



Higher trait reappraisal predicts stronger HPA axis habituation to repeated stress

Lydia G. Roos^a, Johanna Janson^b, Sarah C. Sturmbauer^b, Jeanette M. Bennett^{a,c},
Nicolas Rohleder^{b,*}

^a Health Psychology PhD Program, University of North Carolina at Charlotte, United States

^b Department of Psychology, Friedrich-Alexander-Universität Erlangen-Nürnberg, Germany

^c Department of Psychological Sciences, University of North Carolina at Charlotte, United States

ARTICLE INFO

Keywords:

HPA axis
Habituation
Cortisol
Emotion regulation
Reappraisal
Suppression

ABSTRACT

Undergoing stress can be advantageous when it leads to adaptation and growth; however, failure of the hypothalamic-pituitary-adrenal (HPA) axis to habituate (i.e., nonhabituation) involves continuing to become highly activated in response to repeated exposure of the same stimulus and is considered maladaptive. Although 50–75% of individuals assessed in a laboratory exhibit adaptive habituation to repeated stress, variability in habituation suggests psychological processes used in response to stress may play a role, such as emotion regulation (ER). Nonetheless, no research to date has investigated whether ER strategies affect HPA axis habituation. We investigated whether tendency to use two ER strategies, reappraisal and suppression, influenced HPA axis habituation among 84 healthy young adults (60.7% female; $M_{age} = 24.8$ years, $SD = 6.0$) exposed to a standardized experimental stress paradigm on two consecutive days. HPA axis stress responses were assessed using salivary cortisol concentrations. We also examined whether non-manipulated state ER strategies (i.e., those used by the participant during and following the stressor on the first day) modulated HPA axis habituation over and above trait-use in a subsample ($N = 60$). Trait, but not state, reappraisal was associated with stronger HPA axis habituation. Neither trait nor state suppression were significantly associated with HPA axis habituation. These findings expand our current understanding of how ER can affect stress-related health outcomes and suggest habitual reappraisal plays an important role in adaption of the HPA axis to stress.

1. Introduction

Psychosocial stress is a common occurrence in daily life, consisting of various challenges, threats, loss, and harm (Lazarus, 1966). Undergoing stress can be advantageous when it leads to adaptation and growth. Conversely, the inability to adapt to repeated stress is linked with declines in cognitive functioning, systemic inflammation, premature aging, immune system dysfunction, and higher likelihood of suffering from metabolic disorders such as diabetes and cardiovascular disease (Glaser and Kiecolt-Glaser, 2005; Graham et al., 2006; Cohen et al., 2012; Tamashiro et al., 2011).

In response to stress, the sympathetic nervous system (SNS) and the HPA axis send signals via autonomic innervation and hormones about the threatened state of the individual, leading to heightened overall physiological activation. Adequate physiological activation and timely recovery of the stress systems are necessary for successful adaptation to

situational demands without damaging effects on the individual (McEwen, 1998). However, in addition to exaggerated or missing responses to stress exposure, failure of the HPA axis to habituate to stress has been proposed as a maladaptive pattern (McEwen, 1998; Schommer et al., 2003). Nonhabituation occurs when the HPA axis continues to become highly activated in response to a repeated exposure of the same stimulus, and leads to wear and tear on the individual (McEwen, 1998; Sapolsky et al., 2000). Nonhabituation of the HPA axis has been linked with higher self-reported depression, more physical health symptoms, and higher inflammatory responses to stress (Kirschbaum et al., 1995; Thoma et al., 2017).

Prior research has found about 50–75% of individuals examined in a laboratory exhibit adaptive habituation to repeated stress, while the remainder do not (Kirschbaum et al., 1995; Schommer et al., 2003; Wüst et al., 2005). Individual differences in HPA axis habituation might be attributed to variability in psychological factors. For example, lower

* Corresponding author at: Chair of Health Psychology, Department of Psychology, Friedrich-Alexander-Universität Erlangen-Nürnberg, Nögelsbachstr. 49a, 91052 Erlangen, Germany.

E-mail address: nicolas.rohleder@fau.de (N. Rohleder).

<https://doi.org/10.1016/j.psyneuen.2018.10.018>

Received 16 August 2018; Received in revised form 21 October 2018; Accepted 22 October 2018

0306-4530/© 2018 Elsevier Ltd. All rights reserved.

self-esteem and post-stressor rumination are associated with non-habituation to repeated stress (Kirschbaum et al., 1995; Gianferante et al., 2014). Strategies for regulating emotions, such as through reappraisal and suppression, may also influence habituation, however they have not yet been investigated as predictors of habituation to stress.

1.1. Emotion regulation and health

Emotion regulation (ER) strategies are the ways in which people influence control over which emotions they have, how they are expressed, and when they have them (Gross, 1998). Prior research has demonstrated individuals' ER strategies as predictors of how stress impacts psychological and physical health outcomes (e.g., Gross and John, 2003; Sagui and Levens, 2016). In particular, two ER strategies continually linked with health outcomes are expressive suppression and cognitive reappraisal (Gross and John, 2003).

Expressive suppression is the behavioral inhibition of emotion expression while emotionally or physiologically aroused, such as withholding facial expressions and bodily gestures (Gross, 1998), and is related to a lack of social sharing of emotions (Gross and John, 2003). Suppression is generally considered a maladaptive process when used frequently (John and Gross, 2004) and is associated with more depressive symptoms, lesser positive emotion, and greater negative emotion (Gross and John, 2003). Suppression has also been associated with heightened sympathetic nervous system activation (Gross and Levenson, 1993, 1997) and has been suggested as a contributing factor to cardiovascular disease (Mauss and Gross, 2004).

Cognitive reappraisal encompasses cognitively reinterpreting a potentially emotion-eliciting event or situation in such a way as to alter its emotional impact (Gross and John, 2003). Reappraisal is generally regarded as an adaptive method to regulate emotions; habitual use is associated with a greater experience of positive emotion and fewer depressive and anxiety symptoms (Garnefski et al., 2001; Gross and John, 2003; Troy et al., 2010). It has also been related to an increased ability to recover from negative emotionally-arousing situations both on a day-to-day basis (Meyer et al., 2012) and in response to experimentally-induced stimuli (Augustine and Hemenover, 2009). The ability to cognitively reappraise also may act as a buffer against increased body mass index and type 2 diabetes when highly stressed (Sagui and Levens, 2016).

Despite differences in suppression's and reappraisal's psychological effects, prior research has linked trait-use of both reappraisal and suppression with greater HPA axis reactivity to novel social-evaluative stressors (Lam et al., 2009), with the same effect replicated when participants were instructed to reappraise during an acute stressor (Denson et al., 2014). The disconnect between the effects of ER strategies on long-term well-being and short-term HPA axis reactivity suggests differences in how these strategies influence physiology over time, possibly via habituation, as the explanation; studies thus far that have investigated the physiological effects of these emotion regulation strategies have done so by examining single stress responses (e.g., Lam et al., 2009; Denson et al., 2014; Roos et al., 2018). Measuring responses to repeated acute stress could enhance our knowledge of how the use of suppression and reappraisal influences health over time.

1.2. Present study

The overall goal of the current investigation was to expand upon our current knowledge of how emotion regulation strategies influence health by assessing healthy young adults' habituation to a standardized laboratory stress paradigm on two consecutive days. This research expands upon our current understanding of ER and health in two ways: First, the effects of suppression and reappraisal on the habituation process are unknown; the present study addresses this gap.

Second, research on the effects of suppression and reappraisal on

stress reactivity has focused solely on the habitual use of ER strategies (i.e., trait-use) or experimentally manipulated use during the stressor (Lam et al., 2009; Denson et al., 2014) and does not account for possible differences of individuals' use of chosen ER strategies during and following the stressor. Therefore, we also examined whether specific ER strategies used in response to this particular stressor (i.e., state ER strategy) modulates habituation of the HPA axis habituation over and above trait-use. Measuring specific ER strategies in response to a repeated acute stressor allows us to assess factors influencing habituation to stress and could enhance our knowledge of how suppression and reappraisal use affect health.

2. Methods

2.1. Participants

Participants were 97 healthy young adults (65.6% female) with an average age of 24.8 years ($SD = 6.0$) with a body mass index (BMI) less than 30 kg/m^2 ($M = 22.3$, $SD = 2.4$). They were primarily German (92.8%) and identified as White (97.9%). Volunteers were recruited from the Friedrich-Alexander-Universität Erlangen-Nürnberg campus via print and multi-media advertising.

Participants were screened using an online survey tool ("Unipark", Questback, Germany) prior to their first visit for inclusion and exclusion criteria standard for acute laboratory stress studies with endocrinological and immunological assessments. Volunteers were eligible for the study if they met the following eligibility criteria: 1) between 18 and 35 years old, 2) body mass index (BMI) between 18 and 30 kg/m^2 , 3) non-smoker, 3) not dependent on alcohol or illicit drugs, 4) not taking medications (e.g., anti-depressants, beta blockers, glucocorticoids) except hormonal contraceptives in women, or have medical conditions known to have immunomodulatory or neuroendocrine effects, and 5) no major psychiatric disorders or psychiatric symptoms as measured by the German version of the Centers for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977; Hautzinger et al., 2012) during screening; participants with scores above the cutoff of 22 were not invited to participate, since scores above 22 have been shown to indicate the presence of depressive symptomatology (Hautzinger et al., 2012). If eligible, participants were contacted for scheduling their laboratory visits and were directed to an additional online survey used to collect data on trait self-report measures (including trait ER). Pre-menopausal female participants were scheduled for their laboratory visits in the luteal phase of their menstrual cycle. Participants were compensated with 45 EUR for their time. All procedures and measures were approved by Friedrich-Alexander-University Erlangen-Nürnberg's Faculty of Medicine Institutional Review Board.

2.2. Procedures

2.2.1. Laboratory visit 1

Participants were scheduled for their lab visit in the afternoon between 14:00 and 18:00 h to control for circadian cortisol rhythm. Participants were instructed to refrain from exercising for 24 h prior to the visit, and from brushing teeth, or eating or drinking anything except water for one hour before the visit. Upon arrival, informed consent was obtained and baseline saliva samples were collected. Thirty-three of the 97 participants were also fitted with an indwelling catheter for blood draws to assess inflammation in a subsample of participants. The subsample that was fitted for blood draws was not significantly different from the other participants on any sociodemographic or key study variable, including trait or state ER strategies or cortisol habituation.

After a 45-min rest period, another saliva sample (-1) was collected prior to being introduced to a standardized socially evaluative acute stressor, the Trier Social Stress Test (TSST; Kirschbaum et al., 1993). The TSST is a standardized procedure that involves public speaking and verbal arithmetic task in front of a group of committee members and

reliably activates biological systems (Dickerson and Kemeny, 2004). Per established procedures at the Chair of Health Psychology (e.g., Breines et al., 2014; Janson and Rohleder, 2017), participants were escorted into a testing room with one male and one female committee member. Participants were given five minutes to prepare for a speech in which they would describe why they were the best candidate for their dream job. After the preparation period, participants were instructed to stand and deliver the five-minute speech in front of the committee, who refrained from providing verbal and nonverbal (e.g., facial expression) feedback or encouragement throughout the TSST. Following the speech, participants were asked to serially subtract as quickly as possible for an additional five minutes; they were instructed to start from the beginning again if they made an error. The speech and serial subtraction portions were audio- and video-taped. Saliva samples were taken at +1, +10, +20, +30, +45, and +60 min after TSST to assess reactivity.

2.2.2. Laboratory visit 2

The second visit took place on the following day at approximately the same time. Procedures were similar to visit 1 procedures, with two exceptions: 1) During the rest period prior to the second TSST, participants reported what ER strategies were used relating to visit 1's TSST as an indicator of state ER strategy, and 2) A different arithmetic challenge was used in the second TSST (i.e., participants were asked to subtract 17 from 2043 on day 1, and 13 from 2011 on day 2). One or both committee members changed for 78% of the participants.

The addition of state ER strategies occurred after data collection started; thus, only 60 participants provided this information. This subsample was not significantly different from the other participants on sociodemographic or key study variables. Participants were debriefed at the completion of their second laboratory visit.

2.3. Measures

2.3.1. Trait emotion regulation

Trait ER strategy was assessed using a German version of the Emotion Regulation Questionnaire (ERQ; Gross and John, 2003), a 10-item self-report scale used to assess the habitual use of cognitive reappraisal and expressive suppression. The reappraisal subscale consists of 6 items, including “When I am faced with a stressful situation, I make myself think about it in a way that helps me stay calm.” The suppression subscale consists of 4 items such as “I keep my emotions to myself.” Respondents indicate how strongly they agree with statements on a 7-point Likert scale (1 = strongly disagree, 7 = strongly agree). The German ERQ demonstrated good internal consistency for reappraisal ($\alpha = .84$) and acceptable internal consistency for suppression ($\alpha = .69$) in the current sample. Participants completed the ERQ via an online survey prior to their first visit.

2.3.2. State emotion regulation

State ER strategy was assessed using a German version of the State Emotion Regulation Questionnaire (SERQ; Kashdan and Steger, 2006). The SERQ is an 8-item measure modified slightly from the ERQ to capture attempts to regulate emotions during the day using reappraisal or suppression and was used to assess ER strategy used regarding the TSST. The reappraisal and suppression subscales consist of 4 items each and respondents indicate how strongly they agree with statements on a 7-point Likert scale (1 = strongly disagree, 7 = strongly agree). The German SERQ demonstrated good internal consistency for the reappraisal ($\alpha = .87$) subscale and acceptable internal consistency for the suppression subscale ($\alpha = .66$).

2.3.3. Salivary cortisol

HPA axis stress responses were assessed using salivary cortisol concentrations. Saliva samples were collected via Salivettes (Sarstedt, Nümbrecht, Germany) and were stored for later assessment at -30°C

after laboratory sessions. Saliva samples were centrifuged at 2000 g and 20°C for ten minutes and cortisol concentrations were assessed in duplicate using chemiluminescence immunoassay (CLIA, IBL International, Hamburg, Germany). Intra- and inter-assay CVs were below 10%.

2.4. Analytic strategy

Analyses were conducted using IBM SPSS Statistics Version 25. Cortisol data were first screened for outliers using z-scores. Ten participants were removed from further analyses due to elevated baseline cortisol levels (i.e., z 's > 3.29) upon arrival or at -1 min TSST. An additional three participants were excluded following listwise deletion because of missing survey data. Thus, the final sample for trait ER strategies included 84 participants (60.7% female) with an average age of 24.8 years ($SD = 6.0$). A subsample ($N = 60$; 66.7% female) that also completed the state ER measure were included in analyses assessing TSST-specific ER use. Of note, one participant included in trait ER analyses had a missing value at +45 min; a value was imputed using the average of the participant's values at +30 and +60 min.

Shapiro-Wilk tests were used to check for normal distribution and cortisol values were log-transformed to reduce skewness. Subsequent data analyses were conducted using transformed values. Analysis of variance (ANOVA) for repeated measures was used to assess stress-induced changes in cortisol with “day” (i.e., day 1 vs. day 2) and “time” (i.e., seven time points) as the within-subject factors.

To test whether ER strategies influenced HPA axis habituation to repeated stress, indices of HPA axis habituation were computed. Specifically, delta scores were computed for the initial and repeated exposure using peak values relative to baseline (i.e., peak cortisol values at 1, 10, or 20 min post-TSST minus cortisol values at pre-TSST baseline [-1]), and then the TSST 2 response was subtracted from TSST 1 to obtain habituation indices (see Breines et al., 2014). We chose to use delta scores for this investigation because the purpose was to assess habituation of the responses to the acute stressor from day 1 to day 2 (i.e., the magnitude of difference that takes place between the days when considering the peak response, compared to baseline for each day) in light of emotion regulation strategy use. Although other analytical strategies are valuable given different research questions (e.g., an ANOVA would provide information about differences in trajectory, and area under the curve with respect to increase would allow estimations of total cortisol output), using delta scores to compute habituation indices was the most appropriate statistical approach for the present research.

Using the habituation indices as the dependent variables for all analyses, separate hierarchical linear regression models were conducted, regressing HPA axis habituation indices onto ER strategies. Because sex and BMI have been repeatedly shown to affect cortisol stress responses (Kudielka and Kirschbaum, 2005; McInnis et al., 2014), sex and BMI were included as covariates in all analyses. When examining state ER strategies, the respective trait ER strategy was also included as a covariate. Because only a portion of the final sample (28.5%) was fitted with indwelling catheters for blood draws due to a protocol variation and one or more TSST committee members changed for 78.6% of participants, we examined whether the blood draw protocol variation or change in committee member(s) should be included as covariates in analyses. Neither the protocol variation nor TSST committee member changes significantly altered any results; thus, they were not included as covariates in the reported analyses. Results were considered significant at $p < .05$.

3. Results

3.1. Preliminary analyses

Descriptive statistics and zero-order correlations are shown in

Table 1
Descriptive characteristics of sample population including zero-order correlations.

Variable	M ± SD/n (%)	1	2	3	4	5	6	7	8	9
1. Age (years)	24.8 ± 6.0	–								
2. Sex (female)	51 (60.7%)	–0.03	–							
3. BMI (kg/m ²)	22.23 ± 2.4	0.30**	–0.14	–						
4. Trait reappraisal	27.05 ± 6.5	–0.03	0.00	–0.05	–					
5. State reappraisal ^a	28.02 ± 6.9	–0.06	0.08	–0.18	0.42**	–				
6. Trait suppression	14.29 ± 4.5	0.01	–0.30**	0.29**	–0.12	0.00	–			
7. State suppression ^a	14.48 ± 3.9	–0.12	–0.19	0.26*	–0.05	0.05	0.47**	–		
8. Day 1 cortisol response (nmol/L)	7.58 ± 7.8	0.22*	–0.35**	0.34**	0.09	–0.12	0.12	0.15	–	
9. Day 2 cortisol response (nmol/L)	3.99 ± 6.0	.28*	–0.29**	0.31**	–0.05	–0.03	0.15	0.29*	0.69**	–
10. Cortisol habituation (nmol/L)	3.59 ± 4.8	.07	–0.36**	0.16	0.27*	–0.03	.01	–0.03	.58**	0.02

Note. N = 84 for full sample. ^a N = 60 participants. * p < .05, ** p < .01. BMI = Body Mass Index; kg/m² = kilograms per meters squared; nmol/L = nanomoles per liter. Delta scores for cortisol responses on days 1 and 2 were computed using peak values relative to baseline.

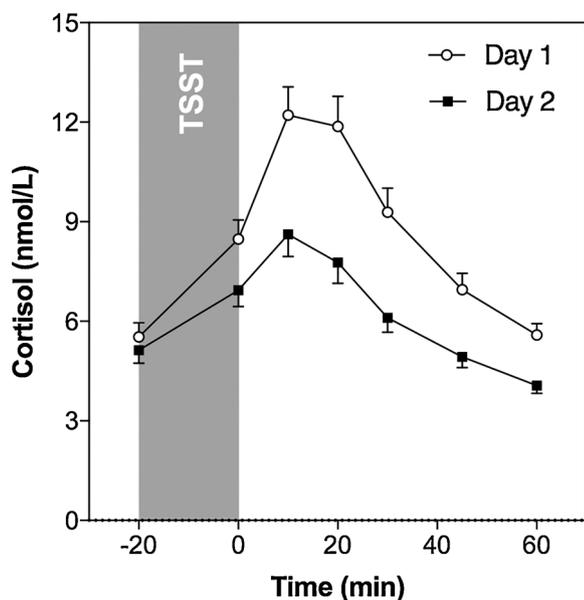


Fig. 1. HPA axis responses to the Trier Social Stress Test (TSST). Graph show means and standard errors of the mean (SEM) of salivary concentrations (raw values) at baseline and at +1, +10, +20, +30, +45, and +60 min after TSST.

Table 1. Repeated measures ANOVA showed a significant main effect of time on cortisol for both study days ($F_{1,96,158.45} = 91.097, p < .001$, partial $\eta^2 = 0.529$). A significant day x time interaction also occurred ($F_{2,91,235.80} = 17.784, p < .001$, partial $\eta^2 = 0.180$), suggesting that cortisol response habituation occurred from day 1 to day 2. Fig. 1 displays mean cortisol values over time on days 1 and 2.

Older participants were more likely to have a higher BMI and larger cortisol responses on days 1 and 2 using delta scores. Men were more likely to report a general tendency to use suppression. They also had larger cortisol responses on days 1 and 2 and greater habituation. People with higher BMIs reported more suppression in general as well as specifically in response to the TSST on day 1. BMI was also positively associated with cortisol responses on days 1 and 2. Trait emotion regulation (ER) strategies and their respective state ER strategies were moderately correlated, indicating that although the variables share some variance in these data, they appear to represent distinct constructs. The correlations between ER strategies and cortisol responses on days 1 and 2 using delta scores were nonsignificant, except for state suppression; participants that reported using suppression in response to the TSST on day 1 experienced a higher cortisol response to the TSST on day 2. We followed up with the zero-order correlation for state suppression and day 2 cortisol response using a hierarchical multiple regression analysis controlling for sex and BMI; state suppression was not

significantly associated with day 2 cortisol response when covariates were included ($p = .149$).

3.2. ER strategies and HPA axis habituation

Trait reappraisal was significantly associated with greater HPA axis habituation to a repeated acute stressor ($\beta = 0.279, \Delta R^2 = 0.078, p = .006$; see Fig. 2). The association between trait suppression and HPA axis habituation was not significant ($\beta = -0.149, \Delta R^2 = 0.012, p = .182$; see Fig. 3). Neither state reappraisal ($\beta = -0.153, \Delta R^2 = 0.019, p = .263$) nor state suppression ($\beta = -0.087, \Delta R^2 = 0.006, p = .557$) were significantly associated with HPA axis habituation to stress (see Figs. 4 and 5).

4. Discussion

Habituation of the HPA axis is considered a common and adaptive response to repeated stress (e.g., Kirschbaum et al., 1995; McEwen, 1998; Thoma et al., 2017), however our knowledge of predictors of habituation is limited. The current study sought to determine first, whether trait-use of two emotion regulation (ER) strategies, cognitive reappraisal and expressive suppression, were related to HPA axis habituation; and second, whether stressor-specific use of ER strategies influences habituation over and above trait-use. To assess habituation, participants were exposed to an experimental stress paradigm, the Trier Social Stress Test (TSST), on two consecutive days. HPA axis stress

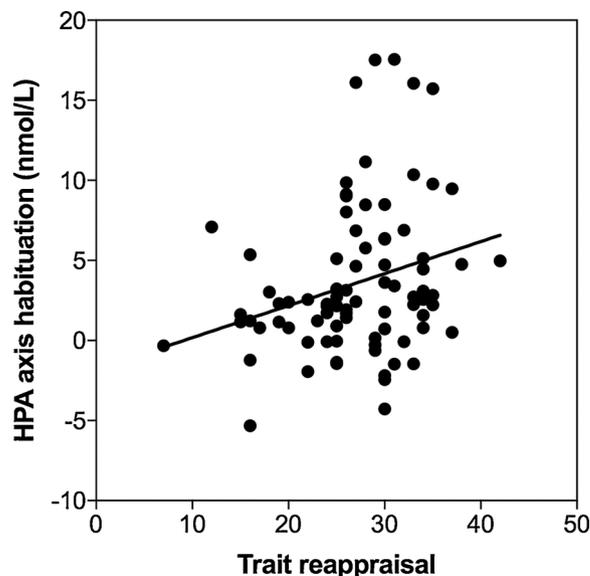


Fig. 2. Scatterplot displaying the association between trait reappraisal and HPA axis habituation using standardized regression weights.

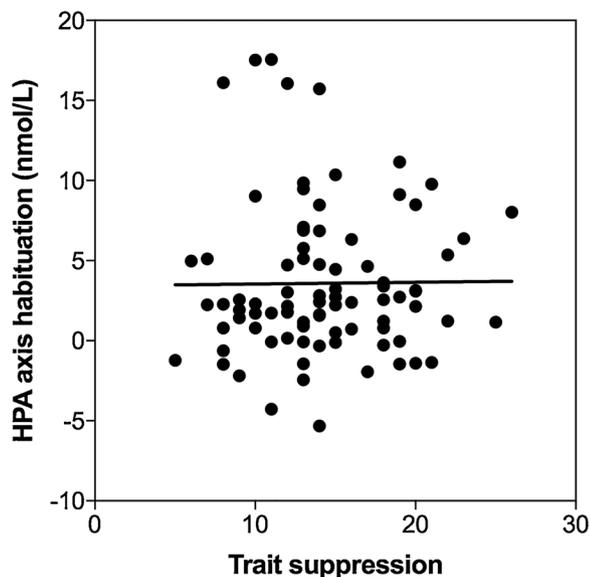


Fig. 3. Scatterplot displaying the association between trait suppression and HPA axis habituation using standardized regression weights.

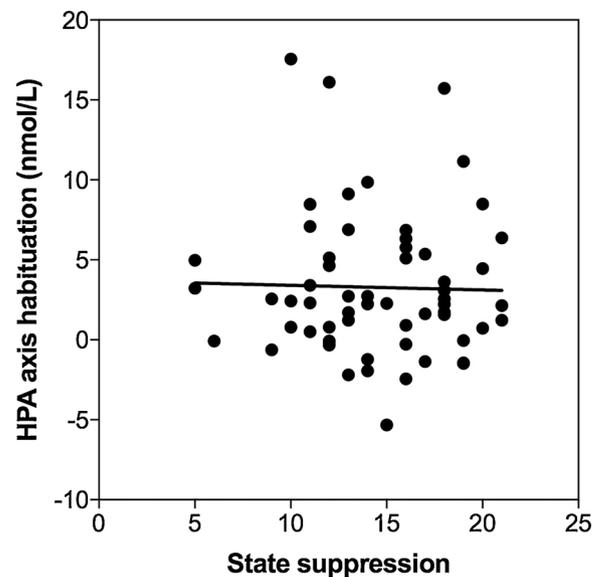


Fig. 5. Scatterplot displaying the association between state suppression and HPA axis habituation using standardized regression weights.

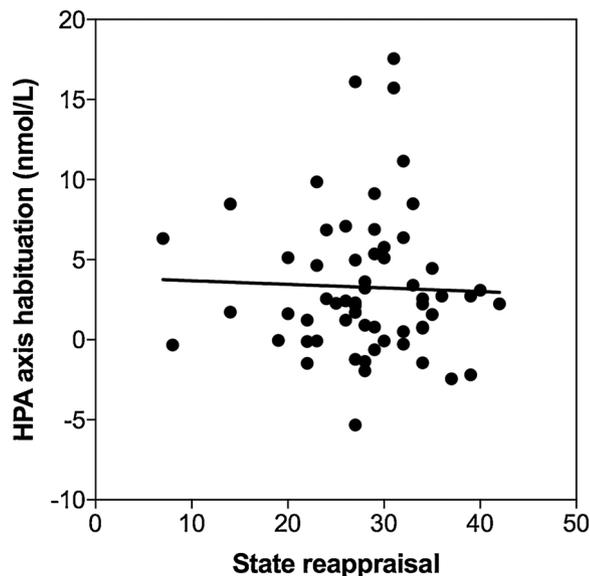


Fig. 4. Scatterplot displaying the association between state reappraisal and HPA axis habituation using standardized regression weights.

responses were measured using salivary cortisol repeatedly before and after stress on both days.

Trait reappraisal predicted HPA axis habituation to stress as indicated by a lower cortisol response on day 2 relative to day 1; this finding suggests that habitual reappraisal impacted how the HPA axis responded to repeated stressors. Specifically, the tendency to reappraise accounted for roughly 8% of the variance in HPA axis habituation. Previous studies have shown that trait and experimentally-manipulated reappraisal are related to heightened HPA axis reactivity to novel acute stressors (e.g., Lam et al., 2009; Denson et al., 2014); however, it is also linked with adaptive outcomes following stressful life events and long-term reductions in negative affect (Moore et al., 2008; Troy et al., 2010; Denson et al., 2014).

One explanation for the disconnect between acute HPA axis responses and long-term health outcomes is that although reappraisal is effortful in the short term (Shafir et al., 2015), habitual use may increase the ability of the HPA axis to adapt to stressors in the long term (Roos et al., 2018). The current findings support that idea by

demonstrating that self-reported tendency to reappraise may promote HPA axis habituation to repeated stress. That is, frequent practice of reappraisal over an extended period may increase the HPA axis' ability to adapt to a repeated stressor. Further research is needed to determine whether the tendency to reappraise results in greater psychological resilience and better health through its capacity to increase stress system adaptation to repeated stressors.

Interestingly, it appears that although the general tendency to reappraise is related to stronger HPA axis habituation, reappraisal specific to the first exposure of the TSST on day 1 (i.e., state reappraisal) was not associated with habituation. These results suggest that state reappraisal may not be impactful enough on the HPA axis to promote or inhibit habituation. Our findings warrant further research which could shed light on why state reappraisal does not influence HPA axis habituation.

Alternatively, since the state emotion regulation measure was given during the second visit, it is possible that the retrospective nature of the assessment or its timing yielded an inaccurate estimation of emotion regulation strategy use for the period in between days 1 and 2. We also do not know the frequency with which the strategies were used (i.e., were strategies used throughout the period between the two visits, intermittently during recovery, or only shortly afterward the TSST on day 1?). To minimize recall bias and ensure accurate collection of when strategy use occurred, perhaps measuring state emotion regulation during recovery and/or using ecological momentary assessment following the first visit, instead of retrospective report, would yield different results. Additionally, it is possible that results could be more salient if reappraisal is experimentally manipulated. Further research, including direct and conceptual replication, is necessary to determine whether the results found here are consistent.

Neither trait nor state suppression were significantly associated with HPA axis habituation. These results were surprising, since findings from one study have indicated an association between trait suppression and heightened HPA axis reactivity (Lam et al., 2009); we expected that either trait or state suppression would produce some effect on habituation, but it appears that suppression does not influence HPA axis reactivity to repeated stressors over time. Other research has provided evidence for a link between expressive suppression and heightened sympathetic nervous system activation to acute stress (Gross and Levenson, 1993, 1997) and has suggested that suppression may contribute to cardiovascular disease (Mauss and Gross, 2004). Although the results presented here are not comprehensive, it is possible that

suppression's long-term maladaptive effects on health occur primarily through a dysregulated autonomic nervous system (ANS). Although previous research has demonstrated non-habituation of the ANS when assessing plasma epinephrine and norepinephrine (Schommer et al., 2013), it may be possible for changes in the cardiovascular system to be discernible if cardiovascular biomarkers that are more sensitive to moment-to-moment variability in (e.g., heart rate variability) are used. Further, cardiovascular system dysregulation may occur through other avenues (e.g., exaggerated responses to stress exposure). Future research replicating these results, with the inclusion of cardiovascular responses that are sensitive to moment-to-moment variability (e.g., heart rate variability, electrodermal activity) and examination of additional stress reactivity patterns, could help to elucidate the mechanism(s) by which suppression produces maladaptive effects on health over time.

This study was strengthened by its inclusion of young adults without psychopathologies or major medical conditions requiring medication, showing that these effects can be seen in healthy adults. Nonetheless, the well-controlled nature of this study also gives rise to some practical limitations that should be considered; specifically, it is unclear whether results here would generalize to a less psychologically or physically healthy population (e.g., clinical populations, individuals with chronic health issues). Additionally, although it is theorized that nonhabituation of the HPA axis to stress is maladaptive for health because it disallows for proper physiological adaptation to stress (e.g., Kirschbaum et al., 1995; McEwen, 1998; Schommer et al., 2003), longitudinal studies are needed to assess how reappraisal's effect on habituation can influence health and well-being in the long term.

Taken together, this investigation provides preliminary data suggesting that trait reappraisal, but not suppression, may influence HPA axis habituation to stress. Further, state reappraisal and suppression did not appear to be related to HPA axis habituation when accounting for self-reported tendency to engage in each emotion regulation strategy. These findings contribute to the understanding of how emotion regulation strategies may lead to stress-related health outcomes. Understanding how the use of different emotion regulation strategies affect health in the face of repeated acute stress could also help guide future studies. For example, future research should investigate whether modifying the use of emotion regulation strategies could result in more adaptive stress responses and overall health. Future studies should also assess whether HPA axis habituation that is aided by trait reappraisal can contribute to psychological adaptation to stress as well. Ultimately, this area of research may help direct stress reduction efforts, such as interventions focusing on emotion regulation strategies.

Conflicts of interest

Authors have no conflicts of interest to disclose.

Acknowledgements

This research was partially supported by the 2017 Health and Behavior International Collaboration Award from the Society for Health Psychology (LGR). In addition, LGR received the Health Psychology Summer Fellowship from the UNC Charlotte Health Psychology PhD Program that supported her time to conduct analyses and prepare this manuscript. The authors additionally thank the study staff and participants.

References

Augustine, A.A., Hemenover, S.H., 2009. On the relative effectiveness of affect regulation strategies: a meta-analysis. *Cogn. Emot.* 23, 1181–1220. <https://doi.org/10.1080/02699930802396556>.

Breines, J.G., Thoma, M.V., Gianferante, D., Hanlin, L., Chen, X., Rohleder, N., 2014. Self-compassion as a predictor of interleukin-6 response to acute psychosocial stress. *Brain Behav. Immun.* 37, 109–114. <https://doi.org/10.1016/j.bbi.2013.11.006>.

Cohen, S., Janicki-Deverts, D., Doyle, W.J., Miller, G.E., Frank, E., Rabin, B.S., Turner, R.B., 2012. Chronic stress, glucocorticoid receptor resistance, inflammation, and disease risk. *Proc. Natl. Acad. Sci. U. S. A.* 109, 5995–5999. <https://doi.org/10.1073/pnas.1118355109>.

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

Dickerson, S.S., Kemeny, M.E., 2004. Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research. *Psychol. Bull.* 130, 355–391. <https://doi.org/10.1037/0033-2909.130.3.355>.

Garnefski, N., Kraaij, V., Spinhoven, P., 2001. Negative life events, cognitive emotion regulation and emotional problems. *Pers. Individ. Dif.* 30, 1311–1327. [https://doi.org/10.1016/s0191-8869\(00\)00113-6](https://doi.org/10.1016/s0191-8869(00)00113-6).

Gianferante, D., Thoma, M.V., Hanlin, L., Chen, X., Breines, J., Zoccola, P.M., Rohleder, N., 2014. Post-stress rumination predicts HPA axis responses to repeated acute stress. *Psychoneuroendocrinology* 49, 244–252. <https://doi.org/10.1016/j.psyneuen.2014.07.021>.

Glaser, R., Kiecolt-Glaser, J.K., 2005. Stress-induced immune dysfunction: implications for health. *Nat. Rev. Immunol.* 5, 243–251. <https://doi.org/10.1038/nri1571>.

Graham, J.E., Christian, L.M., Kiecolt-Glaser, J.K., 2006. Stress, age, and immune function: toward a lifespan approach. *Behav. Med.* 29, 389–400. <https://doi.org/10.1007/s10865-006-9057-4>.

Gross, J.J., 1998. Antecedent- and response-focused emotion regulation: divergent consequences for experience, expression, and physiology. *J. Pers. Soc. Psychol.* 74, 224–237.

Gross, J.J., John, O.P., 2003. Individual differences in two emotion regulation processes: implications for affect, relationships, and well-being. *J. Pers. Soc. Psychol.* 85, 348–362.

Gross, J.J., Levenson, R.W., 1993. Emotion suppression: physiology, self-report, and expressive behavior. *J. Pers. Soc. Psychol.* 64, 970–986.

Gross, J.J., Levenson, R.W., 1997. Hiding feelings: the acute effects of inhibiting negative and positive emotion. *J. Abnorm. Psychol.* 106, 95–103.

Hautzinger, M., Bailer, M., Hofmeister, D., Keller, F., 2012. Allgemeine Depressionsskala (ADS). *Psychiatr. Prax.* 39, 302–304.

Janson, J., Rohleder, N., 2017. Distraction coping predicts better cortisol recovery after acute psychosocial stress. *Biol. Psychol.* 128, 117–124. <https://doi.org/10.1016/j.biopsycho.2017.07.014>.

John, O.P., Gross, J.J., 2004. Healthy and unhealthy emotion regulation: personality processes, individual differences, and life span development. *J. Personal.* 72, 1301–1334. <https://doi.org/10.1111/j.1467-6494.2004.00298.x>.

Kashdan, T.B., Steger, M.F., 2006. Expanding the topography of social anxiety: an experience sampling assessment of positive emotions and events, and emotion suppression. *Psychol. Sci.* 17, 120–128. <https://doi.org/10.1111/j.1467-9280.2006.01674.x>.

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

Kirschbaum, C., Prussner, J.C., Stone, A.A., Federenko, I., Gaab, J., Lintz, D., Schommer, N., Hellhammer, D.H., 1995. Persistent high cortisol responses to repeated psychological stress in a subpopulation of healthy men. *Psychosom. Med.* 57, 468–474.

Kudielka, B.M., Kirschbaum, C., 2005. Sex differences in HPA axis responses to stress: a review. *Biol. Psychol.* 69, 113–132. <https://doi.org/10.1016/j.biopsycho.2004.11.009>.

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

Lazarus, R.S., 1966. *Psychological Stress and the Coping Process*. McGraw-Hill, New York, NY.

Mauss, I.B., Gross, J.J., 2004. Emotion suppression and cardiovascular disease: Is hiding your feelings bad for your heart? In: Temoshok, L.R., Vingerhoets, A., Nyklicek, I. (Eds.), *The Expression of Emotion and Health*. Brunner-Routledge, London, pp. 62–81.

McEwen, B.S., 1998. Protective and damaging effects of stress mediators. *N. Engl. J. Med.* 338, 171–179. <https://doi.org/10.1056/NEJM199801153380307>.

McInnis, C.M., Thoma, M.V., Gianferante, D., Hanlin, L., Chen, X., Breines, J.G., Hong, S., Rohleder, N., 2014. Measures of adiposity predict interleukin-6 responses to repeated psychosocial stress. *Brain Behav. Immun.* 42, 33–40. <https://doi.org/10.1016/j.bbi.2014.07.018>.

Meyer, T., Smeets, T., Giesbrecht, T., Merckelbach, H., 2012. The efficiency of reappraisal and expressive suppression on everyday affective experiences. *Psychiat. Res.* 200, 964–969. <https://doi.org/10.1016/j.psychres.2012.05.034>.

Moore, S.A., Zoellner, L.A., Mollenholt, N., 2008. Are expressive suppression and cognitive reappraisal associated with stress-related symptoms? *Behav. Res. Ther.* 46, 993–1000. <https://doi.org/10.1016/j.brat.2008.05.001>.

Radloff, L.S., 1977. The CES-D Scale: a self-report depression scale for research in the general population. *Appl. Psychol. Meas.* 1, 385–401. <https://doi.org/10.1177/014662167700100306>.

Roos, L.G., Levens, S.M., Bennett, J.M., 2018. Stressful life events, relationship stressors, and cortisol reactivity: the moderating role of suppression. *Psychoneuroendocrinology* 89, 69–77. <https://doi.org/10.1016/j.psyneuen.2017.12.026>.

Sagui, S.J., Levens, S.M., 2016. Cognitive reappraisal ability buffers against the indirect effects of perceived stress reactivity on Type 2 diabetes. *Health Psychol.* 35, 1154–1158. <https://doi.org/10.1037/hea0000359>.

Sapolsky, R.M., Romero, L.M., Munck, A.U., 2000. How do glucocorticoids influence

- stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. *Endocr. Rev.* 21, 55–89. <https://doi.org/10.1210/edrv.21.1.0389>.
- Schommer, N.C., Hellhammer, D.H., Kirschbaum, C., 2003. Dissociation between reactivity of the hypothalamus-pituitary-adrenal axis and the sympathetic-adrenal-medullary system to repeated psychosocial stress. *Psychosom. Med.* 65, 450–460.
- Shafir, R., Schwartz, N., Blechert, J., Sheppes, G., 2015. Emotional intensity influences pre-implementation and implementation of distraction and reappraisal. *Soc. Cogn. Affect. Neurosci.* 10, 1329–1337. <https://doi.org/10.1093/scan/nsv022>.
- Tamashiro, K.L., Sakai, R.R., Shively, C.A., Karatsoreos, I.N., Reagan, L.P., 2011. Chronic stress, metabolism, and metabolic syndrome. *Stress* 14, 468–474. <https://doi.org/10.3109/10253890.2011.606341>.
- Thoma, M.V., Gianferante, D., Hanlin, L., Fiskdal, A., Xuejie, C., Rohleder, N., 2017. Stronger hypothalamus-pituitary-adrenal axis habituation predicts lesser sensitization of inflammatory response to repeated acute stress exposures in healthy young adults. *Brain Behav. Immun.* 61, 228–235. <https://doi.org/10.1016/j.bbi.2016.11.030>.
- Troy, A.S., Wilhelm, F.H., Shallcross, A.J., Mauss, I.B., 2010. Seeing the silver lining: cognitive reappraisal ability moderates the relationship between stress and depressive symptoms. *Emotion* 10, 783–795. <https://doi.org/10.1037/a0020262>.
- Wüst, S., Federenko, I.S., van Rossum, E.F., Koper, J.W., Hellhammer, D.H., 2005. Habituation of cortisol responses to repeated psychosocial stress-further characterization and impact of genetic factors. *Psychoneuroendocrinology* 30, 199–211. <https://doi.org/10.1016/j.psyneuen.2004.07.002>.