Depression Symptoms (DID Total Score)	5.455 (3.558)	6.42 (3.628)	NS
Trait Rumination (RRS) Item-Mean	1.782 (0.538)	1.945 (0.565)	NS
State-Dependent Baseline Measure Equivalence			
Cortisol (nmol/L)	4.353 (2.566)	4.48 (2.514)	NS
PA Item-mean	2.472 (0.639)	2.544 (0.691)	NS
NA Item-mean	1.283 (0.304)	1.382 (0.462)	NS
Shame Item-mean	1.142 (0.296)	1.208 (0.473)	NS
Manipulation Checks			
Post-TSST Perceived Evaluation	2.35 (0.751)	1.26 (0.803)	<.001
Perceived Positive Evaluation	0.49 (0.605)	1.25 (0.767)	<.001
Perceived Negative Evaluation	2.09 (0.727)	0.82 (0.695)	<.001

Note: P-values refer to tests examining group differences. See text for type and range of test statistics. NS = not significant, $p > .05$.

Cortisol reactivity

The final model was a random slope model that permitted random effects for both the intercept and slope; the ICC was .70 indicating that approximately 70% of variance was attributable to moments within people (level 1), rather than between person variance (level 2). The simple main effects and lower order interactions characterise a pattern where the Control group's cortisol declined in a linear fashion over time, while the Stress Condition's cortisol shows an overall upward trend, qualified by a significant curvilinear component (Table 3). A three-way Trait Rumination × Stress × the Quadratic Time interaction, $\beta = 0.158$, $SE(\beta) = 0.072$, $t(231) = 2.19$, $p = 0.029$, was in the opposite direction predicted: Higher trait rumination was associated with blunted reactivity in Stress versus Controls (Figure 1). The pattern of results was the same when omitting subclinical depression symptoms as a covariate (Table S2). A posthoc model within the Stress condition participants only showed that the Trait Rumination × the Quadratic Time interaction approached significance; trait rumination was associated with a flatter cortisol reactivity curve, $\beta = 0.109$, $SE(\beta) = 0.063$, $t(104) = 1.72$, $p = 0.088$. There was no significant parallel interaction among Controls (with an effect size in the opposite direction), $\beta = -0.048$, $SE(\beta) = 0.040$, $t(128) = -1.20$, $p = 0.233$.Footnote2,Footnote3

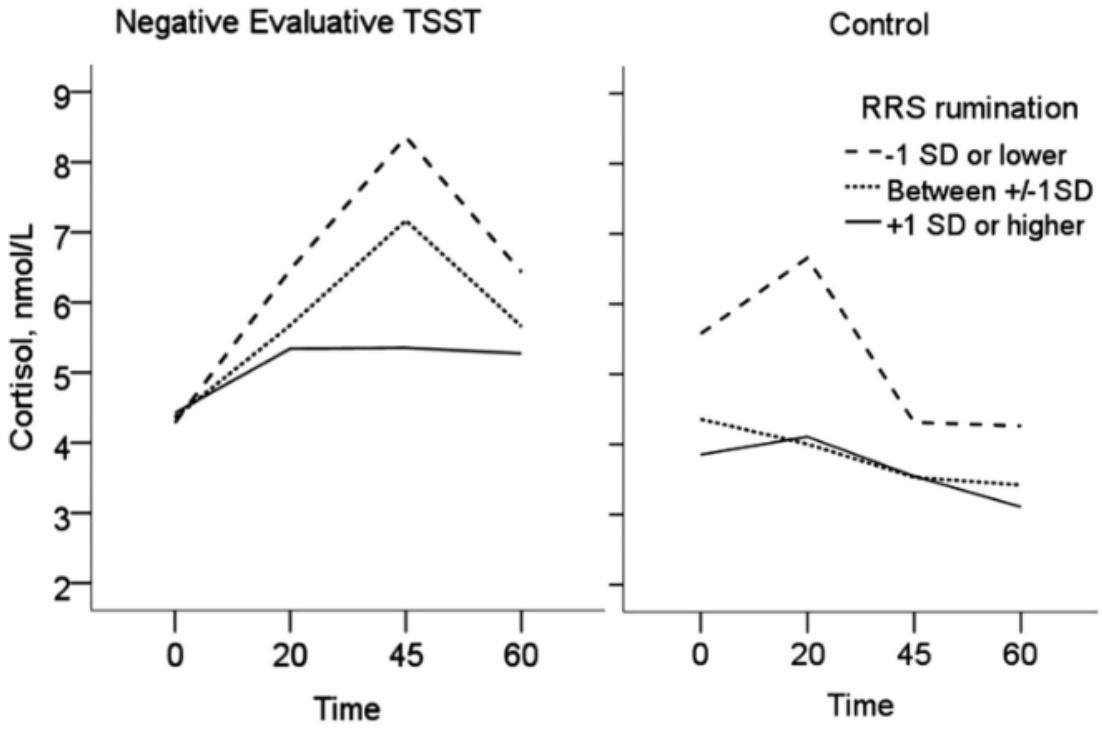

Figure 1. Cortisol levels by stress, trait rumination, and time.
Note: Trait rumination was analysed dimensionally in growth curve models, but is depicted categorically to aid visualisation. Groups represent all participants at or below −1SD from the mean of trait rumination, between −1 and +1 SD of the mean, and those at +1 SD from the mean and above.

Table 3. Multilevel regression model results for salivary cortisol and shame.

	Cortisol					Shame				
Variable	**Beta**	**SE(B)**	**DF**	**t**	**p-value**	**Beta**	**SE(B)**	**DF**	**t**	**p-value**
Time	−0.199	0.046	117	−4.340	<.0001	−0.150	0.040	351	−3.740	0.000
Quadratic Time	−0.010	0.025	231	−0.410	0.684	−0.105	0.040	351	−2.650	0.009
Stress	0.306	0.085	231	3.590	0.000	0.039	0.058	351	0.660	0.510
Rumination	−0.095	0.107	231	−0.880	0.377	0.165	0.074	351	2.220	0.027
Dep Symptoms	−0.021	0.016	231	−1.270	0.206	0.018	0.011	351	1.580	0.114
Dep Symptoms × Stress	−0.002	0.025	231	−0.070	0.944	−0.027	0.017	351	−1.520	0.128
Dep Symptoms × Time	0.000	0.013	231	0.010	0.989	−0.001	0.012	351	−0.100	0.920
Dep Symptoms × Quadratic Time	−0.001	0.007	231	−0.100	0.920	−0.023	0.012	351	−1.960	0.051
Stress × Dep Symptoms × Time	0.019	0.020	231	0.960	0.340	0.008	0.018	351	0.420	0.674
Stress × Dep Symptoms × Quadratic Time	0.009	0.011	231	0.840	0.403	0.049	0.018	351	2.750	0.006
Stress × Time	0.426	0.068	231	6.250	<.0001	−0.019	0.060	351	−0.320	0.747
Stress × Quadratic Time	−0.206	0.037	231	−5.540	<.0001	−0.112	0.060	351	−1.880	0.060
Rumination × Time	0.105	0.086	231	1.220	0.222	−0.113	0.076	351	−1.490	0.138
Rumination × Quadratic Time	−0.048	0.047	231	−1.020	0.307	0.043	0.076	351	0.570	0.570
Stress × Rumination	0.057	0.165	231	0.350	0.730	0.023	0.114	351	0.200	0.839
Stress × Rumination × Time	−0.120	0.132	231	−0.910	0.364	0.005	0.116	351	0.040	0.968
Stress × Rumination × Quadratic Time	0.158	0.072	231	2.190	0.029	−0.309	0.116	351	−2.670	0.008

Note: Dep symptoms = subclinical depression severity (DID total scores).

Affective reactivity

The final models were random intercept models that permitted random effects for the intercept only (random slope models for affect failed to converge); ICCs were .39, .28, and .62 respectively for shame, negative affect, and positive affect. In the primary model with subclinical depression symptoms covaried, the simple main effects and lower order interactions characterised shame reactivity in a curvilinear pattern that approached significance in terms of being more pronounced in Stress versus Controls ($p = .060$) at the mean of trait rumination, (Table 3). As hypothesised, a significant three-way Trait Rumination × Stress × Quadratic Time interaction, $\beta = -0.309$, $SE(\beta) = 0.116$, $t(351) = -2.67$, $p = 0.008$, indicated greater shame reactivity with higher trait rumination for Stress versus Controls (Figure 2). In Stress, a significant Rumination × Quadratic Time interaction, $\beta = -0.266$, $SE(\beta) = 0.105$, $t(157) = -2.54$, $p = 0.012$, indicated that trait rumination predicted greater shame reactivity. Further post-hoc probing of Stress condition rumination-shame correlations indicated no significant correlations at the baseline or final measures ($rs = .137–.165$, $ps = .229–.324$) but a significant correlation at the second sample that occurred immediately following the TSST ($r = .341$, $p = .011$) and one approaching significance at the third administration following the cognitive task ($r = .240$, $p = .078$). As expected, the Rumination × Quadratic Time effect was non-significant in Controls, $\beta = 0.043$, $SE(\beta) = 0.061$, $t(194) = 0.70$, $p = 0.483$. In a model without subclinical depression symptoms, the hypothesised effect approached significance, $\beta = -0.1941$, $SE(\beta) = 0.1093$, $t(356) = -1.78$, $p = 0.0765$ (Table S2). We attribute this to a significant Subclinical Symptoms × Stress × Quadratic Time effect in the opposite direction as that observed for Rumination (Table 3), such that symptoms were positively associated with shame reactivity in Controls (supplemental Figure S1).

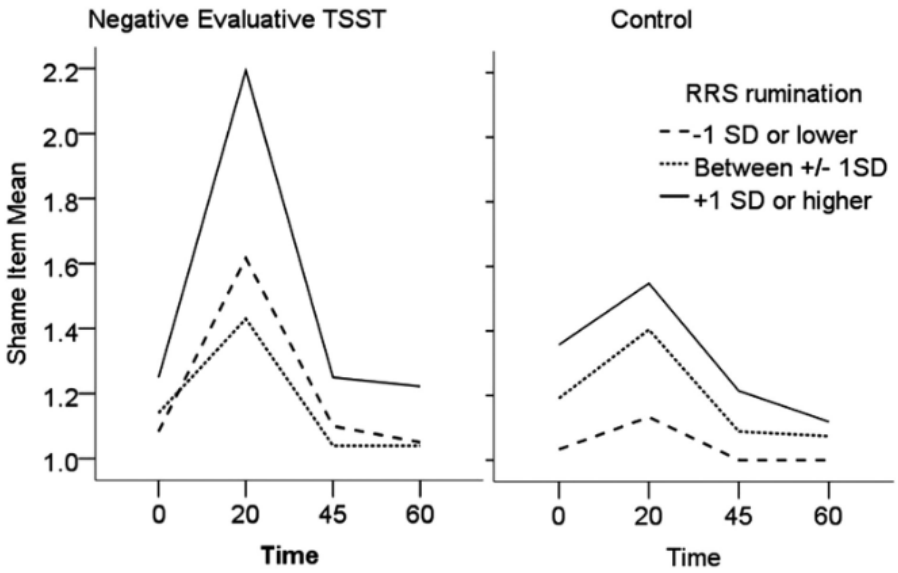

Figure 2. Shame levels by stress, trait rumination, and time.
Note: Trait rumination was analysed dimensionally in growth curve models, but is depicted categorically to aid visualisation. Groups represent all participants at or below −1SD from the mean of trait rumination, between −1 and +1 SD of the mean, and those at +1 SD from the mean and above.

In the full sample, the Trait Rumination × Stress × Quadratic Time interaction approached significance for NA, $\beta = -0.221$, $SE(\beta) = 0.129$, $t(351) = -1.70$, $p = 0.090$, and was non-significant for Positive Affect, $\beta = 0.056$, $SE(\beta) = 0.163$, $t(351) = 0.34$, $p = 0.731$ (supplemental Table S3).

A test of the difference between dependent correlations using Steiger's z-test focused on the stress condition and relied on area under the curve with respect to increase (AUCI) as the index of affect reactivity. The correlation between shame reactivity AUCI and trait rumination approached significance ($r = .256$, $p = .062$), while the correlation between general negative affect reactivity AUCI and trait rumination was not significant ($r = .216$, $p = .116$); however, Steiger's z-test indicated that the two effects did not significantly differ ($z = 0.314$, $p = .753$).

Working memory performance

We examined whether trait rumination and stress interact to heighten how irrelevant negative stimuli remain as a residue in working memory, and are thus more difficult to dismiss than new negative words, using a three-way interaction, Trait Rumination × Stress × Trial Type.

Accuracy (Table 1). Unsurprisingly, a Trial Type main effect, $F(1,116) = 23.220$, $p < .001$, $\eta 2p = .167$ indicated New Negative Trials had greater accuracy than Irrelevant Negative Trials. Consistent with prior findings, there were no other significant accuracy effects (Joormann & Gotlib, Citation2008). Condition and trait rumination main effects were non-significant, all $Fs(1,116) \leq 2.139$, $ps \geq .146$, $\eta 2ps \leq .018$, as were two-way interactions, all $Fs(1,116) \leq 1.329$, $ps \geq 251$, $\eta 2ps \leq .011$. The 3-way Trait Rumination × Stress × Trial Type interaction approached significance, $F(1,116) = 3.114$, $p = .080$, $\eta 2p = .026$.

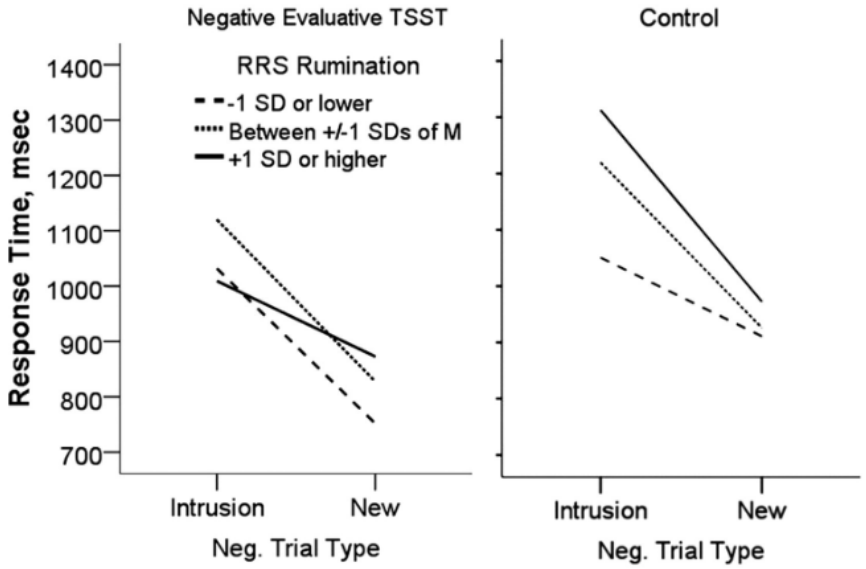

Figure 3. Sternberg affective working memory updating performance for negative intrusions and new words by stress and trait rumination.
Note: Trait rumination was analysed dimensionally in growth curve models, but is depicted categorically to aid visualisation. Groups represent all participants at or below −1SD from the mean of trait rumination, between −1 and +1 SD of the mean, and those at +1 SD from the mean and above.

Response times (Table 1). Contrary to expectations, a main effect of condition, $F(1,116) = 4.670$, $p = .033$, $\eta 2p = .039$, indicated that Stress facilitated faster response times. Consistent with the expectation that negative intrusions would be more challenging than new negatives, a Trial Type main effect indicated response times were slower for irrelevant negative words versus new negatives, $F(1,116) = 222.410$, $p < .001$, $\eta 2p = .657$ (Figure 3). There were not significant main effects of trait rumination, $F(1,116) = 0.986$, $p = .327$, $\eta 2p = .008$, nor two-way interactions of Condition × Trial Type, $F(1,116) = 0.008$, $p = .929$, $\eta 2p = .000$, or Trait Rumination × Trial Type, $F(1,116) = 0.503$, $p = .480$, $\eta 2p = .004$. There was a significant Stress × Trait Rumination interaction, $F(1,116) = 4.040$, $p = .047$, $\eta 2p = .034$. Parsing this within condition, in Controls, higher trait rumination predicted slower response times overall, $F(1,63) = 4.949$, $p = .030$, $\eta 2p = .073$, while no significant effect emerged under Stress, $F(1,53) = 0.488$, $p = .488$, $\eta 2p = .009$. Finally, the a priori hypothesised three-way interaction of Trait Rumination × Stress × Trial Type was non-significant, $F(1,116) = 0.214$, $p = .654$, $\eta 2p = .002$.

Discussion

We showed for the first time that trait rumination predicts blunted cortisol reactivity and heightened shame reactivity to lab-induced explicit negative evaluative stress relative to non-stressful conditions in non-depressed emerging adults. Further, in a first test of whether negative-evaluative lab-induced stress would induce difficulty removing irrelevant negative information from working memory as a function of trait rumination, we instead found a facilitating main effect of stress on updating working memory. However, under control conditions, we also identified that trait rumination level predicts overall greater impairment in accessing working memory.

Implications of cortisol and shame findings

That trait rumination predicted blunted rather than heightened cortisol reactivity under stressful versus control conditions is not without precedence (i.e. similar findings emerged under "evaluative" stress; Zoccola et al., Citation2008), but demands explanation. In other work, one of us has recently empirically shown that the direction of the rumination-cortisol reactivity relationship depends upon the lab-based stressor severity, with a moderate stressor (similar to a standard TSST) yielding a positive association, and an intense stressor (the current negative evaluative TSST) yielding a negative association (Vrshek-Schallhorn, Avery, Ditcheva, & Sapuram, Citationin press). Further, trait rumination predicted greater shame reactivity when also accounting for subclinical depression symptoms. Prior work has suggested that shame is more relevant to depression than is general negative affect (Kim et al., Citation2011), and we predicted that rumination would be more closely related with shame reactivity than with negative affect reactivity given the self-focus shared by trait rumination and shame (Smith & Alloy, Citation2009). The present results, however, indicate that effect sizes of rumination on shame and negative affect did not significantly differ.

Taken together, blunted cortisol reactivity can be interpreted here as a maladaptive response in which affectively stressed individuals are not mounting a sufficient HPA response to rise to a challenge. Indeed, this mimics actively depressed individuals' responses to modest stress (Burke et al., Citation2005), suggesting both acute depression and dimensional risk (i.e. in non-depressed individuals, as captured by trait rumination) are capable of association with a "collapsed" HPA response. This view builds on conceptualizations of cortisol as a "boosting" agent (Adam,

Hawkley, Kudielka, & Cacioppo, Citation2006) that facilitates energy expenditure in the face of challenges (e.g. Sapolsky, Romero, & Munck, Citation2000). We speculate that these effects are cognitively mediated by threat and negative evaluation appraisals (which we did not measure in this study), consistent with prior work showing that anticipatory stress appraisals positively predicted reactivity to a standard TSST (Juster, Perna, Marin, Sindi, & Lupien, Citation2012). These interpretations suggest that, in future work, trait rumination will be associated with insufficient behavioural activation in the face of threats, and that this inactivation will contribute to depression symptoms following naturalistic stress.

Working memory

Working memory performance did not conform to hypotheses. First, although we predicted stress would confer performance decrements, we instead observed facilitation effects. This was consistent with other reports of stress's facilitative effects on various cognitive processes (Beckner, Tucker, Delville, & Mohr, Citation2006; Buchanan & Tranel, Citation2008; Smeets, Giesbrecht, Jelicic, & Merckelbach, Citation2007; Smeets, Otgaar, Candel, & Wolf, Citation2008). In a Yerkes-Dodson framework (for a review, see Teigen, Citation1994), this suggests that Controls' physiological arousal was suboptimal for this task, and Stress boosted arousal to more task-optimal levels. Second, although we predicted that trait rumination would differentially confer decrements in updating irrelevant negative information in working memory under stressful versus basal conditions, no such Trait Rumination × Stress × Trial Type interaction effect emerged. A prior report examining affective Sternberg working memory performance of depressed outpatients under basal conditions showed effects of trait rumination on heightened negative interference with working memory (Joormann & Gotlib, Citation2008). Our results may conceptually contrast these findings because we employed a de novo stimulus set rather than one identical to that previously used, or perhaps more likely because we tested a non-depressed sample for whom the negative stimuli were less self-relevant as compared to the original report's depressed outpatients.

Instead of our predicted interaction, we observed an unpredicted interaction effect between rumination and stress, such that rumination predicted poorer working memory access across both trial types analysed but only under control conditions. This finding can be understood in light of evidence that, in both healthy controls and depressed individuals, trait rumination is associated with increased activity in a network of brain regions engaged off-task, the default mode network (DMN; Berman et al., Citation2011). Trait rumination appears to capture difficulty inhibiting self-focused mind wandering indicated by DMN activity, leading to working memory impairment. Indeed, among combined healthy controls and remitted depressed individuals, greater trait rumination levels predicted poorer DMN suppression during a working memory task performed under basal conditions (Bartova et al., Citation2015). To the extent that non-stressful conditions are more common than acutely stressful situations, our findings suggest that rumination may be linked with working memory dysregulation (although not specifically for updating working memory and not necessarily only for affective working memory) much of the time.

Limitations

Despite strengths including a controlled stressor, biomarker measurement, diagnostic interviews, stringent inclusion/exclusion criteria, and cutting-edge growth curve models, the study has several limitations. Selecting non-depressed individuals may have impaired identifying trait rumination-

working memory associations; we studied only non-depressed individuals because we did not want depressed individuals to undergo an explicitly negative evaluative stressor. Second, having participants complete a cognitive task inherently prevented them from brooding after the stress induction, which may have facilitated mood and cortisol recovery; as such, we focused hypotheses and analyses on reactivity rather than recovery. Third, it may be that working memory performance is related to state rumination (which we did not measure given our primary interest in trait markers of depression-risk) even though we could not link it significantly with trait rumination in the present study. Finally, we are unable to provide a test of differential effects of explicitly versus implicitly negative evaluative methods with the present dataset; thus our suggestion that explicit negative evaluation may be important for interpretation must be considered a preliminary one.

Conclusions

Taken together, evidence suggests that heightened trait rumination contributes to maladaptive and dysregulated responses to social stress – namely, collapsed neuroendocrine responding despite heightened shame. We speculate this pattern contributes to naturalistic deficits in behavioural activation following stress. Although trait rumination has been linked to affective working memory deficits particularly among depressed individuals, our results do not provide support for the notion that updating working memory is a key mechanism by which trait rumination contributes to maladaptive stress responding immediately following objective social stress.

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Disclosure statement

No potential conflict of interest was reported by the authors.

Additional information

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Notes

1 To ensure that the two start times did not influence results, we conducted an additional cortisol model with start time (early, 1300 h = 0, late, 1530 h = 1) as a covariate, including all interactions necessary to partial its influence out of the primary effect of interest, Rumination × Stress × Time2. The primary effect remained significant, $t(233) = 2.63$, $p = 0.0090$, the simple main effect of start time (representing the influence of start time on intercept/baseline cortisol for the control group, when effects of stress condition are partialed out) was significant and was consistent with expected lower values for later start times, $b = -0.2281$, $SE(b) = 0.1123$, $t(233) = -2.03$, $p = 0.0434$, and interactions involving start time (e.g., indicating stress reactivity) were not significant, $ps \geq 0.4713$.

2 Results for the brooding subscale of rumination were similar but not identical to those for the full scale. Without covariates, brooding's relationship to reactivity under stress (i.e. Brooding Rumination × Stress × Time2) was significant for cortisol, $p = .017$, but not for shame, $p = .2516$, NA, $p = .2923$, or PA, $p = .7650$. When subclinical depression symptoms and its higher order interactions were covaried, this interaction approached significance for cortisol, $p = .0593$, and for shame, $p = .0505$, but remained non-significant for NA, $p = .1516$, and PA, $p = .7438$.

3 When covarying gender and necessary higher order interactions, the pattern of results for cortisol reactivity (Rumination × Stress × Quadratic time) remained the same, $p = 0.0158$, as did shame, $p = 0.1650$ (but $p = 0.0253$ with depression symptoms covaried). Similarly, the pattern of results remained the same when covarying past MDD and all necessary higher order interactions: Rumination predicted cortisol reactivity, $p = 0.0017$ ($p = 0.0082$ with DID covaried), but not shame reactivity, $p = 0.1214$ ($p = 0.0157$ with subclinical depression symptoms also covaried).

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