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**INDIVIDUAL DIFFERENCES
IN EMOTION REGULATION AND CARDIOVASCULAR RESPONDING TO
STRESS**

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Abstract

Instructed use of reappraisal to regulate stress in the laboratory is typically associated with a more adaptive cardiovascular response to stress, indexed by either: (i) lower cardiovascular reactivity (CVR; e.g., lower blood pressure); or (ii) a challenge-oriented response profile (i.e., greater cardiac output paired with lower total peripheral resistance). In contrast, instructed use of suppression is associated with exaggerated CVR (e.g., greater heart rate, blood pressure). Despite this, few studies have examined if the *habitual* use of these strategies are related to cardiovascular responding during stress. The current study examined the relationship between cardiovascular responses to acute stress and individual differences in emotion regulation style: trait reappraisal, suppression, and emotion regulation difficulties. Forty-eight participants (25 women, 23 men) completed a standardised laboratory stress paradigm incorporating a 20-minute acclimatization period, a 10-minute baseline, and two 5-minute speech tasks separated by a 10-minute inter-task rest period. The emotional valence of the speech task was examined as a potential moderating factor; participants spoke about a block of negative-emotion words and a block of neutral-emotion words. Cardiovascular parameters were measured using the Finometer Pro. Greater habitual use of suppression was associated with exaggerated blood pressure responding to both tasks. However, only in response to the negative-emotion task was greater use of reappraisal associated with a challenge-oriented cardiovascular response. The findings suggest that individual differences in emotion regulation translate to differing patterns of CVR to stress, but the emotional valence of the stressor may play a role.

Key words: Reappraisal, Suppression, Emotion Regulation Difficulties, Cardiovascular Reactivity, Hemodynamic Profile

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Research on the use of two emotion regulation strategies, reappraisal (cognitively reframing a situation to change the emotional impact; Gross, 1998a, 2015; Jamieson, Mendes, et al., 2013) and suppression (consciously inhibiting the expression of any emotional response; Gross, 2015; Gross & Levenson, 1997), has grown exponentially in the past few decades. Use of these strategies, whether instructed in the laboratory or measured via self-report (habitual use of these strategies in day-to-day life), are thought to have implications for psychological, and by extension, physical health. Less research has examined the consequences of difficulties in emotion regulation (Gratz & Roemer, 2004), which focuses on the different facets involved in successful emotion regulation rather than the use of certain strategies; an approach that emphasises awareness and understanding of emotions, as well as the ability to monitor, evaluate, and modify the emotional experience.

Reappraisal and Suppression

In general, habitual use of reappraisal is seen as a more healthful strategy to regulate emotional responses and is associated with greater positive affect and lower negative affect, perceived stress, anxiety, and depression (e.g., Balzarotti et al., 2017; Chervonsky & Hunt, 2018). Habitual use of suppression tends to be associated with greater negative affect, greater anxiety and depressive symptoms, and lower positive affect (e.g., Nolen-Hoeksema & Aldao, 2011). However, under certain conditions reappraisal may not always be positive (Ford & Troy, 2019) and suppression always negative (Geisler & Schröder-Abé, 2015; Girme et al., 2020). Importantly, habitual use of these strategies appears to reflect stable behavioural tendencies and show temporal stability (e.g., Balzarotti et al., 2010; Batistoni et al., 2013; Gross & John, 2003). It is assumed that habitual use of these strategies reflects actual use of these strategies in daily life, but only a handful of studies have empirically tested this claim. However, available evidence suggests high concordance between habitual use of, and implementation of, these strategies (Egloff et al., 2006; Gračanin et al., 2017; Peters et al.,

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2020). Likewise, *state* and *trait* measures of emotion regulation correlate highly (Lavender et al., 2017).

While the associations between habitual emotion regulation style and well-being are well-documented, less research has examined how the use of these strategies affects physical health. Evidence from studies *instructing* the use of reappraisal and suppression in the laboratory suggest these strategies may influence long-term physical health via physiological responses to stressful stimuli. Reappraisal refers to both *situation*-based and *arousal*-based reappraisal strategies (Gross, 2015); both conceptualisations reflect changing *appraisals* of stress, which is hypothesised to lead to changes in cardiovascular responses during stress. For instance, reappraising a stress-eliciting situation in a way to view it as less “stressful” (situation-based reappraisal) should be reflected by lower cardiovascular responding to the situation, consistent with the transactional model of stress and coping (Lazarus & Folkman, 1984). Whereas, arousal reappraisal focuses on reframing feelings of stress arousal as a resource (e.g., view increased heartbeat as a sign the body is adapting to stress), and is thought to change *resource* appraisals, resulting in a challenge-oriented cardiovascular response (for reviews see; Crum et al., 2020; Jamieson et al., 2018), consistent with the biopsychosocial model of challenge and threat (Blascovich & Tomaka, 1996). Conversely, suppression involves continuously inhibiting the emotional response, and this is thought to incur high cognitive and physiological costs (e.g., Appleton & Kubzansky, 2014), resulting in exaggerated cardiovascular responding (Richards & Gross, 1999).

Indeed, instructed situation-based reappraisal results in lower cardiovascular reactivity, such as lower blood pressure (e.g., Ayduk & Kross, 2008; Gruber et al., 2014) and instructed suppression leads to greater cardiovascular reactivity, such as greater heart rate (HR) and blood pressure responses (Butler et al., 2003; Harris, 2001; Quartana & Burns, 2010; Roberts et al., 2008). Results from experimental studies, therefore, suggest reappraisal

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may buffer physiological responses to stress, while use of suppression exacerbates responses to stress. This may have implications for long-term physical health considering that exaggerated cardiovascular responses to stress in the laboratory predict increased incidence of future cardiovascular disease (for a review see; Chida & Steptoe, 2010), consistent with the predictions of the well-established cardiovascular reactivity hypothesis (Treiber et al., 2003; Treiber et al., 1997).

However, only a handful of studies have examined the physiological correlates of the *habitual* use of these strategies during stress. Some evidence suggests trait reappraisal moderates cardiovascular reactivity, but there is little evidence to suggest trait suppression influences cardiovascular reactivity during stress. For example, greater habitual use of reappraisal was associated with lower blood pressure reactivity in response to a speech task; however, trait suppression had no influence on blood pressure (Memedovic et al., 2010). Arguably, examining individual differences in the use of reappraisal and suppression is more elucidating of the longer-term psychophysiological outcomes of using these strategies, compared to artificially manipulating them in the laboratory, if indeed habitual use of these strategies reflects a stable behavioural tendency. Results from longitudinal research provides some evidence linking differences in emotion regulation style with disease risk, as indexed by levels of C-reactive protein (a potential inflammatory marker of cardiovascular disease; Danesh et al., 2004) and overall cardiovascular disease risk (Kubzansky et al., 2011). Furthermore, cross-sectional evidence has shown that greater habitual use of reappraisal was associated with lower C-reactive protein levels, while suppression was associated with higher C-reactive protein levels (Appleton et al., 2013).

More recently, research has focussed on the *type* of cardiovascular response elicited by examining how the two parameters underlying blood pressure responding, cardiac output (CO) and total peripheral resistance (TPR), respond; the underlying hemodynamic response

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profile. A marked increase in CO, accompanied by a decrease in TPR, is reflective of a myocardial (challenge-oriented) response, which is thought to be a more healthful response to active stress (i.e., stress tasks that require active engagement, such as speech tasks). Whereas, an increase in TPR accompanied by a decrease in CO is indicative of a vascular (threat-oriented) response. An increase in both indicates a mixed response. Changes in blood pressure can be marked by different hemodynamic responses; myocardial, vascular, or mixed (e.g., Light & Sherwood, 1989). Previous research experimentally manipulating the use of arousal-based reappraisal has shown reappraisal is associated with greater CO and lower TPR during active stress tasks (e.g., Jamieson et al., 2012; Jamieson, Nock, et al., 2013; Oveis et al., 2020), or a challenge-oriented response as indexed by the challenge-threat index (Hangen et al., 2019). Although one study found instructed reappraisal did not influence patterns of CO and TPR responding (Griffin & Howard, 2021), the general consensus from this research adds weight to the hypothesis that use of reappraisal buffers the negative physiological effects of stress. Furthermore, one study has shown *habitual* reappraisal to be associated with increased CO and decreased TPR during a math task (Mauss et al., 2007). Other research has shown that instructed suppression is associated with a more vascular response profile (greater TPR responding) during dyadic interaction tasks (e.g., Peters & Jamieson, 2016; Peters et al., 2014), but no study has examined *habitual* suppression in this way.

Difficulties in Emotion Regulation

Examining specific emotion regulation strategies, such as reappraisal and suppression, offers a micro-level approach to researching emotion regulation processes, where the focus is on what strategies are implemented to regulate the emotional response. However, some researchers have focused on a more macro-level approach (Tull & Aldao, 2015), examining one's ability to adaptively respond to emotions experienced. Indeed, effective emotion regulation is argued to relate to an ability to function adaptively when presented with

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experiences that elicit intense emotional responses (Bonanno & Burton, 2013; Pollock et al., 2016). Some research in this area has focused on problematic responding to emotional stimuli, examining difficulties in six key areas thought to underlie successful emotion regulation; acceptance of emotional responses, impulse control over thoughts and actions, goal-directed behaviour, emotional awareness, emotional clarity, and perceived access to effective emotion regulation strategies (Gratz & Roemer, 2004). An assessment of these deficits may offer a more expansive evaluation of emotion regulation than that captured by assessing the use of certain strategies (such as trait reappraisal and suppression).

While research has documented that greater emotion regulation difficulties are associated with greater indices of psychological distress, such as anxiety and depression (e.g., Bardeen et al., 2012; Salters-Pedneault et al., 2006), few studies examine if emotion regulation difficulties are associated with physiological responses to stress. Some evidence stems from research focusing on cortisol responding where emotion regulation difficulties moderated the relationship between early life stress and blunted cortisol responding to stress (Carnuta et al., 2015; England-Mason et al., 2017). In both these studies, only individuals scoring high in emotion regulation difficulties (who had experienced such adversity) demonstrated dysregulated cortisol responding. Furthermore, evidence from a cross-sectional study implicates emotion regulation difficulties in stress responsivity; only in individuals reporting greater emotion regulation difficulties was chronic stress associated with greater cardiovascular disease risk (Roy et al., 2018). This research suggests that emotion regulation ability may buffer the physiological impact of chronic stress on cardiovascular disease risk factors, and by association influence cardiovascular reactivity to stress, however, this is yet to be tested.

Key Issues and Further Considerations

Research examining the use of suppression to regulate arousal has suggested the

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emotional valence of the task is important. Suppression is hypothesised to be an active process, involving effort, whereby a person attempts to inhibit expression of any concurrent emotional responses; as such, this conflict between inhibiting an emotional response and impulses to express this response, leads to greater physiological activation (Gross & Levenson, 1997; Wegner & Gold, 1995). It is therefore argued, and demonstrated empirically, that suppressing responses to non-emotional stimuli (such as neutral emotion film clips) does not lead to changes in physiological responses (Gross & Levenson, 1997) as this express-suppress conflict is not as strong (i.e., less emotion to potentially express, therefore less effort is needed to inhibit this response). Similarly, research on the cognitive costs of suppression demonstrates that instructed suppression does not influence recall of neutral-emotion words from the Affective Norms for English Words (ANEW; Bradley & Lang, 1999), but results in poorer recall of negative-emotion words from this list (Depue et al., 2006). In the present study, we wished to explore if the emotion valence of a task influences the relationship between *habitual* suppression and physiological responding, and explore this for habitual reappraisal use and emotion regulation difficulties. We theorise that individuals who habitually engage in suppression may demonstrate greater physiological responding during the negative-emotion speech task as this may incur more effort compared to the neutral-emotion task.

Furthermore, while some research employs the challenge-threat index (Hangen et al., 2019), most studies that examine hemodynamic response patterns simply report if CO and TPR responses increase and/or decrease. With this approach, the compensatory relationship between the two parameters is ignored; this confounds the hemodynamic profile (HP; the way in which CO and TPR compensate/respond), and the Compensation Deficit (CD; the degree to which they compensate), for a review see Gregg et al. (2002). To overcome this, research can employ an orthogonal model, the HP-CD model, that computes continuous

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measures for hemodynamic profile and the compensation deficit (Gregg et al., 2002; James et al., 2012). Indeed, the way in which the challenge-threat index is computed (e.g., Blascovich et al., 2004), is argued to, and has been shown to, reflect a similar computation to that employed by the HP-CD model (Griffin & Howard, 2021). Research in this area needs to employ one, if not both, of these approaches to confirm if observed patterns of CO and TPR responding reflect a challenge- or threat-oriented cardiovascular response.

The Current Study

While instructed use reappraisal and instructed suppression are associated with cardiovascular responding to stress, less research has taken the *individual difference* approach, particularly when examining blood pressure reactivity. The current study will therefore employ an active stress task (a speech task) to examine the relationship between cardiovascular reactivity to stress and individual differences in emotion regulation within a standardized laboratory paradigm. The emotional valence of the speech task will be manipulated; participants will speak about a block of neutral-emotion and negative-emotion words taken from the ANEW. The ANEW provides a verbal version of the existing International Affective Pictures System (Lang et al., 2008). Past research has employed words from the ANEW to successfully manipulate the emotional nature of the stimulus presented (negative-emotion words versus neutral-emotion words; Depue et al., 2006; Dowd & Barch, 2010). This study will be the first to explore the relationship between emotion regulation difficulties and blood pressure responding, and the relationship between *habitual* suppression and patterns of hemodynamic responding. Furthermore, we will extend past research reporting on patterns of CO and TPR responses, by applying the HP-CD model computation (Gregg et al., 2002) and the challenge-threat index (Hangen et al., 2019). We will compare these computations as arguably they attempt to measure the same construct. We will also report upon patterns of CO and TPR responding so that our results are comparable

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to previous research reporting upon these patterns.

We expected that greater habitual use of reappraisal would be associated with lower blood pressure responses to the task, and this would be underpinned by a myocardial (challenge-oriented) response. In contrast, we expected a greater tendency to use suppression to be associated with greater blood pressure responding, underpinned by a vascular response, but that this might only be evident in response to the negative task. We explored if the relationship between reappraisal and cardiovascular responding would differ between the negative and neutral-emotion task. We also explored if emotion regulation difficulties were related to cardiovascular responding to stress; with an expectation that greater emotion regulation difficulties would be associated greater blood pressure reactivity, and this would be underpinned by a vascular hemodynamic profile.

Method

Design

The current study employed a 3×1 within-subjects design. The within-subjects factor was phase with three levels; baseline, negative-emotion task, and neutral-emotion task. Emotion regulation scores (reappraisal, suppression, and emotion regulation difficulties) were entered as covariates. The dependent variables were self-reported negative and positive affect, and mean systolic blood pressure (SBP), diastolic blood pressure (DBP), HR, CO, and TPR responses for each phase.

Participants

In total 51 participants reporting good health, no history of cardiovascular disease, and not taking medication known to affect blood pressure completed the study. Four participants were excluded from this sample; cardiovascular assessment was not available for one participant, and three participants had resting blood pressure classified as potentially hypertensive (SBP/DBP > 140/90 mmHg), according to the guidelines for hypertension

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(Chobanian et al., 2003; Whitworth & Chalmers, 2004). The analyses report upon a final sample of 48 (25 women and 23 men) healthy young adults aged 18-24 ($M_{age} = 19.77$, $SD = 1.39$), testing as normotensive¹. Participants were undergraduate students enrolled in a third-level Irish institution. Participants were required to be non-smokers, and prior to participation refrain from consuming caffeinated products for six hours and alcohol for 12 hours, due to the influence of these substances on blood pressure (Ginty et al., 2014; McFadden et al., 2005; Savoca et al., 2005).

Previous research has reported medium to large effect sizes for the influence of instructed reappraisal and suppression on cardiovascular parameters (Ayduk & Kross, 2008; Jamieson et al., 2012; Jamieson, Nock, et al., 2013), and medium effect sizes for individual differences in emotion regulation (England-Mason et al., 2017; Mauss et al., 2007; Memedovic et al., 2010). A priori power analyses were conducted with G*Power (Faul et al., 2007), using an alpha of .05, and power of .80. To detect a significant Phase $\times$ Emotion Regulation interaction effect, a minimum of 25 participants were needed to detect large effects ($f^2 = .35$) and 55 to detect medium effects ($f^2 = .15$). The study therefore had sufficient power to detect large, and to a degree medium, effects. As such, the present study lacked power to detect small effects and the results should be interpreted with this in mind.

Participants received a financial contribution of €25 towards any travel expenses incurred when travelling to the laboratory. Ethical approval was obtained from the institutional research ethics committee. All participation was voluntary, participants signed an informed consent form and could withdraw from the study at any time.

Materials and Apparatus

The Finometer PRO

The Finometer PRO (Finapres Medical Systems BV, BT Arnhem, The Netherlands)

¹ For full participant characteristics see supplementary materials (Table S1)

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was employed to measure cardiovascular parameters non-invasively; providing beat-to-beat measures of blood pressure and hemodynamic monitoring, based on the volume-clamp method (Peñáz, 1973). CO and TPR indicators are provided based on the validated Modelflow modelling method (Wesseling et al., 1995; Wesseling et al., 1993).

Individual Differences in Emotion Regulation

The Emotion Regulation Questionnaire (ERQ; Gross & John, 2003) is a 10-item measure of the emotion regulation strategies of cognitive reappraisal (6 items) and expressive suppression (4 items). Higher scores represent a greater tendency to use each strategy. The Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004) is a 36-item measure of emotion regulation deficits when distressed; with higher scores indicative of greater difficulties. Each scale showed good reliability with a Cronbach's α of .85, .65, and .95 for the reappraisal, suppression, and difficulties in emotion regulation, respectively.

Positive Negative Affect Schedule (PANAS)

The PANAS (Watson, Clark, & Tellegen, 1988) is a 20-item scale measuring state positive and negative affect. Scores are summed for each subscale providing a total positive affect score and total negative affect score. The PANAS demonstrated good internal reliability at baseline and post-tasks, Cronbach's α was $\geq .79$ for each scale.

Stressor task

A socio-evaluative speech task was employed, where a block of forty negative-emotion words and a block of forty neutral-emotion words were presented on Superlab (Version 2.02; Cedrus Corporation; San Pedro, CA); word block order was counterbalanced across participants. Words were chosen from the ANEW (Bradley & Lang, 1999) using previously reported cut-offs (Scott et al., 2009) for arousal (high, low) and valence (negative/positive emotions elicited); words with an arousal value >6 and a valence value <4 were deemed negative (e.g., abuse, drown, murderer), and words with an arousal value <5.45

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and valence value between 4 and 6 were deemed neutral (e.g., egg, book, farm). This speech task has been shown to reliably elicit activity of the cardiovascular system (Griffin & Howard, 2020, 2021).

Procedure

All participants visited the laboratory at an assigned time slot between 8:30am and 2pm to minimize the impact of diurnal changes to blood pressure. Participants were greeted by the researcher and seated at a desk with a personal computer screen, separated from the experimenter by a partition. Participants completed psychometric scales and were given neutral reading material to read (the *National Geographic* magazine) during a 20-minute acclimatization period. The Finometer cuff was then attached to the middle finger of the non-dominant hand, and resting cardiovascular measures were taken during an official 10-minute baseline period while participants completed the affective scales. During the baseline and the inter-task rest period, reading material was provided to lower the risk of potential boredom and/or rumination arousal, employing the recommended Vanilla resting baseline (Jennings et al., 1992). Following baseline, the first 5-minute speech task began. Participants were informed verbally that words would appear on the screen and the aim of the task was to talk about each word for as long as possible. Participants were asked to look at the camcorder during the task as this recording would later be evaluated by body language experts (these directions were simply to increase the socio-evaluative aspects of the task). Participants were first presented with either a block of entirely negative-emotion words or a block of entirely neutral-emotion words (block order counterbalanced across participants). The experimenter evaluated when to change the word presented, either after three seconds of silence (as measured by a stopwatch) or repetition. After five-minutes, the task was stopped and participants were asked to fill out affective rating scales. After a ten-minute inter-task rest period, participants completed the speech task for a second time. Participants then completed

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self-reported affect measures, were debriefed, and thanked for their participation.

Results

Overview of Analyses

The mean values for each cardiovascular parameter (SBP, DBP, HR, CO, and TPR) during the baseline, negative-emotion task, and neutral-emotion task were calculated and are presented in Table 1. Internal consistency for each cardiovascular variable was excellent with Cronbach's $\alpha > .98$ for all phases. Reactivity scores to each task, for each cardiovascular parameter, were computed by subtracting mean baseline values from the mean of the task. Reactivity scores were only used for initial correlation analyses and to calculate the challenge-threat index. However, for the main analyses (ANCOVAs) mean values for each cardiovascular parameter were used.

First, a series of correlations were conducted to explore the associations between the primary study variables, and are presented in Table 2. To examine the association between trait emotion regulation (reappraisal, suppression, and difficulties) and changes in cardiovascular responding over time, a series of custom-built 3×1 repeated measures ANCOVAs were conducted. The within-subjects factor was phase (baseline, negative-emotion task, and neutral-emotion task). Continuous scores on trait emotion regulation were entered as a covariate. The dependent variables were *mean* cardiovascular parameters and self-reported affect. To control for the familywise error rate, Benjamini-Hochberg follow-up adjustments were conducted on tests with cardiovascular outcomes (Benjamini & Hochberg, 1995). To examine significant interactions, follow-up tests of simple effects were conducted.

To examine the type of cardiovascular response elicited hemodynamic profile and compensation deficit scores in response to each task were calculated (for method see; Gregg et al., 2002). Correlations were conducted to examine if trait emotion regulation was related to the underlying hemodynamic profile. To test if the hemodynamic response profile differed

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by task, depending on trait emotion regulation, a series of custom-built 2×1 ANCOVAs were conducted. The within-subjects factor was task (negative, neutral) and emotion regulation scores were entered as a covariate. The dependent variable was hemodynamic profile.

The challenge-threat index was calculated using the recommended approach (e.g., Blascovich et al., 2004). CO and TPR reactivity scores were standardized and weighted consistent with the literature (i.e., TPR scores given a weight of -1), and these scores were then summed. Similar to analyses using hemodynamic profile values, first, correlations were conducted to examine if emotion regulation indices were associated with challenge-threat responses to each task. Second, a series of ANCOVAs were conducted to examine if there were significant differences in the underlying responses to the negative and neutral task, depending on trait emotion regulation. Finally, to examine if hemodynamic profile and challenge-threat values were correlated Pearson's r correlation coefficients were conducted.

Preliminary Check

Participants spoke about an average of 9.25 words ($SD = 4.25$) during the negative-emotion task and an average of 8.75 words ($SD = 3.98$) during the neutral-emotion task. Paired samples t -tests confirmed that there were no differences in performance/effort between the negative- and neutral-emotion task, $t(47) = 1.26$, $p = .216$, 95% CI [-0.30, 1.30].

Stress Manipulation Check

Confirmation of cardiovascular responding

A series of repeated measures ANOVAs examined if both tasks elicited a significant physiological stress response. The within-subjects factor was phase (baseline, negative task, neutral task). The dependent variables were mean cardiovascular parameters at each phase. The analyses confirmed that SBP, DBP, HR, and CO increased from baseline to task for both tasks, all $ps < .011$. For TPR, there was no main effect of phase ($p = .118$). Pairwise comparisons found that there were no differences in cardiovascular responses between the

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two tasks, all $ps > .085$. Each task, regardless of emotion content, elicited a significant cardiovascular stress response. Table 1 reports the full results.

Psychological experience of the stressor task

There was a significant main effect of phase (baseline, negative task, neutral task) on positive affect. Pairwise comparisons confirmed that compared to baseline participants reported less positive affect after both tasks ($ps \leq .017$). However, there were no differences in post-task reported affect between the negative and neutral task. Furthermore, there was no significant main effect of phase for negative affect. Full results (with means and *SDs* for each phase) are presented in Table 1.

Emotion Regulation and Psychological Outcomes

There were no significant Phase $\times$ Reappraisal, Phase $\times$ Suppression, or Phase $\times$ Difficulties interactions for negative affect or positive affect, all $ps > .146$. The results suggest that individual differences in emotion regulation had no influence on self-reported affect during the laboratory session.

Emotion Regulation and Cardiovascular Outcomes

Reappraisal and Suppression

A 3×1 repeated measures ANCOVA, as described earlier, found a significant Phase $\times$ Reappraisal interaction effect for TPR ($p = .005$, $\eta_p^2 = .167$), which withstood the Benjamini-Hochberg adjustment. Follow-up tests of simple effects found that individuals scoring lower in reappraisal (below $-1SD$) demonstrated greater TPR responding during the negative task, $b = -0.27$, $t = -3.53$, $p = .001$, $[-0.42, -0.11]$, $r^2 = .237$, compared to individuals scoring higher in reappraisal ($+1SD$), $b = 0.05$, $t = 0.76$, $p = .482$, $[-0.10, 0.21]$, $r^2 = .012$, as shown in Figure 1. Reappraisal was not related to TPR responding during the neutral task, $b = .001$, $t = 0.45$, $p = .657$, $[-0.004, 0.007]$, $r^2 = .005$. There was no significant Phase $\times$ Reappraisal interaction effect for CO ($p = .077$), nor for SBP, DBP, or HR (all $ps > .118$), see Table 3 for full results.

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There was a significant Phase $\times$ Suppression interaction effect for SBP ($p = .016$, $\eta_p^2 = .106$) and DBP ($p = .007$, $\eta_p^2 = .127$); these effects withstood Benjamini-Hochberg adjustment. Simple effects analyses showed that individuals scoring higher in suppression (above $+1$ SD) showed the greatest blood pressure responses to both the negative task (SBP, $b = -16.99$, $t = -7.17$, $p < .001$, $[-21.78, -12.20]$, $r^2 = .562$; DBP, $b = -10.98$, $t = -10.32$, $p < .001$, $[-13.14, -8.83]$, $r^2 = .751$) and the neutral task (SBP, $b = -12.87$, $t = -6.48$, $p < .001$, $[-16.89, -8.86]$, $r^2 = .512$; DBP, $b = -9.03$, $t = -7.89$, $p < .001$, $[-11.35, -6.72]$, $r^2 = .609$). While individuals scoring lowest in suppression (below $-1SD$) had the lowest SBP and DBP responses to both the negative (SBP, $b = -8.49$, $t = -3.58$, $p < .001$, $[-13.28, -3.69]$, $r^2 = .243$; DBP, $b = -6.33$, $t = -5.95$, $p < .001$, $[-8.48, -4.18]$, $r^2 = .470$) and neutral task (SBP, $b = -6.49$, $t = -3.27$, $p = .002$, $[-10.50, -2.47]$, $r^2 = .211$; DBP, $b = -5.52$, $t = -4.82$, $p < .001$, $[-7.83, -3.20]$, $r^2 = .367$); as shown in Figure 2. There were no significant Phase $\times$ Suppression interaction effects for HR, CO, or TPR (all $ps > .269$). Table 3 displays the full results.

Emotion Regulation Difficulties

There was a significant Phase $\times$ Difficulties interaction effect for CO ($p = .014$) and TPR ($p = .027$). However, these effects did not withstand Benjamini-Hochberg adjustments. There were no significant Phase $\times$ Difficulties interaction effects for SBP, DBP, or HR (all $ps > .243$). Table 3 displays the full results. Overall, individual differences in emotion regulation difficulties did not influence blood pressure responses.

Overall, the results suggest that greater habitual use of suppression is associated with exaggerated blood pressure responding during stress (SBP and DBP), while lower habitual use of reappraisal is associated with greater TPR reactivity to the negative task. Emotion regulation difficulties were unrelated to cardiovascular responding during the task.

Sensitivity Analyses.

These analyses were repeated to include all three measures of emotion regulation

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(reappraisal, suppression, difficulties in emotion regulation) in the same model to examine if the pattern of observed results remained controlling for other indices of emotion regulation (i.e., the unique impact of each strategy, controlling for the others); this was confirmed and the results are presented in Table 4.

Examination of the Underlying Hemodynamic Profile

HP-CD model

Values for the hemodynamic profile and compensation deficit were computed in response to the negative- and neutral-emotion task (Gregg et al., 2002). One-sample *t*-tests were conducted to examine if these values differed from zero. A significant result for compensation deficit values indicates a change in blood pressure. A significant result for hemodynamic profile values indicates a vascular response pattern if the *t*-value is positive, and myocardial response if the *t*-value is negative. A significant compensation deficit result, but no significant hemodynamic profile result signals a mixed response. Both the negative and neutral tasks elicited a mixed pattern of hemodynamic responding: negative task, $t(47) = .64, p = .524, d = 0.09, 95\% \text{ CI } [-0.02, 0.04]$; neutral task, $t(47) = 1.21, p = .231, d = 0.18, [-0.01, 0.02]$. Both tasks elicited changes in blood pressure as compensation deficit values were significantly different from zero: negative task, $t(47) = 6.24, p < .001, d = 0.90, [0.03, 0.05]$; neutral task, $t(47) = 9.68, p < .001, d = 1.40, [0.02, 0.04]$.

Emotion regulation and HP-CD to each task. Greater habitual use of reappraisal was associated with a more myocardial response profile to the negative task ($r = -.418, p = .006$), but unrelated to hemodynamic responses to the neutral task ($p = .553$). Suppression was unrelated to hemodynamic responses to both tasks ($ps > .393$). Greater emotion regulation difficulties were associated with a more vascular response profile to the negative task ($r = +.313, p = .041$), and unrelated to hemodynamic responses during the neutral task ($p = .450$). Full results are reported in Table 2.

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Comparing HP to each task.

Reappraisal and Suppression. A series of 2×1 custom-built ANCOVAs examined if hemodynamic profile values to each task differed, depending on trait emotion regulation, as described earlier. There was a significant Phase $\times$ Reappraisal interaction effect on hemodynamic profile, $F(1, 40) = 7.44, p = .009$, partial $\eta^2 = .157$. Follow-up tests of simple effects found that lower habitual use of reappraisal ($-1SD$) was associated with a more vascular response to the negative task, $b = 0.43, t = 2.14, p = .038, [0.002, 0.083], r^2 = .103$, as shown in Figure 3, in comparison to higher use of reappraisal ($+1SD$), $b = -0.04, t = -1.74, p = .089, [-0.08, 0.01], r^2 = .070$. There was also a significant Phase $\times$ Reappraisal interaction effect on compensation deficit, $F(1, 40) = 7.50, p = .009$, partial $\eta^2 = .158$, where lower trait reappraisal ($-1SD$) was associated with more compensation deficit during the negative task, $b = 0.03, t = 3.27, p = .002, [0.01, 0.05], r^2 = .211$, compared to scoring higher in reappraisal ($+1SD$), $b = -0.01, t = -0.63, p = .533, [-0.03, 0.01], r^2 = .009$ (see Figure 3). Habitual suppression use did not influence hemodynamic profile, $F(1, 40) = 0.35, p = .853$, partial $\eta^2 = .001$, or compensation deficit, $F(1, 40) = 0.41, p = .840$, partial $\eta^2 = .001$.

Emotion Regulation Difficulties. There was a significant Phase $\times$ Difficulties interaction effect on hemodynamic profile, $F(1, 41) = 7.91, p = .008$, partial $\eta^2 = .162$. Follow-up tests of simple effects found that greater emotion regulation difficulties ($+1SD$) were associated with a more vascular response profile to the negative task, $b = 0.41, t = 2.09, p = .043, [0.001, 0.0813], r^2 = .098$, in comparison to less emotion regulation difficulties, $b = -0.04, t = -1.91, p = .062, [-0.078, 0.002], r^2 = .084$ (see Figure 4). There was no significant interaction effect on compensation deficit, $F(1, 41) = 2.93, p = .095$, partial $\eta^2 = .067$.

Challenge-threat Index

Emotion regulation and challenge-threat to each task. Greater habitual use of reappraisal was associated with a greater challenge response during the negative task ($r =$

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$+ .416, p = .006$) but unrelated to challenge-threat responding to the neutral task ($p = .556$).

Neither emotion regulation difficulties nor suppression were related to challenge-threat responses to either task ($ps > .051$). Full correlations are presented in Table 2.

Comparing challenge-threat responses to each task.

Reappraisal and Suppression. There was a significant Phase $\times$ Reappraisal interaction effect on the challenge-threat index, $F(1, 40) = 6.87, p = .012$, partial $\eta^2 = .147$. Figure 3 demonstrates this interaction, where it appears that lower habitual use of reappraisal was associated with a more threat-oriented response during the negative task. However, follow-up tests of simple effects were not significant [$-1SD: b = -0.54, t = -1.98, p = .055, [-1.09, 0.12], r^2 = .089$; $+1SD: b = 0.47, t = 1.75, p = .087, [-0.07, 1.03], r^2 = .071$]. There was no significant Phase $\times$ Trait Suppression interaction effect on challenge-threat responses, $F(1, 40) = 0.04, p = .850$, partial $\eta^2 = .001$.

Emotion Regulation Difficulties. There was a significant Phase $\times$ Difficulties interaction effect on challenge-threat scores, $F(1, 41) = 6.91, p = .012$, partial $\eta^2 = .144$. Figure 4 displays this interaction, where it appears that greater emotion regulation difficulties were associated with a more threat-oriented response during the negative-emotion task, compared to the neutral task; however, follow-up simple effects were not significant [$-1SD: b = -0.51, t = 1.89, p = .066, [-0.04, 1.06], r^2 = .080$; $+1SD: b = -0.50, t = -1.85, p = .071, [-1.06, 0.05], r^2 = .077$].

Results using the challenge-threat index somewhat mirror findings from the HP-CD computational model. There was a significant interaction for emotion regulation difficulties and reappraisal, whereby it appears that individuals with greater emotion regulation difficulties demonstrated a threat-oriented cardiovascular response during the negative emotion task, while lower habitual use of reappraisal was associated with a more vascular/threat response; however, follow-up tests of simple effects were not significant. Both

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models confirmed trait suppression was not associated with patterns of hemodynamic responding.

Comparing HP and CD values

Challenge-threat values were negatively correlated with hemodynamic profile in response to the negative ($r = -.964, p < .001$) and neutral task ($r = -.979, p < .001$), confirming that the HP-CD model and challenge-threat index share considerable statistical overlap.

Discussion

The present study confirmed that greater habitual use of suppression was associated with greater blood pressure reactivity to acute stress, while neither habitual use of reappraisal nor emotion regulation difficulties were associated with blood pressure reactivity. However, lower trait reappraisal was associated with a more maladaptive cardiovascular response pattern to the negative-emotion stressor, indexed by a vascular (threat-oriented) response. Emotion regulation difficulties were not significantly associated with CO or TPR responses; however, examination of the underlying hemodynamic response profile found that greater emotion regulation difficulties were associated with a vascular/threat oriented response to the negative-emotion task, as indexed by the HP-CD model. For clarity a synopsis of the main results are provided in Table 5. Individual differences in emotion regulation style did not influence the psychological experience of the stressor (i.e., self-reported affect). Overall, the present study provides evidence that individual differences in emotion regulation style are associated with distinct patterns of cardiovascular responding during active stress.

Consistent with research manipulating instructed suppression use (for reviews see; Gross, 2015; Mauss & Gross, 2004), this study confirmed that greater habitual use of suppression was associated with increased blood pressure responding to stress. Interestingly, this was observed in response to *both* the neutral-emotion and negative-emotion task. In light

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of research demonstrating exaggerated blood pressure reactivity to laboratory stressors is predictive of future cardiovascular disease, this suggests that the propensity to inhibit emotional responses during stress may confer a risk in terms of physical health. Furthermore, the observed differences in blood pressure were not attributable to task engagement/effort (refer to Supplementary Table S2 for these analyses) nor due to differences in experienced affect, thus strengthening the conclusion that differences in blood pressure reactivity were a result of habitual suppression.

An examination of the underlying hemodynamic profile showed that lower habitual use of reappraisal was associated with a vascular/threat-oriented response as indexed by the HP-CD model, but only during the *negative* task. The observed results somewhat differs from results of previous studies showing instructed reappraisal or scoring *higher* in reappraisal was associated with a challenge-oriented response (Jamieson et al., 2012; Jamieson, Nock, et al., 2013; Mauss et al., 2007), but show the same pattern of results. In the present sample we show that it was individuals who scored *lower* in habitual use of reappraisal that demonstrated a threat-oriented response, indicating that greater use of reappraisal protected against this response. Indeed, participants who had reappraisal scores around the mean and $1SD$ above the mean, showed a more myocardial/challenge-oriented response. In addition, our results tentatively suggest the effects of trait reappraisal may be limited, or indeed more pronounced during negative-emotion contexts. We tentatively speculate that perhaps use of reappraisal requires extra cognitive effort when stimuli relate to negative-emotion material (in this case talking about negative-emotion words), and as a result a challenge-oriented response is elicited; but this needs to be empirically tested by future research. It is notable that previous studies demonstrating a relationship between reappraisal and a cardiovascular challenge response employed tasks that could be considered negative, as throughout these tasks participants received negative feedback (Jamieson et al., 2012; Jamieson, Nock, et al.,

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2013; Mauss et al., 2007). Further research is needed to examine the boundary conditions under which reappraisal influences cardiovascular responding to stress.

The present study was the first to examine if individual differences in emotion regulation ability would affect blood pressure responding. Greater emotion regulation difficulties were associated with a vascular (threat-oriented) cardiovascular response during the *negative* emotion task, as indexed by the HP-CD model. Considering an active task was employed, a myocardial or mixed response would be expected (Carnevali et al., 2018; James & Gregg, 2004; Ottaviani et al., 2006); one representing a drive towards homeostasis (Obrist, 1981). Indeed, individuals reporting the least emotion regulation difficulties showed a myocardial/challenge response to this task. This homeostatic relationship was not demonstrated by individuals reporting greater difficulties regulating their emotions. This study presents preliminary evidence on the association between emotion regulation difficulties and a vascular cardiovascular response profile, and this warrants further attention. If future studies can replicate this finding, this may have implications for physical health; given vascular responsiveness has been linked to the development of cardiovascular disease (Mayet & Hughes, 2003; Sherwood & Turner, 1995).

Despite influencing patterns of cardiovascular responding, individual differences in emotion regulation style did not influence the emotional experience of the task, in terms of self-reported affect. In contrast, research involving the instructed use of reappraisal typically reports decreases in negative affect (Mauss et al., 2007; Ray et al., 2010). However, in such studies, participants are typically given explicit instructions such as *think about what you are seeing in such a way that you feel less negative emotion*, then are asked to report negative emotions following the task (e.g., Shiota & Levenson, 2009), potentially resulting in response bias. The present study overcomes this limitation; participants were given no instructions and engaged with the stressor naturally, then reported experienced affect. Indeed, this approach

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may offer a more valid method of examining emotion regulation strategy use and self-reported affect during stress. However, these conclusions must be considered in light of the small sample size employed; future research needs to examine this with a larger sample size to determine if trait emotion regulation is associated with affective responses during stress.

Although the results were in the expected directions and the sample size was sufficient to detect medium-to-large effects, there is the possibility of type I error due to the number of analyses conducted and the relatively small sample size employed. However, Benjamini-Hochberg adjustments were implemented to reduce this possibility. Likewise, the observed results should be interpreted with caution, considering the instability of point estimates of effect sizes in smaller samples (Schönbrodt & Perugini, 2013). It should also be noted that the sample employed was homogenous in terms of age, ethnicity, and education status. While this was intentional to avoid potential confounding variables on cardiovascular responses, it also limits the generalisability of our results to other age cohorts and cultures. These findings need to be replicated in a larger, and more diverse, sample.

Furthermore, the observed results must be interpreted with respect to the design employed. While the focus on individual differences in emotion regulation may increase external validity compared to artificially manipulating emotion regulation in the laboratory, we cannot infer causality. Indeed, this approach assumes that people actually engage in the emotion regulation strategies they report using in day-to-day life (i.e., via self-report scales) when faced with stress, and furthermore used these strategies in response to the speech tasks in this study. We, however, cannot be certain of the degree to which participants used these strategies during the stress task. Furthermore, the Emotion Regulation Questionnaire (Gross & John, 2003) does not delineate which type of reappraisal it assesses; rather it appears to focus on habitual active attempts to try to change cognitions. This may reflect attempts to change thoughts regarding the situation or it may reflect attempts to rethink one's emotional

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response to it, or both, depending on the context.

While the results of this study are consistent with research showing reappraisal and suppression to be associated with positive and negative outcomes respectively, we must be careful not to posit one emotion regulation strategy as truly “adaptive” or “maladaptive”. For instance, in certain contexts (e.g., if a situation is controllable) it may be better to take effective action than reappraise one’s emotions (e.g., Haines et al., 2016; Troy et al., 2013). Similarly, use of suppression can be adaptive, for example to maintain positive functional relationships (e.g., Le Bonnie & Impett, 2013). Considering this, future research should consider the degree to which an individual can switch between regulation strategies flexibly to best meet the demands of the situation, as greater emotion regulation flexibility (for reviews see; Aldao et al., 2015; Bonanno & Burton, 2013) is associated with greater well-being and positive adjustment (e.g., Westphal et al., 2010).

The observed results are strengthened by the employment of a standardised laboratory stress paradigm (acclimatization, baseline, and task periods) and the inclusion of an active stress task. Speech tasks have been shown to reliably elicit cardiovascular responses in the laboratory, and the magnitude of this response is similar to that observed during stress encounters in day-to-day life, assessed via ambulatory monitoring (Johnston et al., 2008). This offers an advantage, in terms of the ecological validity of the stress experience, over some past emotion regulation research where the focus tends to be on passive stressors, such as emotion-eliciting film clips or negative imagery. We also demonstrated that the effects of reappraisal and suppression on cardiovascular reactivity held when controlling for the effects of other indices of emotion regulation, showing the unique predictive ability of reappraisal on TPR responding and suppression on blood pressure responding. Furthermore, patterns of CO and TPR responding were quantified using both the HP-CD model and the challenge-threat index; confirming the type of cardiovascular elicited. We highlight that both computations

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(the HP-CD model and the challenge-threat index) are highly correlated, providing further support for the hypothesis that these indices appear to capture the compensatory relationship between CO and TPR in similar ways (Griffin & Howard, 2021). It is recommended that future research apply either of these computations to confirm the type of response elicited. However, it is worth noting that we did not measure pre-ejection period (PEP) in the present study. PEP is thought to be a direct measure of sympathetic nervous system influence on the heart, and sympathetic activation is necessary feature of both challenge and threat states (Blascovich & Mendes, 2010). A decrease in PEP would confirm engagement with the task; as such, future research could assess PEP in order to strengthen the observed results.

The present study provides evidence that individual differences in emotion regulation are associated with distinct patterns of cardiovascular responding to stress. We found clear patterns of exaggerated blood pressure responses for high trait suppressors to both tasks, and a vascular cardiovascular response pattern for low trait reappraisers (albeit only in response to the negative task). The observed results strengthen the findings of past research that instruct the use of these strategies, and tentatively suggest a potential role of context. Individual differences in emotion regulation style have long been associated with psychological well-being, and this study shows that when faced with an acute stressor habitual emotion regulation correlates with cardiovascular responses in the laboratory. Our results would suggest that use of reappraisal and suppression may be protective and harmful, respectively, in terms of physiological responses to stress, but this is with the assumption habitual use of these strategies does indeed reflect their use in stressful situations. In terms of generalising these effects, the present results offer preliminary evidence in this regard; additional studies replicating the observed effects are needed.

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Table 1.

Mean (with SDs) cardiovascular parameters and self-reported affect at each phase, with ANOVA results testing differences across the phases (baseline, negative task, neutral task).

	Phase								
	Baseline		Negative Task		Neutral Task				
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>F</i>	η_p^2	
SBP (mmHg)	117.94	9.96	130.99	16.74	128.47	15.56	49.09	.511	<.001**
DBP (mmHg)	69.43	6.89	78.38	9.06	77.30	9.29	89.81	.656	<.001**
HR (bpm)	74.71	10.17	79.23	9.57	78.78	9.66	30.93	.397	<.001**
CO (lpm)	6.24	1.65	6.58	1.86	6.43	1.61	5.36	.102	.011*
TPR (pru)	0.92	0.25	1.01	0.40	0.97	0.26	2.47	.050	.118
Positive affect	32.08	7.31	30.19	7.70	30.08	7.94	5.48	.104	.006*
Negative affect	13.58	4.54	14.60	4.51	14.06	4.79	1.34	.028	.266

Note. SBP, DBP, HR, and CO significantly increased from baseline to each task. TPR did not change. Positive affect decreased after both tasks compared to baseline values. *Note.* ** $p < .001$, * $p < .05$

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Table 2.

Correlations between main study variables

	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1. Reappraisal	-.074	-.170	-.038	-.027	+.294	+.311*	-.426**	-.418*	+.416*	+.288	+.150	+.121	+.105	-.070	-.094	+.094
2. Suppression		+.055	+.370*	+.437*	-.011	+.205	-.064	-.098	+.105	+.336*	+.323*	+.186	+.309*	-.018	-.135	+.155
3. DERS			-.152	-.168	-.136	-.312*	+.314*	+.313*	-.300	-.137	-.231	-.067	+.143	-.123	-.118	+.141
Negative Task																
4. SBP				+.915*	.194	+.452*	-.011	-.121	+.079	+.739	+.706*	-.056	+.197	+.135	+.036	+.014
5. DBP					+.268	+.349*	+.074	-.010	-.013	+.731*	+.776*	+.043	+.220	+.251	+.108	-.049
6. HR						+.555*	-.230	-.451*	+.446*	+.357*	+.390*	+.557*	+.305*	-.067	-.243	+.194
7. CO							-.563*	-.824*	+.724*	+.451*	+.398*	+.128	+.340*	-.201	-.295*	+.297*
8. TPR								+.900*	-.879*	-.247	-.191	-.186	-.253	+.152	+.242	-.223
9. HP									-.964*	-.292*	-.217	-.219	-.368*	+.294*	+.407*	-.370*
10. CT-Index										+.271	+.212	+.244	+.351*	-.219	-.387*	+.318*
Neutral Task																
11. SBP											+.940	+.235	+.413*	+.169	-.047	+.099
12. DBP												+.306*	+.315*	+.357*	+.112	-.067
13. HR													+.568*	-.224	-.440*	+.414*
14. CO														-.618*	-.839*	+.871*
15. TPR															+.910*	-.924*
16. HP																-.979*
17. CT-Index																

Note. DERS = Difficulties in Emotion Regulation, HP = Hemodynamic Profile, CT-Index = scores on the challenge-threat index. SBP, DBP, HR, CO, and TPR refers to calculated reactivity to the task (mean task values minus mean baseline values). * $p < .05$

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Table 3.

Phase × Trait ER interaction effects on cardiovascular reactivity (separate models for each emotion regulation measure)

	Reappraisal			Suppression			DERS		
	<i>F</i>	η_p^2	<i>p</i>	<i>F</i>	η_p^2	<i>p</i>	<i>F</i>	η_p^2	<i>P</i>
SBP	1.77	.042	.186	4.75	.106	.016*	0.72	.017	.472
DBP	0.95	.023	.372	5.81	.127	.007**	1.44	.034	.243
HR	2.20	.052	.118	0.92	.022	.915	0.45	.011	.639
CO	2.85	.066	.077	1.34	.032	.269	5.05	.110	.012*
TPR	7.99	.167	.005**	0.14	.004	.738	4.89	.107	.027*

Note. DERS = Difficulties in Emotion Regulation. ** $p < .01$, * $p < .05$. For ANCOVAs, mean cardiovascular parameters at each phase were used.

Table 4.

Phase × Trait ER interaction effects on cardiovascular reactivity (one model)

	Reappraisal			Suppression			DERS		
	<i>F</i>	η_p^2	<i>p</i>	<i>F</i>	η_p^2	<i>P</i>	<i>F</i>	η_p^2	<i>P</i>
SBP	2.22	.057	.116	4.96	.118	.015*	1.07	.028	.336
DBP	1.20	.031	.301	6.03	.140	.007**	1.58	.041	.216
HR	2.32	.059	.107	0.92	.024	.403	0.16	.004	.852
CO	2.80	.070	.080	1.95	.050	.159	4.47	.108	.022*
TPR	8.25	.182	.004**	0.62	.017	.461	3.85	.094	.049*

Note. The results presented reflect one model for each cardiovascular parameter. Reappraisal, suppression, and DERS were entered into one model to examine the Phase × Emotion regulation interactions on each cardiovascular parameter. ** $p < .01$, * $p < .05$

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Figure 1.

Lower habitual use of reappraisal was associated with greater TPR responding to the negative task, and unrelated to CO responding.

Figure 2.

Lower habitual use of suppression was associated with lower SBP and DBP responding to both tasks, while greater habitual use of suppression was associated with greater SBP and DBP responding to both tasks.

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Figure 3.

Individuals scoring low in habitual reappraisal demonstrated a vascular (threat-oriented) response to the negative task as indexed by the HP-CD model (HP). Low trait reappraisers exhibited more compensation deficit (CD) during the negative task compared to the neutral task.

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Figure 4.

Individuals with greater difficulties in emotion regulation demonstrated a more vascular (threat-oriented) response during the negative task compared to the neutral task as indexed by the HP-CD model (HP). There were no differences in compensation deficit (CD).

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Table 5.

Summary of key findings

	Reappraisal		Suppression		DERS	
	<i>Sig.</i>	Direction	<i>Sig.</i>	Direction	<i>Sig.</i>	Direction
SBP	×		✓	Higher suppression = greater SBP to both tasks	×	
DBP	×		✓	Higher suppression = greater DBP to both tasks	×	
HR	×		×		×	
CO	×		×		×	
TPR	✓	Lower reappraisal = greater TPR during the negative task	×		×	
HP	✓	Lower reappraisal = more vascular response during negative task	×		✓	Greater DERS = more vascular response during the negative task
Challenge-Threat	✓*	Lower reappraisal = more threat-oriented response during negative task	×		✓*	Greater DERS = more threat-oriented response during the negative task

Note. *Sig.* refers to a significant test result. Results for SBP, DBP, HR, CO, and TPR refer to the ANCOVA analyses. Results for hemodynamic profile (HP) and the challenge-threat index refer to ANCOVAs testing the difference between the negative and neutral tasks. *Indicates a significant interaction, however, follow-up tests of simple effects were not significant.

Author Note

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