



# The impact of emotion regulation on cardiovascular, neuroendocrine and psychological stress responses



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## ABSTRACT

Emotion regulation (ER) is vital for healthy adaptation and influences how individuals respond to and recover from stress. We investigated whether ER improves cardiovascular, neuroendocrine and psychological stress responses, while taking into account the moderating role of habitual ER tendencies. Eighty-six women applied either cognitive reappraisal or expressive suppression (vs. control) while undergoing a stressor. Reappraisal decreased heart rate variability (HRV) during stress, but also initiated a stronger post-stress HRV-recovery relative to suppression. This reappraisal-induced cardiac-vagal-flexibility was particularly observed in habitual reappraisers. Furthermore, the reappraisal group reported enhanced positive affect, whereas the suppression group experienced more unpleasantness and expressed higher cortisol levels than controls. Heightened cortisol reactivity was also found in the reappraisal group, but only for individuals scoring low or mean on trait reappraisal. These results provide preliminary evidence that reappraisal fosters psychophysiological adaptation in response to stress, but also suggest that ER-strategy-efficacy critically depends on its habitual use.

## 1. Introduction

Stress is often associated with negative emotions and can become a risk factor for maladjustment and psychopathology if dealt with in an unhealthy manner (Grant et al., 2003; Kopp & Réthelyi, 2004). However, most people adapt remarkably well, maintaining normal psychological functioning even when exposed to high levels of stress (Bonanno, 2004, 2005). One factor that may have the potential to alter how individuals respond to and recover from stress is the ability to regulate emotions. Emotion regulation refers to the use of behavioral and cognitive strategies in order to change the nature, intensity, duration or expression of an emotion (Gross & Thompson, 2007). Cognitive reappraisal for instance involves reframing an emotional event as to alter its emotional impact, whereas expressive suppression aims at inhibiting only the outward expression of an emotion (Gross, 2015; Ochsner & Gross, 2005). Accumulating evidence suggests that the habitual use of cognitive reappraisal is related to increased positive affect, well-being and better interpersonal functioning. By contrast, expressive suppression is rather associated with greater experience of negative emotions and depressive symptoms and thus generally considered maladaptive when used frequently (Gross & John, 2003; John & Gross, 2004). Yet, despite its vital role for understanding successful adaptation and resilience to stress, only a few studies focused

specifically on the impact of emotion regulation on psychophysiological stress responding.

The human stress response is characterized by the consecutive activation of the sympathetic nervous system (SNS), leading to the secretion of (nor)adrenaline and a rapid increase in heart rate, blood pressure and respiration frequency and the somewhat slower hypothalamus-pituitary-adrenocortical (HPA) axis, resulting in the release of glucocorticoids (GCs, in humans cortisol; Joëls & Baram, 2009). It has been recently shown that cognitively reappraising stress-induced arousal during a psychosocial stress task could promote adaptive cardiovascular responses, as indicated by increased cardiac efficiency but reduced vasoconstriction (Jamieson, Nock, & Mendes, 2012). Consistently, cognitive reappraisal but not expressive suppression increased heart rate variability (HRV) to an anger-provoking video when compared to a control condition (Denson, Grisham, & Moulds, 2011). HRV, an index of consecutive changes in heartbeats (Shaffer & Ginsberg, 2017), is thought to reflect the flexible regulation of autonomic arousal in line with situational demands (Appelhans & Luecken, 2006) and in recent years has been considered a promising biomarker of adaptive emotion regulation (Balzarotti, Biassoni, Colombo, & Ciceri, 2017). As such, higher HRV at rest has been associated with better down-regulation of negative affect, use of adaptive emotion regulatory strategies, and more flexible emotional responding. In terms of phasic (i.e.

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event-related) HRV changes, research has consistently found decreased HRV in response to stress (also referred to as vagal withdrawal), while HRV increases appeared to reflect recovery from stress (for a comprehensive review see, Balzarotti et al., 2017). Vagal withdrawal from rest to acute stress states and fast subsequent vagal recovery reflects reactivity of the parasympathetic nervous system, enabling the organism to adaptively respond to stressors and may thus serve to quantify the ability for self-regulation (Friedman, 2007; Movius & Allen, 2005; Porges, 1992a, 1992b). Notably, emotion regulation has been shown to promote this phasic HRV regulation, especially when recovering from a stressor (Key, Campbell, Bacon, & Gerin, 2008; Neumann, Waldstein, Sellers, Thayer, & Sorkin, 2004). This corroborates with studies demonstrating that using reappraisal not only improved cardiovascular responses during a stressful task (e.g. by enhancing the amount of blood ejected from the heart and reducing vascular resistance (Jamieson et al., 2012), but also facilitated a quicker recovery to baseline when acute stress subsided (Jamieson, Nock, & Mendes, 2013; Liu, Vickers, Reed, & Hadad, 2017). Importantly, habitual as well as instructed reappraisals also showed improved cognitive performance, higher appraisals of coping resources and less experience of negative emotions in response to stress when compared to controls (Beltzer, Nock, Peters, & Jamieson, 2014; Jamieson, Mendes, Blackstock, & Schmader, 2010; Jamieson et al., 2012; Mauss, Cook, Cheng, & Gross, 2007). Hence, reappraisal appears to make individuals perceiving the stressor as a challenge rather than a threat (Blascovich & Tomaka, 1996). Together, these findings indicate that a brief laboratory-based reappraisal manipulation can lead to enhanced vagal withdrawal (and concurrent sympathetic activation) as well as increased feelings of self-efficacy and control over a stressor, which in turn may prepare for effective coping and a rapid return to homeostasis after stress offset (Jamieson, Crum, Goyer, Marotta, & Akinola, 2018). By contrast, instructed suppression rather increased subjective anxiety and negative affect during challenging or aversive situations in both healthy individuals (Hofmann, Heering, Sawyer, & Asnaani, 2009) as well as in patients suffering from anxiety and mood disorders (Campbell-Sills, Barlow, Brown, & Hofmann, 2006). Given that suppression aims to reduce or hide the behavioral and expressive aspects of emotional responses, it probably conveys the belief that every stressor constitutes a threat per se and thus any response to it should be avoided. These repeated efforts to manage any upcoming response deplete cognitive resources and compromises psychosocial functioning, as the suppressor fails to take up information needed to respond appropriately to the stressor (Cutuli, 2014). Given the link between expressive suppression, stress-related symptoms, anxiety and depression (Moore, Zoellner, & Mollenholt, 2008), the question therefore arises whether this strategy indeed contributes to maladaptive stress response patterns that ultimately might transfer into a vulnerability or causal factor for developing stress-related psychopathologies.

With respect to neuroendocrine measures, correlational work has mostly revealed that trait forms of both reappraisal and suppression are associated with heightened HPA-axis reactivity to an acute psychosocial stressor (Lam, Dickerson, Zoccola, & Zaldivar, 2009; Raymond, Marin, Juster, & Lupien, 2019; but see, Carlson, Dikecligil, Greenberg, & Mujica-Parodi, 2012). Yet, some studies also failed to provide clear evidence for an association between trait reappraisal and cortisol reactivity (Lewis, Yoon, & Joormann, 2018; Roos, Levens, & Bennett, 2018). Still, increased stress-induced cortisol levels were also observed in participants instructed to cognitively reappraise a social evaluative speech task and a physical pain stressor in a detached and objective manner relative to participants without a regulation instruction (Denson, Creswell, Terides, & Blundell, 2014). It is thus reasonable, that deliberately regulating negative emotions under stressful conditions indeed mobilizes active coping resources, but also requires more cognitive effort and attentional control, which in turn could lead to stronger neuroendocrine and cardiovascular stress responses. Consequently, the activation of the HPA-axis should be most pronounced

when a regulatory strategy is employed for the first time or if the instructed technique does not match the person's habitual tendency to regulate emotions in daily life (match-mismatch hypothesis). By contrast, the more a strategy is used the more automatic and less effortful it may become, thus resulting in less neuroendocrine reactivity. This explanation is consistent with an emerging set of studies demonstrating the effectiveness of long-term cognitive-behavioral stress management trainings using reappraisal techniques in reducing neuroendocrine and psychological responses to acute stress (Gaab et al., 2003; Gaab, Sonderegger, Scherrer, & Ehlert, 2006; Hammerfeld et al., 2006). Furthermore, the match-mismatch hypothesis would assume that individual differences in the habitual use of emotion regulation strategies could determine how successful an instructed strategy would be applied in a given situation. However, to date no study has explored whether an experimental manipulation and trait forms of emotion regulation interact to affect psychological or physiological stress reactivity. Likewise, it is still unclear whether emotion regulation interventions and different strategies in particular are equally effective across different psychological and physiological measures of stress. A recent meta-analysis for instance revealed that reappraisal interventions were particularly effective in reducing subjective stress responses, however without having a significant effect on physiological stress responsivity (Liu, Ein, Gervasio, & Vickers, 2019). Yet, the studies included in this analysis did not explore both trait and experimentally induced reappraisal.

To address these issues, we investigated the impact of cognitive reappraisal and expressive suppression on psychological, cardiovascular and neuroendocrine responses to a laboratory stressor, while taking into account the moderating role of the participants' habitual tendencies to use either of these two strategies in daily life. Based on previous work showing positive effects of trait and state reappraisal on psychological stress responses (Beltzer et al., 2014; Jamieson et al., 2012; Mauss et al., 2007), we expected cognitive reappraisal but not expressive suppression to improve the perceived self-concept of own abilities and anticipatory control expectancies about the stressor, to reduce subjective feelings of stressfulness and unpleasantness during the stressor and to generally promote positive affect but reduce negative affect. For cardiovascular responses, we further hypothesized that reappraisal would lead to a phasic decrease in HRV during the stress task, but also a stronger HRV recovery in the post-stressor phase (for a review, see Jamieson et al., 2018). According to the match-mismatch hypothesis, these effects should be especially strong in participants habitually using cognitive reappraisal. Given the mixed evidence for the effects of emotion regulation on HPA axis reactivity (Carlson et al., 2012; Denson et al., 2014; Gaab et al., 2003; Gaab et al., 2006; Lam et al., 2009; Raymond et al., 2019), we expected both active reappraisal and suppression to further increase stress-induced cortisol responses relative to a no-regulation control condition, especially if participants are assigned to a regulation strategy that would not match their habitual tendency to use this particular emotion regulation strategy in daily life.

## 2. Methods

### 2.1. Participants and experimental design

An intended sample size of 25 participants per group (in total 75 participants) was set in advance based on sample sizes from previous studies investigating the impact of emotion regulation on psychophysiological stress responses (cf. Denson et al., 2014; Jamieson et al., 2012).

Eighty-six healthy female students were recruited at the Ruhr University Bochum for study participation. Sex differences in HPA stress responsivity (Kudielka & Kirschbaum, 2005) as well as in emotional processing has been frequently reported, with women typically displaying stronger behavioral, physiological and neural reactivity to

emotional stimuli (Bradley, Codispoti, Sabatinelli, & Lang, 2001; Canli, Desmond, Zhao, & Gabrieli, 2002; Lithari et al., 2010). Moreover, men and women tend to use different emotion regulation strategies and women generally report to engage more in emotion regulation processes (Nolen-Hoeksema, 2012; Tamres, Janicki, & Helgeson, 2002). Thus in order to ensure emotion regulatory engagement and to reduce the between-subjects variance regarding emotional reactivity and emotion regulation, we decided to include only female participants in the present study. Exclusion criteria were checked beforehand in a standardized telephone screening and comprised age < 18 or > 40 years, a body mass index (BMI) < 18 or > 27 kg/m<sup>2</sup>, chronic or acute illnesses, history of psychiatric or neurological treatment, drug use including smoking, regular or excessive alcohol consumption, regular medication (including particularly psychoactive drugs, hormonal supplements or any medication affecting thyroid functionality), working night shifts, and hormonal contraceptive usage. All participants were fluent in German, not familiar with the current stress protocol and tested only outside their menses (Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999); menstrual cycle phase was assessed via self-report.

A between-subjects design with the factor group (cognitive reappraisal vs. expressive suppression vs. control) was used in order to examine the effects of emotion regulation on cardiovascular, neuroendocrine and psychological stress responding, with participants randomly assigned to the three experimental groups.

## 2.2. Procedure

Experimental sessions were conducted between 1 pm and 6 pm to control for diurnal variations in endogenous cortisol concentrations (Horrocks et al., 1990; Joëls & Baram, 2009). Furthermore, participants were advised to refrain from physical exercise and consumption of food and drinks except water within one hour prior to testing. After signing informed consent, participants filled out questionnaires on demographic variables and habitual emotion regulation. They were then equipped with the HRV measurement device and asked to relax and sit still for 5 minutes baseline HRV recording. Following the rest period, participants received either detailed information about the respective emotion regulation strategies or a neutral text (described in detail below) and then completed the stress protocol while their cardiovascular responses were recorded. They remained in the laboratory for another 30 minutes to allow for additional saliva sampling and cardiovascular measurements as proof of the stress and emotion regulation manipulation. Participants were either paid an allowance of 15€ or receive course credit for their participation. All procedures were in accordance to the Declaration of Helsinki and approved by the ethics committee of the Faculty of Psychology at the Ruhr University Bochum. A schematic illustration of the experimental procedure is depicted in Fig. 1.

## 2.3. Emotion regulation manipulation

Prior to the instructions for the stress procedure, all participants received an information text and an accompanying worksheet. For the cognitive reappraisal and the expressive suppression group, this served to familiarize participants with the general concept of emotion

regulation and the respective emotion regulation strategy. The texts and worksheets were based on material provided by the emotion regulation skills training developed by Barnow, Reinelt, and Sauer (2016) and scripted instructions from previous emotion regulation studies (Denson et al., 2014; Jamieson et al., 2012).

More specifically, the cognitive reappraisal text informed participants about the possibility to think about the positive aspects of a challenging or stressful situation, such as lessons one may learn or taking the task as an opportunity to develop skills and improve abilities, which may aid performance in similar tasks in the future. Furthermore, participants were told that physiological arousal during stressful situations is not harmful, but instead represent a functional and adaptive response of our body that helps to successfully address stressors. In the expressive suppression group, participants were informed about the benefits of suppressing the outward expression of emotions by inhibiting overt facial expressions or bodily responses. For instance, they were told that an upright posture and a straight but relaxed face ('poker face') would convey a professional and self-confident impression and would improve task performance. An oral exam was chosen as an exemplary situation in both texts to enhance comprehensiveness of how to apply the emotion regulation strategies. After reading the information text, participants were additionally asked to summarize the respective strategy in their own words, to describe when and how they could apply the strategy to a future situation and to formulate a personal mnemonic sentence. These tasks served to ensure correct understanding of the strategies. In case participants did not come up with an own mnemonic sentence, a short summary and three exemplary sentences were then provided to all participants of the two experimental groups.

The control group received an information text about the human senses that was similar in length and complexity but completely unrelated to the upcoming stress procedure, which they were also required to summarize in their own words. The information texts, worksheets, summaries and instructions for all emotion regulation, stress and control manipulations are available via the Open Science Framework (<https://osf.io/tntbz/>).

## 2.4. Stress induction

Psychosocial stress was induced using the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993), involving a 5 min preparation period, a 5 min free speech about personal characteristics in a mock job interview in front of a neutral and reserved committee (one male, one female), and a 5 min mental arithmetic task while being videotaped. The TSST is a validated and widely used standardized laboratory stressor reliably inducing psychological, cardiac and neuroendocrine stress responses (Dickerson & Kemeny, 2004).

In addition to the instructions for the TSST, participants in the cognitive reappraisal and expressive suppression group were asked to apply the respective emotion regulation strategy to the upcoming challenging situation. To facilitate strategy implementation, they were again provided with the short summary and encouraged to reactivate and internalize their mnemonic sentence during the TSST preparation period. The control group did not receive any additional instructions.

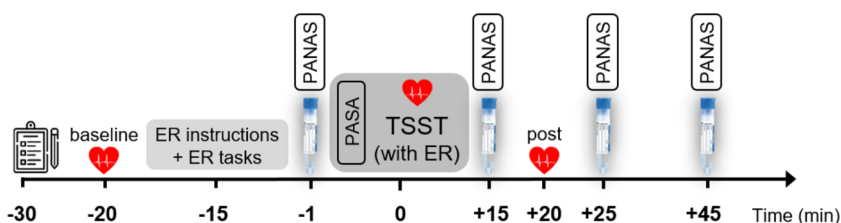


Fig. 1. Experimental procedure. Heart rate variability was measured at baseline (5 min), during the TSST (15 min) and 20 min after TSST onset (post; 5 min). Moreover, participants provided four saliva samples and concurrent positive and negative affect ratings (PANAS) throughout the experiment (-1, +15, +25, +45 min relative to TSST onset). ER = emotion regulation; TSST = Trier Social Stress Test; PANAS = Positive and Negative Affect Scale.

## 2.5. Cardiovascular, neuroendocrine, and psychological stress measures

### 2.5.1. Heart rate variability

RR intervals were recorded using a wireless HR transmitter, consisting of an elastic chest strap with electrodes placed just below the chest muscles and a wrist monitor (Polar RS800CX, Polar® Electro, Finland), providing a sampling rate of 1000 Hz. Recordings were obtained during the 5 min baseline period, the 15 min TSST period, and the 5 min post-stress period. After data acquisition, device specific software (Polar ProTrainer 5; Polar® Electro, Finland) was used to export the raw RR data for further data processing with Kubios HRV 3.1.0 (Tarvainen, Niskanen, Lipponen, Ranta-Aho, & Karjalainen, 2014) according to the guidelines of the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (1996). Recorded data was detrended (smoothn priors:  $\lambda = 500$ ), visually inspected for abnormal or biologically implausible beats and corrected with a threshold based artifact correction algorithm using cubic spline interpolation that was adjusted individually for every participant. The percentage of successive RR intervals that differ more than 50 ms (pNN50; Task Force of the European Society of Cardiology & the North American Society of Pacing & Electrophysiology, 1996) was calculated for each period from 5 min of continuous data during baseline, TSST (i.e. three 5 min intervals) and post-stress. The pNN50 represents a robust short-term measure of changes in HR that are mediated by the vagus nerve and is closely related to parasympathetic activity (Shaffer & Ginsberg, 2017; Shaffer, McCraty, & Zerr, 2014). Polar monitors are frequently used in psychophysiological research (Colzato & Steenbergen, 2017; Lischke et al., 2018) and have been shown to record RR intervals as accurate as conventional electrocardiogram systems (Weippert et al., 2010).

Eight participants were excluded from HRV analyses due to technical failures during RR recording, leaving a final sample of  $N = 78$ .

### 2.5.2. Salivary cortisol

Saliva samples were collected using Salivette sampling devices (Sarstedt, Nümbrecht, Germany) immediately before the TSST ( $-1$  min) as well as  $+15$  min,  $+25$  min and  $+45$  min after TSST onset and stored at  $-20$  °C until assayed. Free cortisol concentrations were analyzed using commercially available enzyme-linked immunosorbent assays (ELISA; Demeditec, Kiel, Germany). Intra-assay and inter-assay coefficients of variations were below 10%. Data were log-transformed to obtain a normal distribution before use in subsequent analyses.

### 2.5.3. Psychological stress measures

**2.5.3.1. Anticipatory stress appraisals.** To assess anticipatory cognitive stress appraisals about the upcoming TSST, all participants were required to fill out the Primary Appraisal Secondary Appraisal Scale (PASA; Gaab, Rohleder, Nater, & Ehlert, 2005) at the beginning of the TSST preparation period. In brief, the PASA is a 16-item self-report questionnaire consisting of four scales measuring threat and challenge (primary appraisals) as well as self-concept of own abilities and control expectancy (secondary appraisals), which are rated on a 6-point Likert scale ranging from 1 (strongly disagree) to 6 (strongly agree). The primary appraisal scale thus refers to a person's judgment about the significance of an event as stressful, positive, controllable, challenging or irrelevant, whereas the secondary appraisal assesses available coping resources and options when faced with the stressor. A tertiary scale – the stress index – combines the primary and secondary scale and provides a summary measure of stress perception.

**2.5.3.2. Subjective stress ratings.** Immediately following the TSST, participants were asked to rate their feelings of stressfulness and unpleasantness during the TSST on a scale ranging from 0 (not at all) to 100 (very much; rating method adopted from Schwabe, Haddad, & Schachinger, 2008). In the two emotion regulation groups, participants also rated how successful they applied the respective emotion

regulation strategies during the TSST from 0 (not at all) to 100 (very much).

**2.5.3.3. Affect ratings.** In addition, we assessed the participant's affect concurrently with the collection of saliva samples at multiple time points ( $-1$  min,  $+15$  min,  $+25$  min, and  $+45$  min) using the Positive and Negative Affect Scale (PANAS; Watson, Clark, & Tellegen, 1988). The PANAS is a self-report questionnaire consisting of two 10-item subscales measuring positive (PA) and negative affect (NA) on a 5-point scale ranging from 1 (not at all) to 5 (very much).

### 2.6. Habitual emotion regulation

We used the Emotion Regulation Questionnaire (german version used: Ablner & Kessler, 2009; ERQ; Gross & John, 2003) to assess the degree to which individuals habitually employ cognitive reappraisal or expressive suppression in their daily lives. Participants rated the 10-item self-report measure on a 7-point Likert scale ranging from 1 (strongly disagree) to 7 (strongly agree).

### 2.7. Statistical analyses

Statistical analyses were performed in IBM SPSS Statistics for Windows 22.0 with the significance level set to  $\alpha = .05$  and Bonferroni-corrected for multiple comparisons when necessary. Analyses of variance (ANOVA) always included the between-subjects factor group (cognitive reappraisal vs. expressive suppression vs. control). Greenhouse-Geisser corrected  $p$ -values were used if assumptions of sphericity were violated and partial eta square ( $\eta_p^2$ ) were reported as estimations of effect sizes. Since we were specifically interested in emotion regulatory modulated stress responses, we first calculated composite stress measures for cardiovascular, neuroendocrine and psychological stress analyses as follows. For HRV, we computed  $\Delta pNN50$  as a stress reactivity measure by subtracting the baseline from the TSST score (i.e. the mean value of the three 5 min intervals) as well as  $\Delta pNN50_{\text{recovery}}$  as an index of stress recovery by subtracting the mean TSST from the post-stress score. For cortisol and affect ratings, the Area Under the Curve with respect to ground ( $AUC_G$ ) was calculated as an aggregated sensitive measure of neuroendocrine and psychological changes to the TSST over time (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003). The  $AUC_G$  is an index of total hormonal secretion typically employed in neuroendocrine research. It is thought to capture both the intensity (overall distance of cortisol samples from the ground) and sensitivity (difference between repeated cortisol samples) of the system (Fekedulegn et al., 2007; Khoury et al., 2015; Pruessner et al., 2003) and appears to be not only related to momentary states such as stress load but also to trait variables (Hellhammer et al., 2007). Given that we were not only interested in the mere sensitivity of the psychological and neuroendocrine system to a stressor, but also on the impact of instructed emotion regulation – and in particular on the interactive effects of trait and state emotion regulation – on the magnitude of the stress responses, we used the aggregated index to assess overall cortisol secretion and psychological changes across the entire experiment. As such, we were able to capture the combined emotion regulatory and stressor-related effects on pre- as well as on post-TSST values. Univariate ANOVAs were then conducted with the composite stress measures (i.e.  $\Delta pNN50$ ,  $\Delta pNN50_{\text{recovery}}$ ,  $AUC_G$  cortisol and  $AUC_G$  positive and negative affect) in order to explore group differences in stress reactivity or stress recovery as well as with the emotion regulation success rating in order to check how successful participants of the two regulation groups applied the instructed strategy to the stressor. The subscales of the anticipatory stress appraisals (PASA) and the subjective stress ratings were analyzed using multivariate ANOVAs.

To examine whether participants' habitual use of cognitive reappraisal or expressive suppression moderated the relationship between instructed emotion regulation and psychophysiological stress

responding, we conducted moderated regression analyses using the PROCESS macro v3.2.01 for SPSS (model 1; Hayes, 2018). We entered group into the model as the predictor X (dummy coded: control = 0, cognitive reappraisal = X1, expressive suppression = X2), the composite stress measures as the outcome variable Y, and trait reappraisal or suppression as the moderator M (i.e. the mean centered ERQ scores). Significant interactions were probed with simple slope analyses at high (+1SD) and low (-1SD) values of the moderator variable. White-Huber standard errors were used if the assumption of homoscedasticity of residuals were violated (Hayes & Cai, 2007).

### 3. Results

#### 3.1. Sample description

Participants were aged between 18 and 35 years ( $M = 23.72$ ;  $SD = 3.76$ ) and had a mean BMI of  $M = 21.94 \text{ kg/m}^2$  ( $SD = 2.04$ ). They were randomly assigned to either the cognitive reappraisal group ( $N = 30$ ), the expressive suppression group ( $N = 27$ ) or the control group ( $N = 29$ ). Menstrual cycle phase was equally distributed across groups ( $\chi^2_{(4)} = 3.32, p = .50$ ). Likewise, groups did not differ regarding their habitual use of cognitive reappraisal or expressive suppression (both  $F_s < 2.0$  and  $p_s > .14$ ; Table 1A).

#### 3.2. Cardiovascular, neuroendocrine and psychological stress response

##### 3.2.1. Heart rate variability

As illustrated in Fig. 2A, participants in the cognitive reappraisal group showed a significantly steeper stress-induced HRV decrease as compared to participants in the expressive suppression group ( $\Delta pNN50$ :  $F_{(2,75)} = 4.57$ ;  $p < .05$ ;  $\eta_p^2 = .11$ ;  $p = .018$ , 95% CI [-17.96, -1.29] for Bonferroni-corrected post hoc group comparison), but also a stronger albeit not significant HRV recovery in the post-stress phase ( $\Delta pNN50_{\text{recovery}}$ :  $F_{(2,74)} = 1.92$ ;  $p = .15$ ;  $\eta_p^2 = .05$ , Fig. 2B, Supplementary Table 1).<sup>1,2</sup> Even though participants in the control group also showed a relatively strong HRV decrease in response to the stressor on a descriptive level, they did not significantly differ either from the reappraisal or the suppression group (both  $p_s > .05$ ).

##### 3.2.2. Salivary cortisol

With regard to neuroendocrine stress reactivity, ANOVA revealed a significant main effect of group for AUC<sub>G</sub> cortisol ( $F_{(2,83)} = 3.94$ ;  $p < .05$ ;  $\eta_p^2 = .09$ ), indicating a significantly larger total cortisol output in response to the TSST in the expressive suppression group compared to the control group ( $p = .03$ , 95% CI [-51.82, -2.00], Fig. 2C, Supplementary Table 1). No other significant group differences occurred (all  $p_s > .05$ ).

##### 3.2.3. Psychological stress measures

##### 3.2.3.1. Anticipatory stress appraisals.

There was no effect of the emotion regulation manipulation on primary and secondary stress appraisals or the overall stress index (all  $F_s < 1.73$ , all  $p_s > .19$ ;

<sup>1</sup> In order to check whether we can confirm the reported results using another index of vagally mediated HRV, we reran our analyses for HRV stress reactivity and stress recovery using the root mean square of successive differences between normal heartbeats (RMSSD). As expected, the reported main effects of group and the Bonferroni corrected post-hoc group comparisons for both HRV reactivity and HRV recovery were highly similar to the original analyses. Furthermore, a strong positive correlation was found between pNN50 and RMSSD for both the reactivity ( $r = 0.89, p < .001$ ) and recovery index ( $r = 0.87, p < .001$ ).

<sup>2</sup> To account for potential differences in HRV reactivity or HRV baseline scores, we additionally ran an ANCOVA for  $\Delta pNN50_{\text{recovery}}$  including the pNN50 baseline scores as a covariate. Similar to the original analysis, the main effect of group did not reach significance ( $F_{(2,73)} = 0.14$ ;  $p = .87$ ;  $\eta_p^2 = .004$ ).

**Table 1**

Habitual emotion regulation and psychological stress ratings. **A)** Mean ( $\pm$  SEM) habitual reappraisal and suppression scores as assessed with the emotion regulation questionnaire (ERQ), **B)** mean ( $\pm$  SEM) subjective stress and emotion regulation ratings, **C)** mean ( $\pm$  SEM) PASA scores and **D)** mean ( $\pm$  SEM) Area Under the Curve with respect to ground (AUC<sub>G</sub>) for positive and negative affect (PANAS scores) are shown for the cognitive reappraisal, the expressive suppression and the control group. The statistics are described in detail in the text.

	control	reappraisal	suppression
<b>A) ERQ score (1-7)</b>			
habitual reappraisal	5.03 $\pm$ 0.16	4.85 $\pm$ 0.16	4.62 $\pm$ 0.19
habitual suppression	2.98 $\pm$ 0.18	3.52 $\pm$ 0.23	3.21 $\pm$ 0.17
<b>B) subjective stress (0-100)</b>			
unpleasant	65.86 $\pm$ 5.00	74.67 $\pm$ 3.77	79.26 $\pm$ 3.58
stressful	66.21 $\pm$ 5.12	76.00 $\pm$ 2.90	75.19 $\pm$ 4.08
regulation success		50.67 $\pm$ 4.70	42.96 $\pm$ 4.59
<b>C) PASA scales (1-6)</b>			
<i>primary scale</i>			
threat	3.82 $\pm$ 0.15	3.84 $\pm$ 0.13	4.16 $\pm$ 0.16
challenge	3.33 $\pm$ 0.21	3.31 $\pm$ 0.19	3.75 $\pm$ 0.20
<i>secondary scale</i>	4.31 $\pm$ 0.15	4.38 $\pm$ 0.12	4.57 $\pm$ 0.15
self-concept of own abilities	4.16 $\pm$ 0.12	4.18 $\pm$ 0.11	4.42 $\pm$ 0.12
control expectancy	3.91 $\pm$ 0.18	3.72 $\pm$ 0.18	3.92 $\pm$ 0.18
<i>stress index</i>	4.41 $\pm$ 0.14	4.61 $\pm$ 0.10	4.92 $\pm$ 0.14*
<b>D) PANAS scores (AUC<sub>G</sub>)</b>			
positive affect	-0.68 $\pm$ 0.47	-0.64 $\pm$ 0.40	-0.51 $\pm$ 0.40
negative affect	47.54 $\pm$ 3.32	56.81 $\pm$ 2.30*	54.81 $\pm$ 2.44
	14.48 $\pm$ 2.32	16.83 $\pm$ 2.49	16.74 $\pm$ 2.39

Note: ERQ score: 1 = strongly disagree, 7 = strongly agree; subjective stress: 0 = not at all, 100 = very much; PASA scales: 1 = strongly disagree, 6 = strongly agree; PANAS scores: 1 = not at all, 5 = very much; the AUC<sub>G</sub> was calculated as a single measure of affective output in response to the TSST. \* $p < .05$  indicate significant difference between the emotion regulation groups and the control group (Bonferroni-corrected post hoc  $t$ -test).

Table 1C). However, participants in the expressive suppression group anticipated greater control expectancies about the upcoming TSST than participants in the control group ( $F_{(2,84)} = 4.17$ ;  $p < .05$ ;  $\eta_p^2 = .09$ ;  $p = .015$ , 95% CI [-0.95, -0.08] for Bonferroni-corrected post hoc test; Table 1C). No other significant group differences occurred for anticipatory control expectancies (all  $p_s > .27$ ).

##### 3.2.3.2. Subjective stress ratings.

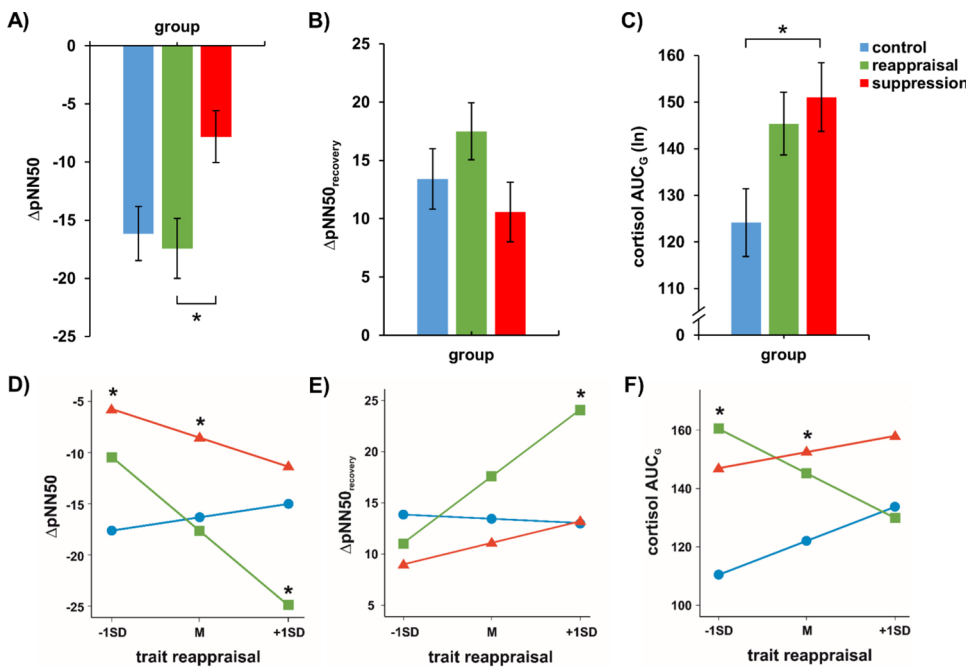
Subjective stress ratings obtained immediately after the TSST revealed that the emotion regulation groups did not significantly differ from the control group in how stressful they experienced the preceding stress procedure ( $p = .18$ , see Table 1B). Yet, the expressive suppression group tended to experience more unpleasantness during the TSST than the control group ( $F_{(2,86)} = 2.61$ ;  $p = .08$ ;  $\eta_p^2 = .06$ ;  $p = .084$ , 95% CI [-28.03, 1.24] for Bonferroni-corrected post hoc test). No other significant group differences occurred for the unpleasantness ratings (all  $p_s > .41$ ). Subjective emotion regulation success did not significantly differ for the cognitive reappraisal ( $M = 50.67$ ,  $SD = 25.72$ ) and expressive suppression group ( $M = 42.96$ ;  $SD = 4.59$ ;  $F_{(1,55)} = 1.37$ ;  $p = .25$ ;  $\eta_p^2 = .02$ ).

##### 3.2.3.3. Affect ratings.

Overall, the reappraisal group reported more positive affect over time relative to the control group (AUC<sub>G</sub> PA:  $F_{(2,86)} = 3.24$ ;  $p < .05$ ;  $\eta_p^2 = .07$ ;  $p = .051$ , 95% CI [-18.58, 0.04] for Bonferroni-corrected comparison). No other group differences occurred for positive affect (all  $p_s > .20$ ) or negative affect (AUC<sub>G</sub> NA:  $p = .74$ ).

### 3.3. Impact of habitual emotion regulation

To test, whether habitual reappraisal moderated the effect of the emotion regulation manipulation on HRV stress reactivity, group was entered into the moderation model as the predictor variable, with  $\Delta pNN50$  as the outcome variable, and the ERQ reappraisal score as the



**Fig. 2.** Cardiovascular and neuroendocrine stress reactivity as a function of instructed and trait emotion regulation. Upper panel: Cardiovascular and neuroendocrine responses to the TSST for the two emotion regulation groups, reappraisal and suppression, and the control group. **A)** The cognitive reappraisal group exhibited a significantly steeper HRV decrease ( $\Delta pNN50$ ) in response to the TSST, **B)** but also a more pronounced HRV recovery ( $\Delta pNN50_{recovery}$ ) in the post-stressor period. **C)** The expressive suppression group showed a significantly larger total cortisol output (i.e. log-transformed  $AUC_G$ ) in response to the TSST when compared to the control group. Lower panel: Habitual reappraisal as a moderator between instructed emotion regulation and stress reactivity measures. **D)** Instructed reappraisal leading to the strongest stress-induced decrease in heart rate variability (HRV;  $\Delta pNN50$ ) for higher (+1SD) levels of habitual reappraisal. **E)** Positive relationship between instructed reappraisal and HRV recovery ( $\Delta pNN50_{recovery}$ ) for high but not low habitual reappraisal levels. **F)** Using reappraisal or suppression during the TSST predicted a higher total cortisol output ( $AUC_G$  cortisol) for low and mean levels of habitual reappraisal, but not for high levels of reappraisal. \* $p < .05$ , Bonferroni-corrected comparisons.

**Table 2**  
Moderated regression analyses examining the relation between instructed emotion regulation (i.e. group allocation = predictor) and stress reactivity measures (outcome), moderated by habitual reappraisal (moderator).

Model 1: Predicting $\Delta pNN50$					
predictor	$\beta$	SE $\beta$	t	p	95%-CI
constant	-16.39	2.34	-7.01	.00	[-21.05, -11.73]
reappraisal group	-0.92	3.19	-0.29	.77	[-7.28, 5.44]
suppression group	7.97	3.24	2.46	.02	[1.51, 14.43]
ERQ_R	1.35	2.11	0.64	.52	[-2.86, 5.56]
ERQ_R x reappraisal group	-8.87	2.83	-3.13	.00	[-14.50, -3.23]
ERQ_R x suppression group	-4.27	3.85	-1.11	.28	[-11.94, 3.41]
Model 2: Predicting $\Delta pNN50_{recovery}$					
predictor	$\beta$	SE $\beta$	t	p	95%-CI
constant	13.47	2.53	5.33	.00	[8.43, 18.51]
reappraisal group	3.82	3.29	1.16	.25	[-2.74, 10.38]
suppression group	-2.47	3.72	-0.66	.51	[-9.89, 4.95]
ERQ_R	-0.43	2.15	-0.20	.84	[-4.71, 3.86]
ERQ_R x reappraisal group	7.24	2.61	2.78	.01	[2.04, 12.44]
ERQ_R x suppression group	2.63	3.43	0.77	.45	[-4.21, 9.47]
Model 3: Predicting $AUC_G$ cortisol					
predictor	$\beta$	SE $\beta$	t	p	95%-CI
constant	121.76	7.00	17.39	.00	[107.82, 135.69]
reappraisal group	23.83	9.72	2.45	.02	[4.48, 43.19]
suppression group	30.60	10.11	3.03	.00	[10.49, 50.72]
ERQ_R	12.48	7.18	1.74	.09	[-1.81, 26.77]
ERQ_R x reappraisal group	-28.86	10.50	-2.75	.01	[-49.76, -7.95]
ERQ_R x suppression group	-6.55	10.19	-.64	.52	[-26.83, 13.73]

Note: ERQ\_R, reappraisal score of the emotion regulation questionnaire; SE, standard error. Outcome variables:  $\Delta pNN50$ , indexing stress-induced decreases in heart rate variability (HRV; (Model 1),  $\Delta pNN50_{recovery}$  indexing HRV recovery (Model 2) and  $AUC_G$  cortisol indexing total cortisol output (Model 3). The predictor variable group was dummy coded: 0 = control; X1 = reappraisal; X2 = suppression.

moderator. Analyses were repeated with the other composite stress measures as the outcome variable, as well as with expressive suppression as the moderator variable. Significant moderation findings are displayed in Table 2.

For HRV reactivity, the model predicted a significant amount of variance in  $\Delta pNN50$  ( $R^2 = .22$ ,  $F_{(5, 72)} = 6.04$ ,  $p < .001$ ) and the interaction between reappraisal group and ERQ reappraisal significantly increased the explained variance ( $\Delta R^2 = .07$ ,  $F_{(2,72)} = 4.93$ ,  $p < .01$ ; Table 2), indicating that habitual reappraisal moderated the effect of instructed reappraisal on stress-induced HRV decreases. As illustrated in Fig. 2D, participants with higher levels of habitual reappraisal (+1SD) and allocated to the reappraisal group showed the steepest HRV decrease in response to the stressor ( $\beta = -9.04$ ,  $p < .05$ , 95% CI [-17.48, -0.59]). By contrast, for participants with lower (-1SD) or mean reappraisal scores, allocation to the suppression group was rather associated with a less pronounced stress-induced decrease in HRV ( $\beta = 11.87$ ,  $p < .05$ , 95% CI [2.73, 21.02] and  $\beta = 7.97$ ,  $p < .05$ , 95% CI [1.51, 14.43], respectively).

With regard to HRV stress recovery, the overall model predicted a significant amount of variance ( $\Delta pNN50_{recovery}$ :  $R^2 = .14$ ,  $F_{(5, 71)} = 9.87$ ,  $p < .001$ ) with habitual reappraisal again moderating the effect of instructed reappraisal on  $\Delta pNN50_{recovery}$  ( $\Delta R^2 = .04$ ,  $F_{(2,71)} = 4.16$ ,  $p < .05$ , Table 2). Participants in the reappraisal group showed a significantly stronger HRV increase in the post-stressor phase when they scored high ( $\beta = 10.45$ ,  $p < .05$ , 95% CI [2.05, 18.85]) on habitual reappraisal, while instructed reappraisal had no effect on HRV stress recovery at mean or low levels of habitual reappraisal (see Fig. 2E).

For the total cortisol output over time, the model predicted a significant amount of variance in  $AUC_G$  cortisol ( $R^2 = .17$ ,  $F_{(5, 80)} = 3.33$ ,  $p < .01$ ) and a significant increase in explained variance due to the reappraisal group  $\times$  ERQ reappraisal interaction ( $\Delta R^2 = .08$ ,  $F_{(2,80)} = 4.07$ ,  $p < .05$ , Table 2). As shown in Fig. 2F, for participants scoring low or mean on habitual reappraisal the use of both reappraisal and suppression during the TSST was related to a significantly higher stress-induced total cortisol output (-1SD: reappraisal group:  $\beta = 51.53$ ,  $p < .001$ , 95% CI [22.24, 80.81]; suppression group:  $\beta = 36.89$ ,

$p < .01$ , 95% CI [9.12, 64.65], Mean: reappraisal group:  $\beta = 23.83$ ,  $p < .05$ , 95% CI [4.48, 43.19]; suppression group:  $\beta = 30.60$ ,  $p < .01$ , 95% CI [10.49, 50.72]), while instructed emotion regulation had no effect on AUC<sub>G</sub> cortisol when habitual reappraisal was high.

No significant predictors were found for AUC<sub>G</sub> positive and negative affect. Likewise, no significant results were obtained when habitual suppression was entered as a moderator variable.

#### 4. Discussion

Stress typically elicits negative emotions, impedes executive control functions (Arnsten, 2009; Shields, Sazma, & Yonelinas, 2016; Wolf, 2017) and is often a precursor to psychological disorders (Grant et al., 2003; Kopp & Réthelyi, 2004), making the identification of effective strategies for reducing stress imperative. In the present study, we therefore investigated whether deliberately regulating negative emotions could improve psychophysiological responses to an acute psychosocial stressor. We found that cognitive reappraisal but not expressive suppression fostered an overall adaptive and flexible pattern of cardiovascular, neuroendocrine and psychological responses. In accordance with accumulating evidence from correlational and experimental studies (Beltzer et al., 2014; Gross & John, 2003; Jamieson et al., 2012; John & Gross, 2004; Mauss et al., 2007), reappraisal generally enhanced positive affect as compared to a no-regulation control condition, indicating that reframing negative events in a more positive light might help individuals to maintain positive mood despite being stressed. Furthermore, using reappraisal relative to suppression led to significantly stronger decreases in HRV during stress, but also initiated a more pronounced HRV recovery in the aftermath of stress. In line with our match-mismatch hypothesis, this reappraisal-induced cardiac vagal flexibility was especially prominent in individuals who also habitually used reappraisal as an emotion regulatory strategy in their daily lives. Participants in the control group seemed to show a descriptively similar pronounced HRV decrease in response to the TSST, however, without significantly differing from either the reappraisal or the suppression group. Likewise, controls did not exhibit a stronger HRV recovery when compared to the other two groups, nor did habitual emotion regulation altered HRV reactivity or recovery for controls. Our results are hence consistent with a growing literature demonstrating that emotion regulation, in particular cognitive reappraisal, can increase cardiovascular reactivity under stress but at the same time also promotes a quick return to vagally mediated baseline levels after stress (Jamieson et al., 2012, 2013; Liu et al., 2017; Neumann et al., 2004). Congruently, prolonged cardiovascular reactivity following stress exposure (i.e. poorer recovery) has been linked to emotion regulation difficulties and maladaptive regulatory strategies, such as rumination (Berna, Ott, & Nandrino, 2014; Key et al., 2008). Our data extend these findings by providing first evidence that trait forms of emotion regulation strategies (i.e. the way we usually regulate our emotions) critically moderate the efficacy of instructed emotion regulation to improve stress responses. We thereby support the match-mismatch hypothesis, implying that deliberately regulating stress-related emotions via a certain strategy would be more automatic, less effortful and thus also more effective when it matches our habitual preferences to use this strategy in daily life.

Of note however, this relationship was only observed for cognitive reappraisal, whereas the habitual use of expressive suppression did not moderate the effects of instructed suppression on psychophysiological stress responding. A potential explanation for the differential impact of reappraisal and suppression might be related to the temporal characteristics of the two emotion regulation strategies (Gross, 2001). Cognitive reappraisal belongs to the family of antecedent-focused strategies, which are applied before an emotion has completely unfolded and thereby intervene relatively early in the emotion-generative process. By this, it directly alters physiological response tendencies without the need for sustained cognitive effort over time. By contrast, expressive suppression is a response-focused strategy and by definition implemented following emotion generation, which in turn may involve

increasing efforts to actively inhibit prepotent emotional behaviors and expressions (Gross, 1998a,b). It is thus reasonable, that even a frequent use and almost automated process of suppression does not necessarily foster its' adaptability in stressful contexts. This idea lines up with a large body of literature showing that both trait as well as instructed expressive suppression is usually associated with increases rather than decreases in negative affect, SNS reactivity and emotion-related brain activity (Gross, 2015).

As expected and consistent with correlational data (Lam et al., 2009; Raymond et al., 2019), we further found participants required to actively suppress stress-related emotions and expressions to experience the TSST as slightly more unpleasant and to display a significantly stronger cortisol secretion when compared to controls that did not actively engage in any emotion regulation strategy. Contrary to the results reported by Denson and colleagues (2014) however, we did not observe this heightened cortisol reactivity in the cognitive reappraisal group compared to controls. The result pattern is yet consistent with another study, revealing trait reappraisal but not suppression to be negatively correlated with cortisol, heart rate and state anxiety but positively correlated with state euphoria in response to a first-time tandem skydive (Carlson et al., 2012). Moreover, it has been recently shown that enhancing positive feelings by recalling positive memories could dampen cortisol responses to stress (Speer & Delgado, 2017), whereas ruminating about a stressor was rather related with non-habituation of cortisol to repeated stress exposure (Gianferante et al., 2014). Given that reappraisal but not suppression increased positive affect in the current study, it may thus be speculated that the effects of positive emotionality and increased cognitive load on cortisol responses could have canceled each other out in the reappraisal group. However, since we did not measure emotion regulation effort directly, it remains to be explored whether task load is a potential mechanism by which cognitive reappraisal leads to either heightened or dampened HPA axis reactivity.

Pointing into a similar direction, our moderation analyses further revealed that for participants scoring low on habitual reappraisal, employing cognitive reappraisal during the TSST was related to significantly higher stress-induced cortisol levels. Importantly, these findings provide further evidence for the idea that cognitive reappraisal, which is typically considered adaptive and shown to be effective in downregulating negative reactivity, might be cognitively too demanding and thus ineffective when this strategy is not frequently used in daily life. Our data suggest that such a mismatch between instructed and the usually preferred emotion regulation strategy is especially problematic under stressful conditions. In line with this notion, post-stressor ratings indicated that both emotion regulation groups were only moderately successful (mean success of 47 on a scale ranging from not at all (0) to very much (100)) in applying the instructed strategy to the current stress task.

There are some limitations of the present study. First, we tested only women and the findings may not generalize to men. Sex differences in HPA stress responsiveness (Kudielka & Kirschbaum, 2005) as well as its cognitive consequences (Merz & Wolf, 2017) are frequently reported and women typically show enhanced behavioral, physiological and neural reactivity to emotional stimuli (Bradley et al., 2001; Canli et al., 2002; Lithari et al., 2010). Women also report to engage more in (putatively adaptive) emotion regulatory processes (Nolen-Hoeksema, 2012; Tamres et al., 2002). For future studies, it would be therefore desirable to explore if certain emotion regulation strategies are more effective for one sex or the other. Since emotion dysregulation is viewed central to several types of psychopathology (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Sheppes, Suri, & Gross, 2015), it will be essential to investigate if strategy selection, both in terms of individual preferences but also situational demands could predict emotion regulation success.

Second, it was somewhat unexpected that the expressive suppression group but not the cognitive reappraisal group reported greater control expectancies about the upcoming TSST. However, stress appraisals were obtained only prior to the stressor, and we do not know if suppressors

would have continued to feel in control also after being stressed. Although the PASA was originally designed as a measure of anticipatory stress appraisals, it would have been therefore informative to include an additional post-stressor measurement in order to characterize potential changes in stress appraisals or self-efficacy and coping due to emotion regulation when the acute stress state subsides. Related to this, it has been shown that dispositional and state coping styles can not only explain variability in stress-related HPA axis reactivity but also recovery (Biondi & Picardi, 1999; Hühne et al., 2014; Zoccola & Dickerson, 2012), indicating in particular a steeper and thus more rapid post-stressor cortisol decline for individuals using adaptive coping styles (Janson & Rohleder, 2017). For the current study, it is thus reasonable to assume that participants in the reappraisal group would have also shown a stronger or quicker cortisol recovery, particularly when they are used to apply this strategy in their daily lives. However, in order to compute a reliable index for cortisol recovery additional post-stressor cortisol samples would have been necessary. Future studies extending the post-stressor follow-up time period are thus warranted in order to provide more fine-grained insights into the impact of emotion regulation on psychological and neuroendocrine stress recovery. Given that both excessive cortisol secretion as well as insufficient recovery in response to stress are associated with mental and physical health impairments (Cohen, Janicki-Deverts, & Miller, 2007), it is crucial to pinpoint which factors might contribute to long-term stress adaptation and recovery or maladjustment and psychopathology. Finally, another issue regards power. Post-hoc power analyses conducted in G\*Power 3.1 (Faul, Erdfelder, Buchner, & Lang, 2009) revealed that for most of our main analyses of interest (ANOVAs and moderated regression analyses for  $\Delta$ pNN50 and AUC<sub>G</sub> cortisol) the given sample size was sufficient to provide an estimated power of at least 73–81% to detect a medium-sized effect. However, we must acknowledge that statistical power was limited for the detection of smaller effects, such as those observed for  $\Delta$ pNN50<sub>recovery</sub>. These results should thus be treated with caution until replication with a larger sample.

In conclusion, the present study demonstrated that regulating negative emotions via cognitive reappraisal (but not suppression) fostered psychological adaptation and cardiac vagal flexibility in response to an acute stressor. Our findings thereby suggest that cognitive reappraisal promotes regulatory flexibility, which may help individuals to dynamically adjust physiological arousal in line with situational demands. Yet, the current data further imply that the efficacy of emotion regulation strategies critically depends on whether they are also habitually used in daily life.

#### Data availability

The data set analyzed during the current study is available at the Open Science Framework (OSF) under <https://osf.io/rntbz/>.

#### Disclosure of data collection and analysis

We hereby confirm that, for all experiments, we have reported all measures, conditions and data exclusions.

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#### Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at [doi:https://doi.org/10.1016/j.biopsycho.2020.107893](https://doi.org/10.1016/j.biopsycho.2020.107893).

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