Effects of trauma-focused rumination among trauma-exposed individuals with and without posttraumatic stress disorder: An experiment

By: Blair E. Wisco, Suzanne Vrshek-Schallhorn, Casey L. May, Allison A. Campbell, Faith O. Nomamiukor, Cameron P. Pugach

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Abstract:

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Effects of trauma-focused rumination among trauma-exposed individuals with and without posttraumatic stress disorder: An experiment

Blair E. Wisco | **Suzanne Vrshek-Schallhorn** | **Casey L. May** | **Allison A. Campbell** | **Faith O. Nomamiukor** | **Cameron P. Pugach**

Department of Psychology, University of North Carolina at Greensboro, Greensboro, North Carolina, USA

Correspondence
Blair E. Wisco, Department of Psychology, University of North Carolina at Greensboro, P.O. Box 26170, Greensboro, NC, 27402, United States. Email: bewisco@uncg.edu

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Abstract

Rumination, or thinking repetitively about one's distress, is a risk factor for posttraumatic stress disorder (PTSD). Current theories suggest that rumination contributes to PTSD symptoms directly, by increasing negative reactions to trauma cues (i.e., symptom exacerbation), or represents a form of cognitive avoidance, if verbal ruminations are less distressing than trauma imagery. The goal of this study was to test the symptom exacerbation and cognitive avoidance accounts of trauma-focused rumination. We recruited 135 trauma-exposed participants ($n = 60$ diagnosed with PTSD) and randomly assigned them to ruminate about their trauma, distract themselves, or engage in trauma imagery. For individuals with and without PTSD, rumination led to larger increases in subjective distress (i.e., negative affect, fear, sadness, subjective arousal, valence) than distraction, η_p^2s = .04–.13, but there were no differences between rumination and imagery η_p^2s = .001–.02. We found no evidence that rumination or imagery elicited physiological arousal, ds = 0.01–0.19, but did find that distraction reduced general physiological arousal, as measured by heart rate, relative to baseline, $d = 0.84$, which may be due to increases in parasympathetic nervous system activity (i.e., respiratory sinus arrhythmia), $d = 0.33$. These findings offer no support for the avoidant function of rumination in PTSD. Instead, the findings were consistent with symptom exacerbation, indicating that rumination leads directly to emotional reactivity to trauma reminders and may be a fruitful target in PTSD intervention.

Rumination, or thinking passively and repetitively about one's distress (Nolen-Hoeksema et al., 2008), has emerged as an important cognitive risk factor for posttraumatic stress disorder (PTSD; Ehlers & Clark, 2000; Moulds et al., 2020; Szabo et al., 2017). The content of ruminative thought can be similar to exaggerated negative beliefs characteristic of PTSD (e.g., PTSD Criterion D2 in the *Diagnostic and Statistical Manual of Mental Disorders* [5th ed.; *DSM-5*]; American Psychiatric Association, 2013), but rumination is distinguished by its perseverative process that keeps

attention focused on negative thoughts. Although much of the rumination literature has focused on depressed mood (Nolen-Hoeksema et al., 2008), the target of rumination can be other emotions (e.g., anger) or life events (e.g., traumatic events). Rumination and PTSD symptoms have been shown to be correlated in cross-sectional samples (see Szabo et al., 2017, for a review), and rumination prospectively predicts the development of PTSD symptoms (e.g., Michael et al., 2007). Surprisingly, meta-analytic findings have demonstrated stronger associations between rumination and PTSD for non–trauma-focused forms of rumination (e.g., depressive rumination) than for trauma-focused rumination (Szabo et al., 2017), although both types of rumination are consistently associated with PTSD. Cross-sectional research indicates that depressive rumination mediates the association between difficulties in emotion regulation and PTSD symptoms (Pugach et al., 2020), and results from treatment outcome studies indicate that PTSD treatment reduces the tendency to ruminate about trauma (Schumm et al., 2022; Wisco et al., 2013).

Although rumination and PTSD appear to be related, the mechanisms through which rumination increases the risk for PTSD remain unclear. In their cognitive theory of PTSD, Ehlers and Clark (2000) proposed several possible mechanisms, including the two mechanisms that were the focus of this experiment. First, trauma-focused rumination may serve as a form of avoidance, termed here as the "cognitive avoidance mechanism" (Ehlers & Clark, 2000). Specifically, verbal ruminations about a traumatic experience (e.g., thinking, "Why did this happen to me?") might be less distressing than mental imagery of the event (e.g., visualizing the trauma as it actually occurred). Because avoidance of trauma memories is thought to be a key maintenance factor for PTSD (Foa, 2011), trauma-focused rumination may maintain PTSD by promoting avoidance of aversive mental imagery of the traumatic event. Support for the cognitive avoidance mechanism comes from related literature showing that mental imagery, in general, tends to evoke stronger emotional responses than verbal thought (Holmes & Mathews, 2010) and that worry, another type of verbal repetitive negative thinking, may serve an avoidant function in generalized anxiety disorder (Borkovec et al., 2004). Research indicates that depressive rumination is correlated with self-reported cognitive avoidance (Dickson et al., 2012; Moulds et al., 2007), and with thought suppression, a type of cognitive avoidance, among individuals with comorbid PTSD and depression (Rosebrock et al., 2019). Little research has examined the possible avoidant function of trauma-focused rumination, specifically, but the authors of one cross-sectional study found that experiential avoidance mediated the association between trauma-related rumination and PTSD (Bishop et al., 2018). Further, Schaich et al. (2013) found that concreteness training (i.e., instructions to focus on the details of an event concretely and vividly, rather than analyzing the event) eliminated the association between trait rumination and intrusive memories, offering additional support.

Second, Ehlers and Clark (2000) proposed that, among other possible mechanisms, rumination may increase negative feelings in the moment and even prompt intrusive memories directly by providing internal cues for memory retrieval, termed here as the "symptom exacerbation" mechanism. This mechanism is similar to the response styles theory of depression (Nolen-Hoeksema, 1991), which argues that depressive rumination leads to long-lasting symptoms by directly contributing to depressed mood. The response styles theory is supported by a large literature indicating that depressive rumination increases momentary negative mood among depressed individuals (see Nolen-Hoeksema et al., 2008, for a review). The effects of trauma-focused rumination have received less attention, but some experiments have examined the effects of rumination on PTSD-like symptoms in response to trauma analog procedures (i.e., videos of events, such as road traffic accidents, shown to reliably induce short-term intrusive memories; Ehring et al., 2009; Kubota & Nixon, 2017; Zetsche at al., 2009). In one such experiment, healthy, non–trauma-exposed participants were randomly assigned to ruminate or engage in other types of thinking following a trauma analog film (Zetsche et al., 2009). Individuals who were induced to ruminate showed significantly larger increases in postfilm sadness relative to the two comparison conditions but showed no differences in postfilm fear or intrusive memories. Other studies with similar designs have also demonstrated that rumination leads to more negative mood but no difference in intrusive memories relative to comparison conditions (Ehring et al., 2009; Kubota & Nixon, 2017). These studies also assessed heart rate, with mixed results: One experiment showed significant differences in heart rate between rumination and comparison conditions (Ehring et al., 2009), whereas two experiments showed no differences (Kubota & Nixon, 2017). In the experiment that showed a difference, heart rate did not significantly change during rumination, but it did significantly decrease from baseline during distraction and concrete thinking, indicating relatively more arousal in the rumination condition (Ehring et al., 2009).

To our knowledge, only one experiment using the trauma analog procedure has found postfilm rumination to increase intrusive memories; interestingly, that study was also the only experiment that demonstrated no effect of rumination on negative mood (Ball & Brewin, 2012). Ball and Brewin (2012) specifically recruited a sample of high ruminators, identified by a prescreening measure, and included rumination inductions both immediately

following the film and daily, at the participant's home, for the following week, which may have bolstered the effect of rumination on intrusive memories during that week. Thus, experimental findings offer only limited support for the theory that trauma-focused rumination prompts intrusive memories but do support that trauma-focused rumination leads directly to negative mood.

The cognitive avoidance and symptom exacerbation mechanisms are seemingly contradictory: The former suggests that trauma-focused rumination decreases distress in the moment, whereas the latter suggests that it increases distress in the moment. We propose that these opposite effects may both be true such that trauma-focused rumination leads to more distress and physiological arousal than thinking about something other than the trauma, consistent with symptom exacerbation, but less distress and arousal than engaging in mental imagery of the trauma, consistent with cognitive avoidance.

Although it has been more than two decades since Ehlers and Clark's (2000) paper, surprisingly little experimental research has examined the effects of trauma-focused rumination, specifically, and all have used trauma analog procedures in healthy individuals (Ball & Brewin, 2012; Ehring et al., 2009; Kubota & Nixon, 2017; Zetsche et al., 2009). To date, there have been no experiments testing the emotional or physiological effects of ruminating about a personally experienced trauma among individuals with and without PTSD (Moulds et al., 2020). Moreover, most rumination research has focused on self-reported depressed mood and has not considered other emotions, such as fear, or assessed physiological responses (see Ehring et al., 2009; Kubota & Nixon, 2017, for exceptions).

The purpose of this study was to examine the emotional and physiological effects of trauma-focused rumination, contrasted with trauma imagery and externally focused distraction, among individuals with and without PTSD. We aimed to clarify whether trauma-focused rumination heightens emotional and physiological responses relative to externally focused distraction, consistent with symptom exacerbation, and whether rumination dampens emotional and physiological responses relative to trauma imagery, consistent with cognitive avoidance. We selected heart rate (i.e., interbeat interval [IBI]) as a primary physiological outcome because increased heart rate in response to trauma reminders is robustly associated with PTSD (Pole, 2007). As heart rate cannot distinguish between sympathetic and parasympathetic influences, we included two cardiovascular measures to capture sympathetic (preejection period [PEP]) and parasympathetic (high-frequency heart rate variability) activity more precisely. We also included a second measure of sympathetic arousal, salivary alpha-amylase (sAA), to examine convergence across sympathetic measures. Finally, we included cortisol as a measure of hypothalamic–pituitary–adrenal axis activity. Because previously reported findings regarding the effects of rumination on cortisol have been mixed (Zoccola & Dickerson, 2012), we did not have specific hypotheses but examined cortisol in exploratory analyses. As the first experimental manipulation of rumination about experienced, not analog, trauma, this study will help to clarify the seemingly contradictory literature and elucidate possible functions of trauma-focused rumination in PTSD.

METHOD

Participants

Our sample consisted of 135 individuals recruited from a medium-sized city in the southeastern United States. Participants had to be at least 18 years old and report exposure to at least one traumatic event. Individuals were excluded if they reported trauma exposure within the past month, were pregnant, had a history of cardiovascular disease, were using medications known to affect cardiovascular functioning, reported dissociative symptoms, or endorsed current active suicidal or homicidal ideation. Participants were screened for trauma exposure using the Life Events Checklist for *DSM-5* (LEC-5; Weathers, Blake, et al., 2013b). This was followed by a phone screen to ensure that the index event met *DSM-5* PTSD Criterion A, which was verified during the experiment using the Clinician-Administered PTSD Scale for *DSM-5* (CAPS-5; Weathers, Blake, et al, 2013a). See Table 1 for sample characteristics.

Procedure

Recruitment and ethical approval

All procedures were approved by the University of North Carolina at Greensboro's Institutional Review Board. Potential participants were recruited from the community through flyers, email listservs, and local newspaper advertisements as well as from an outpatient psychology clinic. Interested individuals completed online prescreening measures, and eligible individuals were recruited into the study. Participants with probable PTSD were oversampled and stratified by trauma type to enable us to recruit approximately equal groups of individuals with and without PTSD from each trauma type category (see Table 1).

TABLE 1 Participant characteristics

Variable	Full sample ($N = 135$)		PTSD ($n = 60$)		Control ($n = 75$)	
	n	%	*n*	%	*n*	%
Gender						
Female	107	79.3	49	81.7	58	77.3
Male	28	20.7	11	18.3	17	22.7
Educational attainment[a]						
High school degree	29	21.5	13	21.7	16	21.3
Some college	77	57.0	40	66.7	37	49.3
Bachelor's degree or higher	29	21.5	7	11.7	22	29.3
Race/ethnicity						
American Indian	3	2.2	2	3.4	1	1.4
Asian	2	1.5	1	1.7	1	1.4
Black	56	42.1	21	35.6	35	47.3
Hispanic/Latino	8	6.0	3	5.1	5	6.8
White	51	38.3	27	45.8	24	32.4
Biracial	12	9.0	5	8.5	7	9.5
Other/not reported	3	2.2	1	1.7	2	2.6
Worst trauma type						
Natural disaster or accident	23	17.2	8	13.3	15	20.0
Physical assault	23	17.0	8	13.3	15	20.0
Sexual assault	63	46.7	32	53.3	31	41.3
Illness or death	24	17.8	11	18.3	13	17.3
Other	2	1.5	1	1.7	1	1.3
	M	***SD***	***M***	***SD***	***M***	***SD***
Age (years)	24.96	8.79	24.22	7.81	25.35	9.49
CAPS-5 score[b]	20.82	12.25	32.12	7.04	11.79	6.78
Time since trauma (years)	4.68	5.80	3.89	4.21	5.30	6.76

Note: The PTSD and control groups did not differ significantly on any variables except where noted. PTSD = posttraumatic stress disorder, CAPS-5 = Clinician-Administered PTSD Scale for *DSM-5*.

[a]Significant difference between the PTSD and control groups: $\chi^2(2, N = 135) = 6.61$, $p = .037$

[b]Significant difference between the PTSD and control groups: $t(133) = 17.02$, $p < .001$, $d = 2.95$.

Experimental design

In the first experimental session, informed consent was obtained, the prescreening health questionnaire was readministered to verify the inclusion and exclusion criteria, and the CAPS-5 was administered. Participants were then asked to complete trauma script forms describing their "worst" (i.e., index) traumatic event as indicated on the CAPS-5. The experimenter wrote and audio-recorded a 60-s script for the event, following the commonly used script-driven imagery procedure (Orr et al., 1998).

In the second session, participants were randomized to one of three study conditions: rumination, imagery, or distraction. Randomization was stratified by PTSD status so that there were approximately even numbers of participants with and without PTSD in each condition. A research assistant first attached electrodes to the participant for psychophysiological data acquisition, and participants were given a few minutes to acclimate prior to closing their eyes for an 8-min resting baseline assessment. Participants then completed questionnaires, provided their first saliva sample, and listened to a neutral script that was not analyzed in this study. They were then asked to complete the preinduction Positive and Negative Affect Scale–X (PANAS; Watson et al., 1988) and Self-Assessment Manikin (SAM; Bradley & Lang, 1994), provided their second saliva sample, and then closed their eyes and heard a 1-min trauma script followed by an 8-min induction (i.e., rumination, imagery, or distraction; described in detail later). Following the induction, participants completed the postinduction PANAS and SAM and provided their third saliva sample. Finally, participants were asked to sit with their eyes closed for an 8-min recovery period, after which the final saliva sample was

collected. Participants then completed a check-out interview to assess any lingering distress and were paid $60 (USD) for their participation.

Induction procedures

Participants were randomly assigned to complete one of three inductions (i.e., rumination, imagery, or distraction). All inductions were prerecorded; study stimuli and ratings were presented and collected using EPrime 2.0 software (Psychology Software Tools, Inc., Sharpsburg, PA). Each induction comprised a set of 16 audio prompts, which were provided every 30 s for 8 min. The trauma-focused rumination prompts were adapted from the self-focused rumination induction commonly used in the depression literature (Nolen-Hoeksema et al., 2008). These standard self-focused rumination prompts are designed to prompt verbal thinking about one's feelings and their causes and consequences (e.g., "Think about why you react the way you do"). For this study, a subset of prompts was selected and adapted to focus on feelings about the traumatic event specifically (e.g., "Think about why you react the way you do to the event"). The trauma imagery prompts were created for this study and were designed to encourage mental imagery focused on the traumatic event (e.g., "Think about what you saw at the time of the event"). The distraction prompts were a subset of those from the distraction induction commonly contrasted with rumination (Nolen-Hoeksema et al., 2008). The distraction induction is designed to be externally focused and to distract one's attention away from one's feelings (e.g., "Think about the movement of a fan on a warm day"). See the Supplementary Materials for the full text of each induction.

Measures

Prescreening measures

Demographic characteristics and health. To determine study eligibility, participants completed an online prescreening questionnaire to capture demographic characteristics, medical history, and other inclusion and exclusion criteria.

Lifetime trauma exposure. The widely used LEC-5 (Weathers, Blake, et al., 2013b) was used to screen for trauma exposure. Participants were asked whether they had directly or indirectly experienced 17 potentially traumatic events and identify which event they considered to be their "worst" (i.e., index) traumatic event.

Self-report PTSD symptom screener. Participants completed the self-report, 20-item PTSD Checklist for *DSM-5* (PCL-5; Weathers, Litz, et al., 2013), using the index traumatic event identified on the LEC-5 as a reference point. This measure was used to oversample individuals with probable PTSD and exclude those who reported dissociative symptoms. The PCL-5 was only used during prescreening and not for any analyses.

PTSD clinical interview

PTSD symptom severity and diagnosis were assessed using the CAPS-5 (Weathers, Blake, et al., 2013a; Weathers et al., 2018), a structured clinical interview that assesses the 20 core *DSM-5* PTSD symptoms. A diagnosis of PTSD was determined by *DSM-5* criteria (i.e., item endorsement at a level of *moderately* or higher for requisite symptoms), symptoms duration of at least 1 month, and clinically significant distress or impairment. Interviews were administered and audio-recorded by trained graduate students, and all interviews were rated by a second graduate student to determine interrater reliability, which was excellent, with an intraclass correlation coefficient of .99. The CAPS-5 was used as the measure of PTSD for all analyses.

Negative affect

We administered the 60-item expanded version of the PANAS (Watson et al., 1988) to assess participants' current emotional state. Participants were instructed to report how they feel "right now (that is, at the present moment)" for each item, rating responses on a 5-point Likert-type scale ranging from 1 (*very slightly or not at all*) to 5 (*extremely*). We analyzed the 10-item Negative Affect subscale, six-item Fear subscale, and five-item Sadness subscale. The PANAS has demonstrated excellent psychometric properties, including good convergent and discriminant validity and strong test–retest reliability. In the current sample, Cronbach's alpha values ranged from .83 to .88.

Self-reported valence and arousal

The SAM (Bradley & Lang, 1994), a brief measure, was used to assess current feelings. Responses are rated on 9-point Likert-type scales that use five pictures as anchors. The Valence scale, which has response options ranging from *extremely happy* to *extremely sad*, and Arousal scale, with options ranging from *extremely aroused* to *extremely calm*, were used in the present study. The SAM has demonstrated strong convergent validity (Bradley & Lang, 1994).

Psychophysiological assessment

Cardiovascular signals were collected using the Mindware Hardware system and BioLab acquisition software (Mindware Tech; Westerville, OH, USA). IBI was calculated to index general physiological arousal, PEP was used to measure sympathetic arousal, and respiratory sinus arrhythmia (RSA) was used to assess parasympathetic activity. IBI is defined as the amount of time between individual heartbeats (i.e., R peaks) on the electrocardiogram waveform, measured in milliseconds per beat. IBI is the inverse of heart rate such that lower IBI values reflect a higher heart rate and higher degree of arousal. PEP is a systolic time interval representing the period from the onset of ventricle depolarization to the ejection of blood from the left ventricle (i.e., opening of the aortic valve; Newlin & Levenson, 1979). PEP was calculated as the time interval between the Q peak and B point on the ECG and cardiac impedance waveforms, respectively. A spectral measure of RSA, high-frequency heart rate variability, was collected with the frequency band set to 0.12–0.40 Hz (Berntson et al., 1997). For electrocardiogram recordings, three disposable electrodes filled with electrolyte paste were placed on the right clavicle, lower left rib, and lower right rib, in a Lead–II configuration. For cardiac impedance recordings, four paired impedance electrodes were placed bilaterally on the neck, torso, and back.

All psychophysiological data were visually inspected, and artifacts were manually corrected using MindWare software (MindWare Technologies, Inc.; Gahanna, OH, USA). The Mindware software was used to compute one value for IBI, PEP, and RSA for each 60-s segment of data collected within each epoch (i.e., baseline, induction), for a total of eight segments per epoch. These eight values were then averaged to create one baseline and one induction value for each cardiovascular measure. Outlying values over 3 standard deviations from the mean were dropped. Note that all reported analyses were also run with the outliers included, and the results did not change.

Salivary biomarkers

sAA. Participants provided repeated samples of saliva by passive drool into sterile cryogenic vials at four points. Vials were stored at -20°C and shipped in one batch to the lab at the University of Trier (Trier, Germany) for assay. All assayed variables were sufficiently normal and did not require transformation. Duplicate assays of sAA were conducted using a 2-chloro-4-nitrophenyl-alpha-D-maltotrioside method and spectrophotometric measurement (Lorentz et al., 1999). The intra-assay coefficient of variation (CV) for sAA ranged from 2.8% to 6.3%, and interassay CV ranged from 5.5% to 7.6%. Six outliers were winsorized to 3 standard deviations above the mean with respect to the full group for baseline and to condition for Salivary Samples 3 and 4. Data from 135 participants remained in the final models, with 390 samples; 15 samples could not be collected or could not be assayed due to sample quality issues.

Salivary cortisol. Salivary cortisol was assayed in duplicate using time-resolved fluorescent-detection immunoassay (DELFIA; Dressendörfer et al., 1992). Five outliers were winsorized to 3 standard deviations above the mean with respect to the full group for baseline and with respect to condition for Salivary Samples 3 and 4. Cortisol intra-assay CV ranged from 4.0% to 6.7%, and interassay variation ranged from 7.0% to 9.0%. Data from 135 participants remained in the final models, with 385 total samples; 20 samples could not be collected or assayed.

Data analysis

We planned a series of repeated-measures analyses of variance (ANOVAs) to test self-reported and cardiovascular outcomes. For each model, we planned to enter time (pre- or postinduction) as a within-person factor and PTSD (PTSD or no PTSD) and condition (rumination, distraction, or imagery) as between-person factors. We predicted significant interactions between time and condition for all outcomes. Specifically, we predicted that the highest levels of negative affect, fear, self-reported arousal, general arousal (IBI), sympathetic arousal (PEP), and parasympathetic withdrawal (drops in RSA) would emerge in the trauma imagery condition, followed by trauma-focused rumination and, finally, distraction. However, given the robust effects of rumination on sad mood in prior research (Nolen-Hoeksema et al., 2008; Zetsche et al., 2009), we predicted that trauma imagery and trauma-focused rumination would elicit similar levels of subjective sadness and self-reported negative valence, which would be significantly higher than those found for distraction. We further predicted significant three-way interactions between time, condition, and PTSD such that the effects of condition would be greater for individuals with PTSD compared to trauma-exposed controls. A power analysis indicated that a sample size of 111 would provide a power of .80 to detect small effects (i.e., $f = .15$ at an alpha level of .05) for within–between interactions. We recruited a sample of 135 participants to allow for any issues with psychophysiological data acquisition.

For our two salivary outcomes, we planned multilevel growth curve modeling in SAS (Version 9.4) to examine whether the effect of PTSD on biomarker reactivity

(i.e., quadratic rise and fall after baseline) depended upon experimental condition (i.e., PTSD x Condition x Quadratic Time interaction). A preliminary examination indicated that levels of both biomarkers unexpectedly fell from the first to second saliva sample; for this reason, and because the conditions did not differ until after Salivary Sample 2, we elected to model potential reactivity across Salivary Samples 2, 3, and 4. Models included linear and quadratic effects of time; time was orthonormalized. Condition was coded dimensionally in order of increasing hypothesized threat (i.e., distraction = -1, rumination = 0, imagery = 1). An autoregressive covariance structure with random slopes and intercepts was used. In the event of significant findings, we planned to examine whether the results persisted after adjusting for common covariates for biomarkers.

RESULTS

Manipulation and randomization checks

As a manipulation check, we asked participants to rate the extent to which they felt "driven to dwell" on the induction prompts. A planned contrast revealed that participants in the rumination condition rated this item significantly higher than those in the other two conditions, $t(127) = 2.30$, $p = .024$, as predicted. We also assessed the extent to which participants with and without PTSD were matched and the success of randomization to condition. The PTSD and trauma-exposed control groups did not significantly differ on any variables except CAPS-5 score, $t(133) = 17.02$, $p < .001$, $d = 2.95$, and educational attainment, $\chi^2(2, N = 135) = 6.61$, $p = .037$, with more control participants reporting having received a bachelor's degree (see Table 1). The conditions did not significantly differ on any variables except gender, $\chi^2(2, N = 135) = 6.37$, $p = .041$, such that significantly fewer men were randomly assigned to rumination than distraction, neither of which differed from imagery. As a robustness check, we reran all analyses with educational attainment and gender as covariates; the significance of the findings did not change.

Subjective emotional response

Data for two participants were lost due to equipment error, leaving 133 participants with subjective emotion ratings. We ran a repeated-measures ANOVA with time as a within-person variable (preinduction, postinduction), and PTSD (PTSD, trauma-exposed control) and condition (rumination, distraction, imagery) as between-person predictors. We found a significant main effect of time indicating that participants, in general, showed a significant increase in negative affect following the induction, $F(1, 127) = 154.67$, $p < .001$, $\eta_p^2 = .55$. As expected, a significant interaction emerged between time and PTSD, $F(1, 127) = 7.52$, $p = .007$, $\eta_p^2 = .06$, such that participants with PTSD showed significantly larger increases in negative affect than trauma-exposed controls. A significant Time x Condition interaction also emerged, $F(2, 127) = 8.66$, $p < .001$, $\eta_p^2 = .12$, but, contrary to predictions, no three-way interaction emerged for Time x PTSD x Condition, $F(2, 127) = 0.71$, $p = .492$, $\eta_p^2 = .01$. To follow-up the significant interaction of Time x Condition, we ran the same repeated-measures ANOVAs for each pair of conditions. Relative to distraction, the increase in negative affect was significantly larger in the rumination, $F(1, 83) = 12.21$, $p = .001$, $\eta_p^2 = .13$, and imagery conditions, $F(1, 86) = 13.47$, $p < .001$, $\eta_p^2 = .14$; however, rumination and imagery did not significantly differ from each other, $F(1, 85) = 0.03$, $p = .857$, $\eta_p^2 < .001$ (see Figure 1).

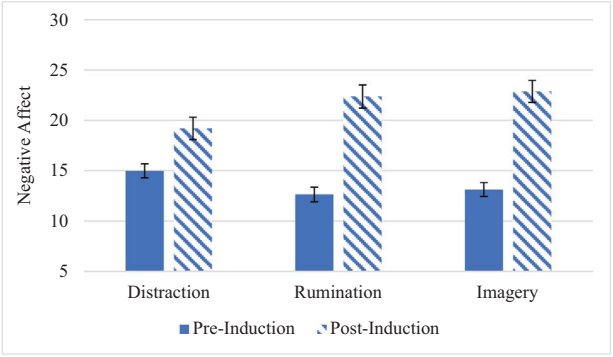

FIGURE 1 Effects of condition on negative affect
Note: Error bars reflect standard errors for marginal means

To explore negative affect-related findings further, we ran the same analyses for sadness and fear. In general, participants showed significant increases in both fear, $F(1,127) = 71.02$, $p < .001$, $\eta_p^2 = .36$, and sadness, $F(1, 127) = 119.31$, $p < .001$, $\eta_p^2 = .48$. Significant Time x PTSD interactions indicated that participants with PTSD showed significantly larger increases in both fear, $F(1,127) = 5.98$, $p = .016$, $\eta_p^2 = .05$, and sadness, $F(1, 127) = 9.46$, $p = .003$, $\eta_p^2 = .07$, compared to trauma-exposed controls. Significant Time x Condition interactions emerged for both fear, $F(2, 127) = 3.92$, $p = .022$, $\eta_p^2 = .06$, and sadness, $F(2, 127) = 7.59$, $p = .001$, $\eta_p^2 = .11$, but no significant three-way interactions emerged for Time x PTSD x Condition, $ps = .347–.366$, $\eta_p^2 s = .02–.02$. Follow-up analyses indicated that, relative to distraction, the rumination condition did not show a significantly larger increase in fear, $F(1,83) = 3.77$, $p = .056$, $\eta_p^2 = .04$, but did show a significantly larger increase in sadness, $F(1,83) = 7.93$, $p = .006$, $\eta_p^2 = .09$. Relative to distraction, participants in the imagery condition showed significantly larger increases in both fear, $F(1, 86) = 8.36$,

$p = .005$, $\eta_p^2 = .09$, and sadness, $F(1, 86) = 12.70$, $p = .001$, $\eta_p^2 = .13$. Participants in the rumination and imagery conditions showed similar increases in fear, $F(1, 85) = 0.55$, $p = .459$, $\eta_p^2 = .006$, and sadness, $F(1, 85) = 1.30$, $p = .257$, $\eta_p^2 = .02$.

For self-reported arousal, we found a significant effect of time, indicating a significant increase in arousal, $F(1, 127) = 41.76$, $p < .001$, $\eta_p^2 = .25$. As expected, a significant interaction emerged for Time x PTSD, $F(1, 127) = 9.47$, $p = .003$, $\eta_p^2 = .07$, such that participants with PTSD showed larger increases in arousal than controls. A significant interaction also emerged for Time x Condition, $F(2, 127) = 4.32$, $p = .015$, $\eta_p^2 = .06$, but, contrary to predictions, no significant three-way interaction emerged (Time x PTSD x Condition), $F(2, 127) = 0.07$ $p = .928$, $\eta_p^2 = .001$. To follow up on the significant Time x Condition interaction, we compared each pair of conditions. Relative to distraction, the increases in arousal were significantly larger in the rumination, $F(1, 83) = 7.25$, $p = .009$, $\eta_p^2 = .08$, and imagery conditions, $F(1, 86) = 4.80$, $p = .031$, $\eta_p^2 = .05$; however, rumination and imagery did not significantly differ, $F(1, 85) = 0.19$, $p = .668$, $\eta_p^2 = .002$.

For self-reported valence, we again found a significant main effect of time, indicating that valence was rated as more negative following the induction, $F(1, 125) = 187.01$, $p < .001$, $\eta_p^2 = .60$. A significant Time x Condition interaction also emerged, $F(2, 125) = 7.78$, $p = .001$, $\eta_p^2 = .11$, but no significant interactions emerged for Time x PTSD, $F(1, 125) = 2.73$, $p = .101$, $\eta_p^2 = .02$, or Time x PTSD x Condition, $F(2, 125) = 0.30$, $p = .743$, $\eta_p^2 = .005$. To follow up on the significant Time x Condition interaction, we again compared each pair of conditions. Relative to distraction, valence change was significantly larger in the rumination, $F(1, 81) = 11.23$, $p = .001$, $\eta_p^2 = .12$, and imagery conditions, $F(1, 85) = 10.13$, $p = .002$, $\eta_p^2 = .11$. Rumination and imagery did not significantly differ, $F(1, 84) = 0.22$, $p = .641$, $\eta_p^2 = .003$.

Physiological reactivity

We measured cardiovascular physiology during an 8-min resting baseline and the 8-min induction. We were unable to obtain valid physiological signals for a subset of participants, and outliers were excluded, leaving useable data for 124 participants for IBI, 113 participants for PEP, and 124 participants for RSA.

General physiological arousal (IBI)

The planned repeated-measures ANOVA revealed a significant main effect of Time (baseline, induction), $F(1,118) = 6.84$, $p = .010$, $\eta_p^2 = .06$, that was qualified by a significant Time x Condition interaction, $F(2,118) = 9.60$, $p < .001$, $\eta_p^2 = .14$. The Time x PTSD and Time x PTSD x Condition interactions were not significant, $ps = .518–.799$, $\eta_p^2 s = .001–.01$, and no significant between-person effects emerged, $ps = .141–.865$, $\eta_p^2 s = .00–.03$ To follow up the significant Time x Condition interaction, we compared each pair of conditions. Change in IBI was significantly larger in distraction condition compared with rumination, $F(1, 78) = 15.06$, $p < .001$, $\eta_p^2 = .16$, and imagery, $F(1, 81) = 14.55$, $p < .001$, $\eta_p^2 = .15$. Rumination and imagery did not significantly differ from each other, $F(1, 77) = 0.05$ $p = .823$, $\eta_p^2 = .001$. The results of paired sample t tests indicated that the distraction condition showed a significant increase in IBI, $t(42) = 5.53$, $p < .001$, $d = 0.84$, indicating a decrease in heart rate, whereas the rumination, $t(38) = 0.07$, $p = .944$, $d = 0.01$, and imagery, $t(41) = 0.34$, $p = .740$, $d = 0.05$, conditions showed no change in IBI (see Figure 2, Panel A).

Sympathetic nervous system arousal (PEP)

For PEP, no significant effects emerged for Time, $F(1, 107) = 0.006$, $p = .937$, $\eta_p^2 < .001$; Time x PTSD, $F(1, 107) = 1.27$, $p = .262$ $\eta_p^2 = .01$; Time x Condition, $F(2, 107) = 0.30$, $p = .745$ $\eta_p^2 = .005$; or Time x PTSD x Condition, $F(2, 107) = 1.40$, $p = .252$, $\eta_p^2 = .03$. A significant between-person effect emerged for PTSD such that participants with PTSD showed significantly lower PEP ($M = 106.15$, $SE = 1.95$), indicating more sympathetic arousal, compared to participants without PTSD ($M = 112.97$, $SE = 1.77$) throughout the experimental session, $F(1, 107) = 6.69$, $p = .011$, $\eta_p^2 = .06$. No other between-person effects were significant, $ps = .961–.968$, η_p^2 s $= .001–.001$.

Despite the nonsignificant Time x Condition interaction, we ran exploratory follow-up paired sample t tests examining change in PEP from baseline to induction in each condition separately to facilitate comparison with the significant IBI results. PEP did not significantly change following any of the three inductions: rumination, $t(36) = 0.15$, $p = .880$, $d = 0.03$, imagery, $t(37) = 0.52$, $p = .607$, $d = 0.08$; distraction, $t(37) = 0.72$, $p = .476$, $d = 0.12$ (see Figure 2, Panel B).

Parasympathetic nervous system arousal (RSA)

The RSA analyses also indicated that there were no significant effects of Time, $F(1,118) = 0.05$, $p = .819$, $\eta_p^2 < .001$; Time x PTSD, $F(1,118) = 0.83$, $p = .364$, $\eta_p^2 = .007$; Time x Condition, $F(2,118) = 2.19$, $p = .117$, $\eta_p^2 = .04$; or

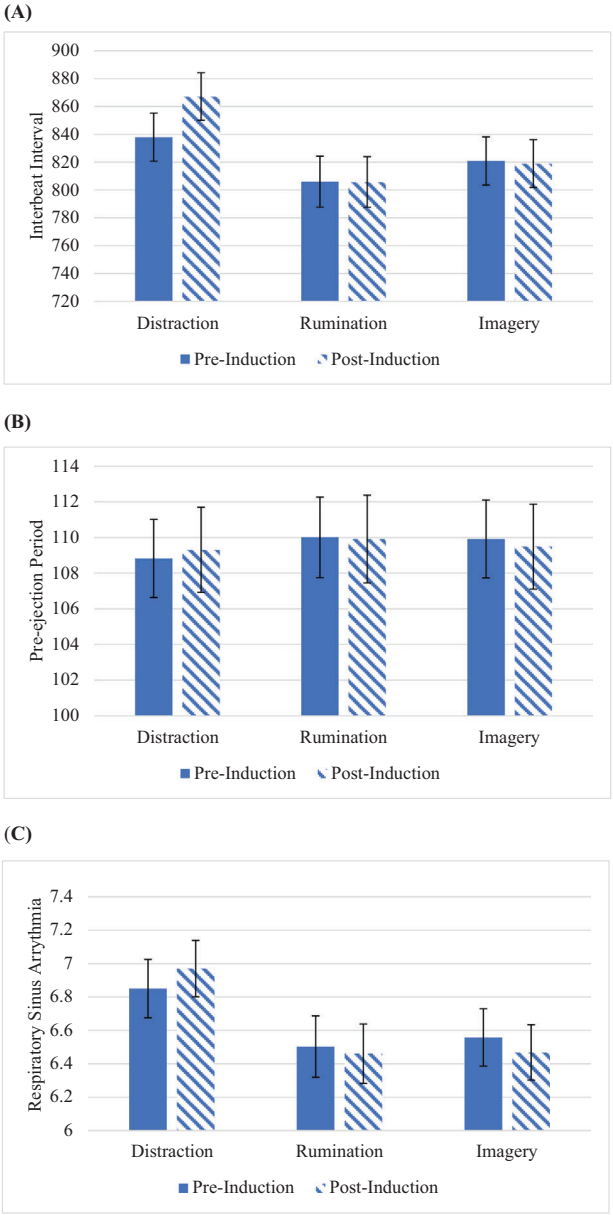

FIGURE 2 Effects of condition on (A) general physiological arousal, (B) sympathetic arousal, and (C) parasympathetic activation

Note: In Panel A, increases in interbeat intervals (i.e., the amount of time between heartbeats) reflect decreases in heart rate and, thus, decreases in overall arousal. In Panel B, higher preejection period values reflect less sympathetic activity. In all panels, error bars reflect standard errors for marginal means.

Time x PTSD x Condition, $F(2,118) = 1.60$, $p = .205$, $\eta_p^2 = .03$. No between-person effects emerged, $ps = .055–.451$, $\eta_p^2 s = .005–.05$. Despite the nonsignificant Time x Condition interaction, we again ran exploratory follow-up paired sample t tests. RSA significantly increased from pre- to postinduction in the distraction, $t(41) = 2.14$, $p = .039$, $d = 0.33$, but not rumination, $t(38) = 0.75$, $p = .456$, $d = 0.12$, or imagery conditions, $t(42) = 1.22$, $p = .231$, $d = 0.19$ (Figure 2, Panel C).

sAA and cortisol

The results indicated an on-average flat pattern in sAA after baseline (Salivary Sample 2) rather than reactivity irrespective of condition. The hypothesized PTSD x Condition x Quadratic Time interaction was not significant for sAA, $B = 4.48$, $SE = 6.74$, $t(124) = 0.66$, $p = .508$. The results also indicated a pattern of declining cortisol following baseline rather than reactivity irrespective of condition. There were no significant effects of PTSD or condition, and the PTSD x Condition x Quadratic Time interaction was not significant for cortisol, $B = -0.51$, $SE = 0.26$, $t(122) = -1.95$, $p = .053$. Given the general pattern of declining cortisol values rather than reactivity, we did not pursue this further. Full models are presented in Supplementary Tables S1 and S2; levels are shown in Figures 3 and 4.

DISCUSSION

The purpose of this study was to examine the emotional and physiological effects of trauma-focused rumination among individuals with and without PTSD. We aimed to clarify whether trauma-focused rumination heightens emotional and physiological responses compared with externally focused distraction, consistent with symptom exacerbation, and whether rumination dampens emotional and physiological responses relative to trauma-related imagery, consistent with cognitive avoidance. The findings were broadly consistent with the symptom exacerbation mechanism of trauma-focused rumination in that rumination led to more subjective distress than distraction. In contrast, we found no evidence that trauma-focused rumination dampened emotional or physiological responses relative to trauma imagery, offering no support for the cognitive avoidance mechanism.

Our manipulation check indicated that participants in the trauma-focused rumination condition reported feeling more "driven to dwell" than those in the other conditions, as expected. We also found that the sample as a whole reported significant increases in distress from pre- to postinduction (i.e., significant effects of time), indicating that participants were emotionally engaged with the task. Consistent with predictions, individuals with PTSD exhibited larger increases in subjective distress than individuals without PTSD, as indicated by significant Time x PTSD interactions for all emotions except valence. This finding indicates that the trauma script procedure was sensitive to PTSD-related differences.

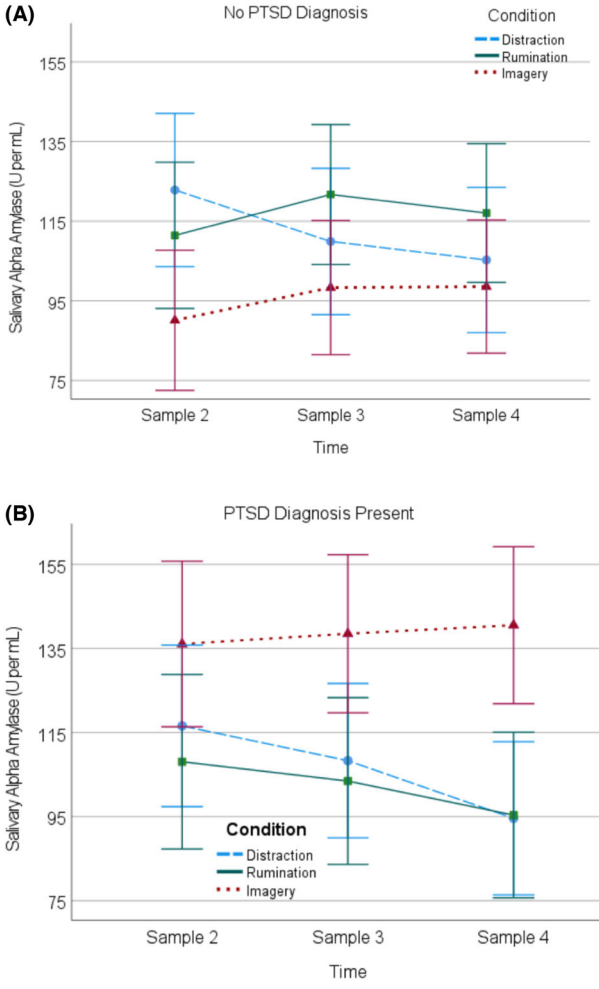

FIGURE 3 Effects of condition on salivary alpha-amylase, by posttraumatic stress disorder (PTSD) diagnostic status
Note: Error bars reflect standard errors for marginal means.

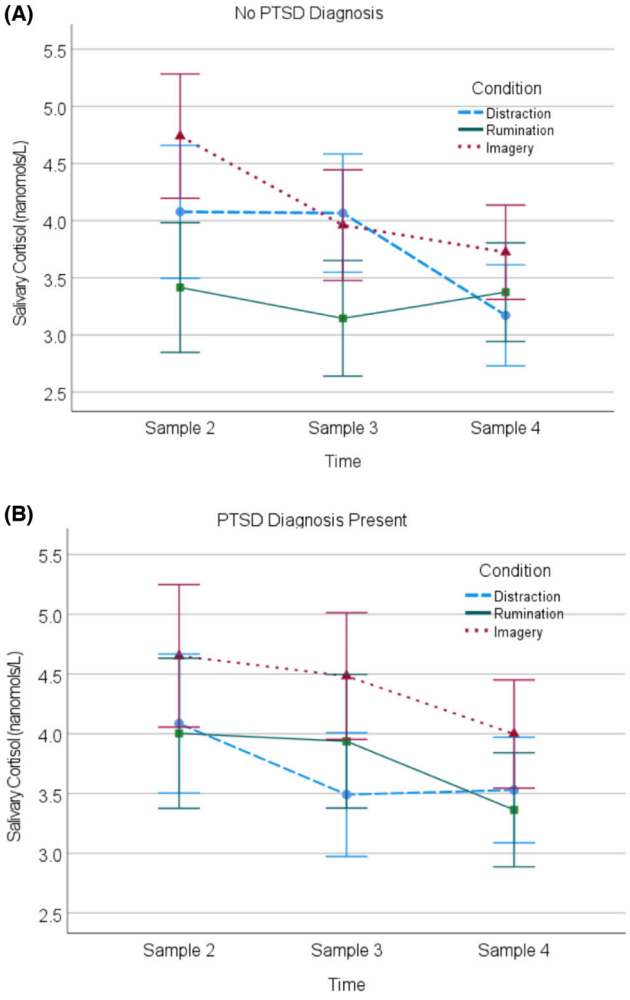

FIGURE 4 Effects of condition on salivary cortisol, by posttraumatic stress disorder (PTSD) diagnostic status
Note: In all panels, error bars reflect standard errors for marginal means.

In terms of physiological reactions, the experimental inductions did not elicit the expected effects. We expected that participants in the rumination and imagery conditions would show increases in physiological arousal across all cardiovascular indicators and salivary biomarkers, but we instead found no significant increases. Participants in the distraction condition showed an unexpected decrease in physiological arousal on two measures, IBI and RSA, as discussed further later in this section. Also counter to predictions, individuals with PTSD did not exhibit larger physiological reactions to the inductions compared with those without PTSD. These unexpected findings may be due to our use of a novel imagery induction that had some methodological differences from typical script-driven imagery (e.g., Orr et al., 1998), including a longer duration and the use of audio-recorded prompts to focus attention. Thus, findings related to physiological measures and biomarkers should be interpreted with some caution.

The results for subjective emotional responses were remarkably consistent. Participants in general, across conditions, showed significant increases in negative affect, fear, sadness, self-reported arousal, and self-reported negative valence following the induction. These increases in subjective distress were significantly larger in the rumination and imagery conditions than in the distraction condition for all measures except for fear in rumination versus distraction ($p = .056$). These findings are consistent with the symptom exacerbation account that rumination leads directly to PTSD symptoms, such as increased emotional reactivity to trauma reminders. These findings are also consistent with a large body of research indicating that rumination about depressed mood prolongs distress (Nolen-Hoeksema et al., 2008) and shows that these findings generalize to trauma-focused rumination.

Interestingly, PTSD status did not moderate the effect of condition on any subjective emotions. As mentioned, there were significant Time x PTSD effects, but the three-way Time x PTSD x Condition interactions were not significant. This pattern of results indicates that individuals with PTSD have stronger emotional reactions than individuals without PTSD, and that trauma-focused rumination leads to stronger emotional reactions than distraction, but that the relative effects of rumination versus distraction are of a similar magnitude for individuals with and without PTSD. In other words, trauma-focused rumination prolongs negative mood among individuals with or without PTSD. This effect among individuals who already have PTSD could explain why rumination maintains PTSD symptoms over time, and this effect among individuals without PTSD could explain why rumination increases the risk for future PTSD among individuals who do not currently have the disorder. Our finding that rumination prolongs momentary negative mood, coupled with prior literature indicating that rumination prospectively predicts the development of PTSD (Michael et al., 2007), suggests that trauma-focused rumination leads both to short- and long-term negative consequences.

As mentioned, the results concerning physiological measurements and biomarkers ran counter to predictions. Neither sAA nor cortisol showed patterns of trauma reactivity, and no effects of condition were observed for either biomarker. For cardiovascular indicators, trauma-focused rumination was associated with no changes rather than the expected increase in overall and sympathetic arousal. In contrast, distraction was associated with a significant decrease in overall physiological arousal (i.e., IBI) and an increase in parasympathetic activity (i.e., RSA), and this effect was not moderated by PTSD status. This result is consistent with findings from a prior study using a trauma analog film, which also demonstrated that heart rate did not change during rumination but significantly decreased during distraction (Ehring et al., 2009). Distraction is commonly taught in interventions such as dialectical behavior therapy (Lynch et al., 2007) as a short-term strategy for regulating intense emotions. Our findings offer a potential physiological explanation for the well-established short-term emotional benefits of distraction such that distraction may downregulate physiological arousal by activating the parasympathetic nervous system.

Based on theories of trauma-focused rumination as a form of cognitive avoidance, we had expected trauma-focused rumination to elicit less subjective distress and less physiological arousal than trauma imagery. The findings offered no support for the cognitive avoidance model. The trauma-focused rumination and imagery conditions did not differ significantly from each other on any subjective emotional, physiological, or biomarker measure, and effect sizes were small for all comparisons (i.e., η_p^2s $\leq$ .02). Our findings stand in contrast to prior research indicating that, in general, mental imagery leads to stronger affective responses than verbal thought (Holmes & Mathews, 2010). However, prior work has typically compared mental imagery to verbal thought using potentially negative hypothetical scenarios, not experienced events, and such hypothetical scenarios are likely less distressing than one's worst traumatic memory. It is possible that our findings reflect a ceiling on the evocative effects of mental imagery—thinking about one's worst trauma is so distressing that imagery cannot further exacerbate the distress elicited by verbal thoughts. Nevertheless, this pattern draws into question the possible avoidant function of trauma-focused rumination. If trauma-focused rumination is just as distressing as trauma imagery, rumination cannot allow one to avoid the distress associated with trauma-related images. Because, to our knowledge, this was the first experimental test of the cognitive avoidance mechanism, no definitive conclusions can be drawn. It is possible that the findings are an artifact of the specific imagery condition that we used and that a more evocative or different kind of imagery induction would lead to a different pattern of results. If replicated, however, these findings could have important implications for the field's understanding of the role of trauma-focused rumination in PTSD risk. Rather than serving as cognitive avoidance, rumination may lead to PTSD through other mechanisms, such as leading to symptoms directly (i.e., the symptom exacerbation account) or by reinforcing negative beliefs about the trauma (Ehlers & Clark, 2000).

These findings also have potential implications for exposure-based PTSD treatment. Counter to common clinical reasoning, mental imagery may not lead to more emotional engagement than verbal representations. Indeed, PTSD treatment can be effective with trauma exposure grounded either in mental imagery (e.g., imaginal exposure in prolonged exposure therapy; Foa, 2011) or in verbal processing (e.g., written trauma narratives of written exposure therapy; Sloan & Marx, 2019). These findings suggest that engaging with the trauma either verbally or through imagery may be effective and that clinicians should not assume that verbal modes of processing will lead to underengagement.

To our knowledge, this study represents the first experimental manipulation of trauma-focused rumination among individuals with and without PTSD. Experimental manipulations offer stronger support for causal claims than correlational designs, and the inclusion of two comparison conditions permitted us to examine symptom exacerbation and cognitive avoidance accounts within the same study. Other methodological strengths of this study include a clinical sample of individuals with a PTSD

diagnosis derived from a clinician-administered interview and a trauma-exposed control group matched with the PTSD group on key variables, including trauma type.

The study also has some important limitations to discuss. We used a mixed-trauma sample in which sexual assault was the most commonly reported traumatic event; the extent to which the findings generalize to other trauma-exposed samples (e.g., combat veterans) is unknown. The exclusion criteria (e.g., current use of antidepressant medications) could also affect the generalizability of the findings. Like all induction-based experiments, we cannot ensure that participants followed the instructions. Although the inductions had the predicted effects on subjective emotions, there were several unexpected findings for physiological outcomes, including an unexpected drop in salivary markers between the two baseline salivary samples and surprising null findings for PTSD diagnostic status. Future research using a different trauma imagery task could clarify the physiological effects of trauma-focused rumination relative to imagery. Future research should also include a manipulation check in the form of a question about the extent to which participants used mental imagery versus verbal processing during the inductions. Other limitations are that the control group reported significantly higher educational attainment than the PTSD group and that more men were randomly assigned to the rumination condition, although robustness checks indicated these variables did not affect the results. We also grouped participants based on their current PTSD diagnostic status; future research could use continuous measures of PTSD symptom severity or include a subthreshold PTSD comparison group to examine the effects of subthreshold PTSD.

Taken together, the findings lend more support to the symptom exacerbation model than the cognitive avoidance model of trauma-focused rumination in PTSD. Trauma-focused rumination may be associated with the development and maintenance of PTSD due to its direct effects on PTSD symptoms, particularly increased emotional reactivity to trauma reminders. The lack of support for the cognitive avoidance function of rumination leads to an important question: If rumination is not a form of avoidance, why do people ruminate? Cognitive avoidance is maintained because it is negatively reinforced—avoiding thoughts of difficult topics reduces one's distress in the short term. If rumination increases distress in the short term and leads to negative long-term outcomes, why do people keep doing it? Prior work suggests that people hold positive beliefs about depressive rumination, such as the idea that rumination leads to insight into one's problems (Papageorgiou & Wells, 2001). Similar beliefs about trauma-focused rumination could explain why people are motivated to ruminate about traumatic events. Additionally, the contrast avoidance theory of worry (Newman & Llera, 2011) suggests that people may prefer to experience continuous negative mood rather than rapid shifts between positive and negative moods; similar processes could operate for trauma-focused rumination. It will be important for future researchers to continue to examine what factors reinforce rumination about traumatic events despite the negative consequences in both the short and long term.

OPEN PRACTICES STATEMENT

The study reported in this article was not formally preregistered. Neither the data nor the materials have been made available on a permanent third-party archive; some of the materials are available as supplementary material to this article. Requests for data or other materials can be sent via email to the first author at bewisco@uncg.edu.

AUTHOR NOTE

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ORCID

Blair E. Wisco https://orcid.org/0000-0003-2412-9012
Suzanne Vrshek-Schallhorn https://orcid.org/0000-0003-2825-200X
Casey L. May https://orcid.org/0000-0002-9482-0741
Allison A. Campbell https://orcid.org/0000-0002-7778-1795
Faith O. Nomamiukor https://orcid.org/0000-0002-1728-7402
Cameron P. Pugach https://orcid.org/0000-0001-8185-2982

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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