EMOTION REGULATION STYLE AND CARDIOVASCULAR REACTIVITY TO
ACTIVE AND PASSIVE STRESS:
CROSS-SECTIONAL AND EXPERIMENTAL ANALYSES

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Abstract

Emotion Regulation Style and Cardiovascular Reactivity to Active and Passive Stress: Cross-Sectional and Experimental Analyses

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Introduction. The present thesis examines the influence of individual differences in emotion regulation style on stress responsivity. Three key methodological refinements were incorporated to help elucidate if individual differences in emotion regulation influences cardiovascular reactivity (CVR) to psychological stress. First, a standardised laboratory stress paradigm was employed, yielding more reliable assessment of CVR; emotion regulation research rarely incorporates a valid resting baseline measurement. Second, more sophisticated indices of physiological arousal were assessed, such as blood pressure, cardiac output, and total peripheral resistance; emotion regulation research typically uses skin conductance responses as an index of physiological responding. Third, this research examined the underlying patterns of hemodynamic responding. Methods. Five empirical studies are reported. Study 1 examined associations between habitual emotion regulation style, perceived stress, and psychological well-being in a sample of 170 members of the general population. Study 2 reported on the influence of trait emotion regulation style on CVR to an active stress task in the laboratory in a sample of 48 young adults. Study 3 tested the construct validity of a novel passive stress task, in a prospective study of 25 young adults. In the same sample, Study 4 examined emotion regulation style and CVR to this passive stress task. Study 5 manipulated the use of instructed reappraisal and examined the effects of reappraisal on cardiovascular adaptation to recurrent stress in a sample of 139 young adults. Results. Study 1 confirmed greater use of suppression, and greater emotion dysregulation, were associated with greater perceived stress and poorer psychological health. Study 2 found trait emotion regulation style influenced CVR during active stress; reappraisal was associated with a more healthful response, while suppression and difficulties in emotion regulation were associated with a less adaptive response. Study 3 confirmed the construct validity of a novel passive task. Study 4 demonstrated that individual differences in emotion regulation style did not influence CVR during passive stress. Study 5 found instructed reappraisal did not influence cardiovascular adaptation to recurrent stress. Conclusion. The present results provide evidence that emotion regulation style results in altered patterns of CVR, which in the long-term may have consequences for physical health. However, the results suggest that instructed use of a healthful strategy, reappraisal, in the laboratory does not help adaption to recurrent stress. Rather, trait emotion regulation style is the important determinant in predicting how emotional regulation influences stress responsivity.
Declaration

The substance of this thesis is the original work of the author, and due reference and acknowledgement has been made, where necessary, to the work of others. No part of this thesis has been submitted in candidature of any other degree.

_____________________________________

Siobhán Griffin (Candidate)
For my family
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List of Abbreviations

ANCOVA: Analysis of covariance

ANEW: Affective Norms for English Words

ANOVA: Analysis of variance

bpm: beats per minute

CD: Compensation deficit

CHD: Coronary Heart Disease

CI: Confidence Intervals

CO: Cardiac Output

CRP: C-reactive protein

CVD: Cardiovascular Disease

CVR: Cardiovascular Reactivity (to psychological stress)

DBP: Diastolic blood pressure

DERS: Difficulties in Emotion Regulation Scale

ER: Emotion Regulation

ERQ: Emotion Regulation Questionnaire

HADS: Hospital Anxiety and Depression Scale

HP: Hemodynamic profile

HPA: Hypothalamic-Pituitary-Adrenal
HR: Heart rate

HRV: Heart rate variability

lpm: Litres per minute

LVM: Left ventricular mass

MAP: Mean arterial pressure

MI: Myocardial Infarction

mmHg: Millimetres of mercury

NA: Negative Affect

PA: Positive Affect

PANAS: Positive Negative Affect Schedule

pru: Peripheral resistance units

PSS: Perceived Stress Scale

SAM: Sympatho-adrenal-medullary

SBP: Systolic blood pressure

SNS: Sympathetic Nervous System

SWLS: Satisfaction with Life Scale

TPR: Total peripheral resistance

TSST: Trier Social Stress Task
1. Emotion Regulation: Effects on Health

1.1. Definition of Emotion Regulation

Emotion regulation refers to attempts made by individuals, whether consciously or unconsciously, to influence which emotions are experienced, when they are experienced, and how they are expressed; this may involve up-regulation (increasing the emotional response), down-regulation (decreasing the emotional response), or maintenance of the emotional response (Gross, 1998a; Richards & Gross, 2000). It is almost impossible to clearly define what constitutes an emotion, or what does not (Gross & Barrett, 2011). For the purposes of this research “emotion” refers to an umbrella term of affect. This relates to all the psychological states that involve an evaluation process, including stress responses and discrete emotions, such as sadness, anger, and happiness (Gross, 2015). Different research perspectives agree that emotion involves changes in an array of domains, including subjective experience, behaviour, and physiology (Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005).

Effective emotion regulation does not necessarily mean an ability to diminish or avoid negative emotional experiences, but rather relates to the ability to function adaptively when presented with experiences that elicit intense emotional responses.

1 The content of this chapter has been published, in part, as a book chapter in Stress and Anxiety - Contributions of the STAR Award Winners (Griffin & Howard, 2019).
(Pollock, McCabe, Southard, & Zeigler-Hill, 2016). When individuals respond to emotional stimuli in problematic ways, for example, experiencing emotions as overwhelming, they are argued to have difficulties in regulating their emotions (Gratz & Roemer, 2004). Emotion regulation difficulties, and use of more “maladaptive” emotion regulation strategies, are associated with negative emotional, cognitive, and social consequences for the individual, and in the long-term have been implicated in the development of psychological and physical ill-health (e.g., Aldao & Nolen-Hoeksema, 2010; Campbell-Sills & Barlow, 2007; Eisenberg, Hofer, & Vaughan, 2007; Gross & John, 2003; Gross & Levenson, 1997; Pennebaker, 1997; Richardson, 2017). Research in this area, therefore, has predominantly focused on identifying which emotion regulation strategies allow for effective emotion regulation.

The affective and physiological consequences of two emotion regulation strategies, reappraisal and suppression, have been the focus of much research (e.g., Gross, 1998a, 1999, 2002, 2013, 2014; Gross, 2015; Gross & Thompson, 2007; Schafer, Naumann, Holmes, Tuschen-Caffier, & Samson, 2017). Reappraisal involves reinterpreting a situation in order to alter the emotional response, for example looking at the positive aspects of a situation to reduce negative emotion. Whereas, suppression involves consciously inhibiting the behavioural expressions of the emotional response. Habitual use of these strategies is captured through self-report. The Emotion Regulation Questionnaire (ERQ; Gross & John, 2003) assesses the degree to which individuals habitually engage in reappraisal and suppression. Other research manipulates the use of these strategies in the laboratory; participants are instructed to employ either reappraisal or suppression techniques when presented with an emotion-eliciting situation, and the
consequences of this on self-reported emotion and/or indices of sympathetic nervous system (SNS) activity are examined.

1.2. Theoretical Perspectives on Emotion Regulation

1.2.1. The process model of emotion regulation. The most influential model of emotion regulation, the process model, describes the strategies of reappraisal and suppression in detail (Gross, 1998a). This theoretical framework classifies emotion regulation strategies based on when they have their primary impact in the emotion generation process; before the emotion has been fully elicited (antecedent strategies) or after the emotion has been experienced (response-focused). The model distinguishes between five “families” of emotion regulation strategies, with the temporal nature of each strategy hypothesised to impact the behavioural, experiential, and physiological response tendencies (e.g., Gross, 1998b; Gross, 1999). Antecedent strategies include situation selection (e.g., avoid certain situations/people, find a friend when feeling upset), situation modification (e.g., ask for help, change something in the environment), attentional deployment (e.g., focus attention elsewhere, distraction), and cognitive strategies (e.g., reappraisal). Response-focused strategies include response modulation, for example, suppression of behavioural expression of emotions, or using drugs/exercise/relaxation to modify the emotional experience.

All emotional states are argued to begin with an evaluation of internal or external cues (real or imagined); and this appraisal results in a coordinated set of behavioural, experiential, and physiological emotional response tendencies that either serve to meet the emotion regulation goal or not (e.g., Gross, 1998a, 1999, 2013). Figure 1.1. demonstrates this process, and highlights that response tendencies serve to increase, decrease, or maintain the subsequent response. This feedback system of response and
evaluation is reciprocal and continuous during the emotional response and results in the final experiential, behavioural, and physiological responses to the stimulus (Gross & Thompson, 2007).

Figure 1.1. The process model of emotion regulation. Response tendencies increase (+) decrease (-), or do not change as a result of emotion regulation (adapted from Gross, 1998a).

1.2.2. The extended process model of emotion regulation. The extended process model of emotion regulation (Gross, 2015), unsurprisingly, builds on the process model but underlines the dynamic nature of evaluation systems, and separates these systems into three stages: (i) the identification stage (identify the need to regulate or not); (ii) the selection stage (how to regulate); and (iii) the implementation stage (implementing a strategy to alter the emotional response), as displayed in Figure 1.2. Each of these stages is thought to elicit their own valuation system. At the most basic
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level, a valuation system identifies if the core feature of affect is good or bad. Following this, a valuation takes place where the representation of the external (or internal) world is compared against a representation of the desired “world” or state of affect. This evaluation leads to action impulses that aim to address the discrepancies between the perceived state and desired state. These actions can refer to mental processes such as reframing, or to physical processes, such as the activation of the SNS. The extended process model highlights that these action tendencies may in turn change the representation of the world, prompting another valuation and further action tendencies. It is hypothesised that the process of emotion regulation is a second-level valuation system which acts on another valuation system, the first-level valuation system, which is what gives rise to the emotion.

The extended process model (Gross, 2015) essentially still rests on the premise that emotion regulation occurs through the five families of emotion regulation strategies outlined in the process model. These families are argued to be the five ways in which emotion regulation can target the first-level evaluation system; changing the situation, changing an aspect of the “world”, changing the target of attention, altering the cognitive representations of the world, or modifying emotion-related outcomes. However, by highlighting the dynamic nature of emotion regulation, and outlining the three valuation systems that take place (identification, selection, and implementation) this extended model allows further understanding of how emotion dysregulation may occur, and when it may occur. For example, a lack of emotional awareness at the identification stage has implications for the selection and implementation stages. Individuals differ in their emotional awareness (Taylor, 1994), and if individuals are not aware of what they are feeling this can result in problems determining if, and how, an emotion is regulated.
Likewise, at the identification stage pre-held beliefs about what constitutes a “good” or “bad” emotion can have consequences for emotion regulation. Emotion dysregulation may also arise during the selection stage, in particular, if people have low self-efficacy in their ability to adequately respond to emotion-eliciting or stressful experiences, such that they perceive lower ability to access and use effective emotion regulation strategies (Gratz & Roemer, 2004). Also, the actual choice of strategy can matter – in some situations a certain emotion regulation strategy might be helpful, in others it may be harmful. For example, individuals with social anxiety might avoid certain social situations (situation selection), while in the short-term this may mitigate unpleasant affect, in the long-term it serves to create further anxiety in anticipation of social situations. Similarly, use of suppression may hide emotional responses from others, but in the long-term suppression is associated with a range of negative health outcomes (e.g., Gross, 2013). While the model cannot capture all the intricacies of emotional responding it serves as a basis for understanding how emotional responses begin, are regulated, and finally manifest through the use of different emotion regulation strategies. It also highlights mechanisms through which emotion dysregulation occurs and influences the effectiveness of regulatory attempts.

1.2.3. Emotion regulation difficulties. Other research advocates for looking beyond “adaptive” and “maladaptive” strategies, as suggested by the process model, and rather examine deficits in emotion regulation. This approach conceptualises the different facets involved in successful emotion regulation, with an emphasis on awareness and understanding of emotions, as well as the ability to monitor, evaluate, and modify the emotional experience. Gratz and Roemer (2004) proposed six emotional regulation
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Figure 1.2. The extended process model of emotion regulation. Emotion regulation acts on a first stage valuation system (V), and distinguishes three stages; (a) identification, (b) selection, and (c) implementation. This may change the first level valuation system, which can then influence action tendencies and the perceived world (W). These stages extend over time and are linked (taken from Gross, 2015).

competencies; (i) acceptance of emotional responses, (ii) impulse control over thoughts and behaviours, (iii) goal-directed behaviour, (iv) emotional awareness, (v) emotional clarity, and (vi) perceived access to effective emotion regulation strategies. Deficits in any of these competencies, when distressed, is believed to signify emotion regulation difficulties. Individual differences in these competencies can be measured using the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004). This measure
offers an advantage over previous assessments of emotion dysregulation such as the Negative Mood Regulation Scale (Catanzaro & Mearns, 1990) and Trait Meta-Mood Scale (Salovey, Mayer, Goldman, Turvey, & Palfai, 1995). These scales measure individual differences in the avoidance and elimination of negative emotion, as opposed to assessing the ability to manage emotional responses. The DERS therefore allows assessment of individual difficulties in awareness and understanding of emotions, acceptance of negative emotions, as well as the ability to act in desired ways when experiencing negative emotions. In particular, this individual difference measure is employed extensively in clinical populations to elucidate the relationship between emotion regulation ability and psychological health; typically difficulties in emotion regulation are associated with indices of psychological distress, such as anxiety, depression, eating disordered behaviour, and addiction (e.g., Fowler et al., 2014; Fox, Hong, & Sinha, 2008; Gratz, 2007; Racine & Wildes, 2013; Salters-Pedneault, Roemer, Tull, Rucker, & Mennin, 2006; Weiss et al., 2012).

Few studies have directly examined the relationship between emotion regulation difficulties and habitual use of reappraisal/suppression. Use of reappraisal has been found to predict lower difficulties in emotion regulation, while use of suppression predicts greater difficulties in emotion regulation (Jelvani, Etemadi, Jazayeri, & Fatehizade, 2018). Similarly, a study examining the predictors of suicide risk, reported a positive association between suppression and emotion regulation difficulties, and a negative correlation between difficulties and trait reappraisal (Ghorbani, Khosravani, Bastan, & Ardakani, 2017); both lower habitual use of reappraisal and greater difficulties in engaging in goal-directed behaviour predicted suicide risk. These studies provide support for the hypothesis that reappraisal is a more “adaptive” strategy and that suppression is a
more “maladaptive” strategy; habitual use of these strategies is associated with lower and greater emotion regulation difficulties, respectively. Importantly, the DERS assesses aspects of emotion regulation competencies not quite captured by trait reappraisal and trait suppression scales.

1.3. Emotion Regulation and Stress

The importance of valuation systems, and specifically the role of reappraisal is emphasised in the modified transactional model of stress (Folkman, 1997). Responses to stress are not uniform across individuals, rather people show individual variability in the psychological and physiological consequences of stress exposure (Lazarus & Folkman, 1987). Similar to the extended process model of emotion regulation, the transactional model of stress posits that stressful stimuli undergo an evaluation process, and as a result of this process the stressor is either appraised as a threat (the individual does not have the necessary resources to meet the demands of the stressor), or as a challenge (the individual has the resources to meet the demands). These “resources” include individual differences in personality, social support, as well as the contextual elements of the situation (Lazarus & Folkman, 1987). If resources do not meet the demands of the stressor a threat response is activated. However, if stress-eliciting situations are reappraised as not “stressful” or non-threatening, it is expected that this will be reflected in the affective and physiological responses to the stressor. Reappraisal is therefore hypothesised to buffer the negative effects of stress by changing the experience of the stressor and therefore resulting in lower negative affect (NA), greater positive affect (PA), and lower physiological arousal. Additionally, as reappraisal occurs early-on in the emotion generation process it is theorised to require less resources and therefore result in a greater ability to modulate the emotional response (Gross, 1998b; Kim & Hamann,
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2012). In contrast, use of suppression does little to alleviate any negative emotions associated with stress. Indeed, as suppression is typically initiated after the stress response has been elicited, it is hypothesised that the effort of inhibiting behavioural expressions of this response results in greater activation of the SNS.

When responses to stress are overwhelming or inappropriate it can have a multitude of negative consequences for the individual, including poorer psychological and physical health (for reviews see; Chida & Steptoe, 2010; Lupien, McEwen, Gunnar, & Heim, 2009; Suls & Bunde, 2005). Emotion regulation ability may help elucidate the relationship between stress responsivity and future health outcomes. In particular, recent research has speculated that individual differences in emotion regulation may have implications for physical health; through both psychological mechanisms (the experience of negative and positive emotions) and physiological mechanisms (via activation of stress-response systems).

Prospective research provides evidence linking emotion regulation with elevated cardiovascular disease (CVD) risk. Individuals with poorer emotion regulation ability (as measured using items from the Minnesota Multiphasic Personality Inventory) were more likely to have suffered a non-fatal myocardial infarction (MI) at a 12-year follow-up, or died as a result of coronary heart disease (CHD), after controlling for well-known CHD risk-factors (Kubzansky, Park, Peterson, Vokonas, & Sparrow, 2011). Furthermore, research directly assessing individual differences in suppression and reappraisal, using the ERQ, have reported a relationship between habitual emotion regulation use and levels of C-reactive protein (CRP); an inflammatory marker of CHD (e.g., Danesh et al., 2004). Greater habitual use of reappraisal was associated with lower CRP levels, while
suppression was associated with higher CRP levels (Appleton, Buka, Loucks, Gilman, & Kubzansky, 2013).

1.4. Psychological Outcomes of Emotion Regulation

1.4.1. Reappraisal and suppression. Individual differences in the propensity to engage in reappraisal and suppression correlate with a range of psychological outcomes, in particular NA, PA, depression, and anxiety. Trait reappraisal has been associated with greater PA, greater satisfaction with social support, lower NA, less perceived stress, lower anxiety, and lower depressive symptoms (e.g., Balzarotti, Chiarella, & Ciceri, 2017; Balzarotti, John, & Gross, 2010; Chervovsky & Hunt, 2018; Gross & John, 2003; Lam, Dickerson, Zoccola, & Zaldívar, 2009; Miklósi, Martos, Szabó, Kocsis-Bogár, & Forintos, 2014). In contrast, greater habitual use of suppression correlates with greater NA, greater depressive symptoms, lower PA, and less satisfaction with social support (Appleton et al., 2013; Chervovsky & Hunt, 2018; Fresco et al., 2007; J. Johnson et al., 2016; Nolen-Hoeksema & Aldao, 2011; Richardson, 2017; Rogier, Garofalo, & Velotti, 2017). Further evidence stems from studies employing experience sampling methods, ranging from 1-3 weeks. Greater trait reappraisal is associated with more PA and less NA, while trait suppression demonstrates the opposite pattern (Brans, Koval, Verduyn, Lim, & Kuppens, 2013; Nezlek & Kuppens, 2008). A number of meta-analyses have confirmed the association between reappraisal and psychological health, and the association between suppression and indices of psychopathology (Aldao & Nolen-Hoeksema, 2010; Schafer et al., 2017). Habitual use of reappraisal is generally associated with better indices of psychological well-being cross-sectionally and longitudinally, while suppression use demonstrates an inverse relationship with psychological health (Dennis, 2007; Fresco et al., 2007; Garnefski & Kraaij, 2006; Gross & John, 2003; John
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& Gross, 2004; Kraaij, Pruymboom, & Garnefski, 2002; Ng, Huebner, Hills, & Valois, 2018; Nolen-Hoeksema & Aldao, 2011; Rogier et al., 2017). Furthermore, studies employing daily diary reports have found that during times of stress, trait reappraisers report less NA, while trait suppressors report less PA (J. Johnson et al., 2016; Richardson, 2017). Evidence from such studies provides support that individual differences in the use of these emotion regulation strategies has consequences for experienced affect and wellbeing, on a day-to-day basis, as well as in the long-term.

Experimental designs highlight the influence of each of these strategies on short-term affective experiences. Instructed reappraisal to a range of passive stressors (e.g., film-clip stimuli, negative imagery) and active stressors (e.g., speech tasks, mental arithmetic) generally support the cross-sectional findings. Participants instructed to use reappraisal have reported less unpleasant emotions when viewing negative imagery (e.g., Dillon, Ritchey, Johnson, & LaBar, 2007; Urry, 2001), lower distress, sadness, fear, and disgust across a range of emotional film-clip stimuli (Gross, 1998b; Gruber, Hay, & Gross, 2014; McRae, Ciesielski, & Gross, 2012; Richards & Gross, 2000; Shiota & Levenson, 2009; Troy, Shallcross, Brunner, Friedman, & Jones, 2018; Wolgast, Lundh, & Viborg, 2011), and greater PA and lower NA after engaging in a social interaction task compared to uninstructed conditions (Butler, Wilhelm, & Gross, 2006). In contrast, instructed suppression results in little or no change in reported affect compared to control conditions. For example, no differences have been found between suppression and control conditions in self-reported negative emotions after viewing negative imagery and sad or distressing film-clips (Dillon et al., 2007; Dunn, Billotti, Murphy, & Dalgleish, 2009; Richards & Gross, 1999; Shiota & Levenson, 2009). Although in response to a social interaction task, instructed suppression lead to lower PA post-task, compared to the
uninstructed group (Butler et al., 2006). Shiota and Levenson (2009) found instructed suppression led to reduced feelings of disgust after a disgust eliciting film-clip; however, others have reported no effect (Gross, 1998b; N. A. Roberts, Levenson, & Gross, 2008). Taken together, cross-sectional and experimental research support the hypothesis that reappraisal may buffer the negative affective consequences of stress. However, contrary to cross-sectional findings which demonstrate greater habitual use of suppression is associated with higher levels of NA, experimental manipulations of this strategy have little or no influence on affective responding during acute stress. This may be a result of self-report bias; individuals were instructed to “feel nothing” or “hide their emotional responses” which may have influenced self-reported affect. Assessment of individual differences, using the ERQ, can overcome this potential bias and help elucidate how the tendency to suppress expressive behaviour during stressful situations influences positive and negative emotions.

1.4.2. Difficulties in emotion regulation. Greater difficulties in emotion regulation have been linked to a number of psychological health outcomes. Indeed, emotion dysregulation is implicated as a central component of several types of psychopathology (Gross & Thompson, 2007; Kring & Sloan, 2010). Greater difficulties in emotion regulation are associated with more binge-eating behaviours in a non-clinical sample (Whiteside et al., 2007), greater frequency of purging and exercise in people diagnosed with bulimia nervosa (Lavender et al., 2014), and greater eating disorder cognitions in a sample of patients diagnosed with anorexia nervosa (Racine & Wildes, 2013). Individuals who presently engaged in self-harm (non-suicidal self-injury) reported more difficulties in emotion regulation, relative to people who previously have engaged in self-harm (but no longer do so) or who have never engaged in self-harm (Anderson &
Crowther, 2012). In a sample of patients diagnosed with a serious mental illness (major depressive disorder, anxiety disorder, psychotic spectrum disorder, or personality disorder) greater emotion regulation difficulties were associated with greater depression and anxiety severity, as well as more somatic complaints (Fowler et al., 2014). In non-clinical samples, greater difficulties in emotion regulation are associated with greater perceived stress (Roy, Riley, & Sinha, 2018), greater NA (Pollock et al., 2016; Salsman & Linehan, 2012), and higher scores on measures of depression, chronic worry, and anxiety (e.g., Allan, Norr, Macatee, Gajewska, & Schmidt, 2015; Bardeen, Fergus, & Orcutt, 2012; Ritschel, Tone, Schoemann, & Lim, 2015; Roemer et al., 2009; Ruganci & Gencoz, 2010; Salters-Pedneault et al., 2006). The reported findings suggest emotion regulation deficits may play a role in the development of psychological ill-health.

1.5. Physiological outcomes of emotion regulation

1.5.1. Reappraisal and suppression. Laboratory studies which assess physiological arousal mirror results from research examining the affective consequences of emotion regulation; in general, instructed reappraisal leads to healthful patterns of cardiovascular responding, while suppression is associated with exaggerated cardiovascular reactivity (CVR) during acute stress. Early evidence for this stems from a representative study by Lazarus and Alfert (1964). Participants were shown a film-clip of a circumcision, but the soundtrack was manipulated. The group who heard a soundtrack where the pain involved was minimised and the celebratory aspects were emphasised, exhibited significantly lower heart rate (HR) and lower skin conductance responding, and reported more pleasant mood, compared to participants hearing the technical aspects of the procedure (emphasising pain and the unsanitary conditions). This was one of the first studies to demonstrate how use of cognitive strategies during a stress-eliciting situation
can influence both psychological and physiological responses; reframing the procedure as something positive altered the emotional and physiological stress response.

In more recent years, instructed reappraisal has been found to result in lower skin conductance responding to a range of emotion-eliciting film-clips (Gruber et al., 2014; Wolgast et al., 2011). Conversely, instructed use of suppression leads to: heightened skin conductance responses and blood pressure responses during film-clip stimuli (e.g., Gross, 1998b; Harris, 2001; N. A. Roberts et al., 2008); greater HR reactivity during a speech task (Hofmann, Heering, Sawyer, & Asnaani, 2009); and exaggerated blood pressure reactivity during mental arithmetic, cold pressor, and social interaction tasks (Butler et al., 2003; Quartana & Burns, 2010). This highlights a potential pathway by which suppression can contribute to CHD risk; repeated activation of the sympathetic system over time can result in arterial damage due sheer stress, heightening the risk of hypertension and atherosclerosis (e.g., Clarkson, Manuck, & Kaplan, 1986). It is worth noting that some studies have reported no relationship between instructed emotion regulation and skin conductance responding during passive stress (Dunn et al., 2009; Gross, 1998b; Kim & Hamann, 2012); however, this may be due to differences in physiological measurements, as well as task differences, in terms of the emotional nature of the stressor and the type of coping context elicited by the task (e.g., Bolli, Amann, Hulthen, Kiowski, & Buhler, 1981; Gross & Levenson, 1993; Obrist, 1981; Obrist, Light, McCubbin, Hutcheson, & Hoffer, 1979; Saab et al., 1993; Sherwood, Allen, Obrist, & Langer, 1986). Overall, reported findings have generally corroborated the argument that reappraisal can be considered an adaptive strategy; instructions to use reappraisal are associated with lower physiological arousal, which may offer a protective function in terms of physical health. While use of instructed suppression is associated with
exaggerated physiological arousal, which may have implications for long-term physical health.

Examining individual differences in the natural tendency to engage in these strategies can offer more insight into the longer-term psychophysiological implications of reappraisal and suppression use. However, few studies have taken the individual difference approach and only a handful include CVR to stressors as an outcome. Research assessing both trait emotion regulation and CVR has largely focussed on reappraisal and has provided evidence that reappraisal moderates cardiovascular responding. For example, in response to a speech task higher habitual use of reappraisal was associated with lower blood pressure reactivity; however, trait suppression had no influence on blood pressure (Memedovic, Grisham, Denson, & Moulds, 2010). Furthermore, individuals scoring high in reappraisal demonstrated a challenge-orientated cardiovascular stress response, as opposed to a physiological threat response, during a mental arithmetic stressor (Mauss, Cook, Cheng, & Gross, 2007). This mirrors results from research that experimentally manipulates reappraisal use; participants instructed to reappraise arousal as adaptive when completing the Trier Social Stress Task (TSST; a mental arithmetic task and speech task) demonstrated a challenge-oriented cardiovascular stress response, compared to individuals instructed to ignore feelings of stress, or given no instructions (Jamieson, Nock, & Mendes, 2012, 2013). To our knowledge, no research has examined the relationship between suppression and the type of stress response elicited (threat or challenge) in response to active stress.

1.5.2. Emotion regulation difficulties. To date no research has directly examined the potential influence of emotion regulation difficulties on blood pressure responses to acute stress. A few studies have examined if there are differences in cardiovascular
parameters, at rest, between individuals reporting high levels of emotion regulation difficulties, and less difficulties. For example, Roy et al. (2018) assessed the relationship between emotion regulation difficulties and resting blood pressure and found no differences in resting blood pressure between individuals scoring high and low in emotion regulation difficulties. Other research has focused on resting Heart Rate Variability (HRV); a proposed marker of emotion regulation capacity, with lower HRV believed to indicate a more inflexible parasympathetic nervous system (Appelhans & Luecken, 2006; Thayer, Ahs, Fredrikson, Sollers, & Wager, 2012). Results have been mixed; some research has reported greater emotion regulation difficulties are associated with lower resting HRV (Visted et al., 2017; Williams et al., 2015), while other research did not find this relationship (Stalder, Evans, Hucklebridge, & Clow, 2011).

In terms of cortisol, emotion regulation difficulties have been found to moderate the relationship between higher levels of childhood maltreatment and blunted cortisol responding to stress (England-Mason et al., 2017), and the relationship between higher levels of early life stress and blunted cortisol responding (Carnuta, Crisan, Vulturar, Opre, & Miu, 2015). In both studies, only individuals scoring high in emotion regulation difficulties (who had experienced such adversity) demonstrated this altered pattern of cortisol responding; individuals who reported less emotion regulation difficulties, but also reported high levels of childhood maltreatment or early life stress did not demonstrate this response. This suggests that emotion regulation difficulties may be implicated in dysregulated hypothalamic-pituitary-adrenal (HPA) axis activity. There is also evidence stemming from studies examining inflammation and CVD risk as outcomes that suggests emotion regulation difficulties may play a role in stress responsivity. Cross-sectionally, greater emotion regulation difficulties have been
associated with a known marker of inflammation - CRP (Powers et al., 2016).

Furthermore, in a sample of middle-aged adults, chronic stress was associated with
greater CVD risk (indexed by greater resting blood pressure, body mass index, and
insulin resistance) only in individuals scoring high in emotion regulation difficulties
(Roy et al., 2018). Individuals scoring low in emotion regulation difficulties did not
demonstrate an association between chronic stress and CVD risk. These findings,
although cross-sectional, suggest a role of emotion regulation ability in buffering the
physiological impact of chronic stress. It therefore seems pertinent to examine, in a
controlled laboratory environment, the role emotion regulation difficulties may play, if
any, in CVR to acute stress.

1.6. Mechanisms linking Emotion Regulation with Physical Health

An abundance of research has described the association between CVD risk and
habitually experiencing greater levels of NA (for a review see; Suls & Bunde, 2005).

More recently research has also highlighted the potential protective influence of positive
emotions (for reviews see; Boehm & Kubzansky, 2012; Folkman, 2008; Pressman,
Jenkins, & Moskowitz, 2019). In particular, the impact of emotion regulation on affect
(short-term episodes of emotions) is important as affective experiences are believed to
contribute to longer-term affective states (moods). Indeed, the more NA or PA
experienced by an individual is predictive of future affect (e.g., Heady & Wearing, 1989;
Pettersson, Boker, Watson, Clark, & Tellegen, 2013).

One mechanism believed to link emotional, and stress, responses with CVD is
exaggerated cardiovascular responses to stressors (Kibler & Ma, 2004). The well-
established cardiovascular reactivity hypothesis implicates exaggerated or prolonged
CVR to stressors as a risk-factor for CVD; a finding supported by numerous longitudinal
and cross-sectional studies (for a review see; Chida & Steptoe, 2010). In these studies, those that had heightened reactions to acute psychological stress in a laboratory setting had, at a 10-15 year follow-up, higher resting blood pressure (Carroll et al., 2001; Light, Dolan, Davis, & Sherwood, 1992), a greater incidence of hypertension (Carroll et al., 2001; Tuomisto, Majahalme, Kahonen, Fredrikson, & Turjanmaa, 2005), and stroke (Everson et al., 2001).

Considering that reappraisal is associated with lower NA, lower psychological symptoms, and more healthful CVR to acute stress, it is hypothesised that this strategy may have health protective benefits. In contrast, the links between suppression and emotion regulation difficulties with poorer psychological outcomes and exaggerated CVR suggest a relationship between maladaptive emotion regulation and poorer health outcomes. The next section will discuss the theory behind the CVR hypothesis and studies evaluating the CVR hypothesis. It is hypothesised that individual differences in emotion regulation may help elucidate the pathway underlying the relationship between stress responsivity and CVD.

2. The Cardiovascular System and the Stress Response

2.1. Theoretical Models of Stress

2.1.1. Fight or flight. Physiological responses to stress or threats in the environment are believed to be first described by Cannon (1929b) in a response known as the “fight-or-flight” response, stemming from an evolutionary perspective. When presented with a threat it is hypothesised that the body prepares for sustained physical effort by increasing HR, pumping blood to the muscles, releasing hormones such as norepinephrine and epinephrine, and slowing down other processes such as digestion.
The fight-or-flight response is thought to be necessary in order to mount an adaptive behavioural response to survival threats. However, in today’s society where survival threats may be not be as dominant, and daily stressors may take the form of more psychological demands (e.g., work conflict, traffic congestion) such processes have been argued to initiate disease mechanisms, leading to diseases such as hypertension and atherosclerosis. It is argued that such a physiological response is in excess of typical daily demands, and thus harmful for health (Obrist, 1981; J. R. Turner, 1994).

2.1.2. Homeostasis and allostasis. It should be noted that this physiological response is not thought to initiate disease when it occurs in an efficient and not too frequent manner; this optimal state of functioning allows organisms to adapt to the demands of their environment (Sterling, 2004). Such bodily processes are referred to as allostasis; achieving stability/homeostasis through changes in the autonomic nervous system, the HPA axis, as well as through cardiovascular, immune, and metabolic processes (McEwen, 1998). These systems are activated in order to respond to the environment and support behaviours necessary for survival, such as eating and sleeping. In the short-term, allostasis is adaptive, and necessary; however, it may lead to a maladaptive state known as allostatic load (McEwen, 1998; McEwen & Stellar, 1993), as shown in Figure 1.3. Excessive activation of these systems is thought to impair regulatory processes, resulting in a progressive failure to maintain homeostasis (normal operating ranges) leading to damaging side-effects. Over time biological systems compensate for the over, or under, reaction of other systems, eventually resulting in physiological dysregulation leading to disease states due to chronic wear and tear (Juster, McEwen, & Lupien, 2010). Therefore, homeostatic regulation can result in both healthful and health-damaging outcomes.
Allostatic load may occur in a number of ways, for example through frequent exposure to stress (chronic stress), an inability to habituate to reoccurring stressors, delays in recovery from stress, or exaggerated stress responses (McEwen & Seeman, 1999). The underlying principles of this model are widely accepted; in particular, the importance of normal operating ranges, and the damaging effects of both over and under activation of these systems. Large longitudinal studies assessing a range of bio-markers (e.g., interleukin-6, resting blood pressure, CRP, cholesterol, fasting glucose levels), have provided substantial evidence for the link between heightened allostatic load and all-cause mortality (e.g., Goldman, Turra, Glei, Lin, & Weinstein, 2006; Goldman, Turra, Glei, Seplaki, et al., 2006; Gruenewald, Seeman, Ryff, Karlamangla, & Singer, 2006; Karlamangla, Singer, & Seeman, 2006), as well as greater incidence of CVD events (e.g., Sabbah, Watt, Sheiham, & Tsakos, 2008; Seeman, McEwen, Rowe, & Singer, 2001).

2.1.2.1. Homeostasis within the cardiovascular system. The cardiovascular system is one of the bodily systems that must maintain homeostasis through allostatic processes. Neuro-hormonal activity results in changes in blood pressure depending on the body’s needs and environmental requirements. When presented with a stimulus, or a change, the cardiovascular system must maintain adequate blood pressure. If blood pressure rises, the heart becomes inhibited and there is less vasoconstrictor nerve activity. If blood pressure falls, the heart becomes stimulated to increase heart rate and cardiac output (CO; blood flow), there is also a compensatory decrease in vasoconstrictor activity in order to lower peripheral blood flow. These processes help maintain blood pressure within a normal range.

In particular, blood pressure is often used as a proxy for cardiovascular health and is the focus of much research (e.g., Lovallo, 2010, 2015; Phillips & Hughes, 2011). This
may be due to the temporal stability of blood pressure responses to psychological stressors (e.g., Hassellund, Flaa, Sandvik, Kjeldsen, & Rostrup, 2010). Blood pressure refers to the force of the blood against arterial walls during a cardiac cycle. The cycle has two phases; contraction and relaxation. Contraction of the heart forces blood into the circulatory system, and the maximum period of this force (pressure) is known as systolic blood pressure (SBP). During relaxation the ventricles refill, and the lowest pressure during this period is referred to as diastolic blood pressure (DBP). Blood pressure above 140mmHg (SBP) and 90mmHg (DBP) is generally classified as hypertensive, according to the guidelines set out by the World Health Organisation and the National Blood

Figure 1.3. The development of allostatic load. A range of factors influence an individual’s perception of stress (such as genes, environment); this perception results in behavioural, physiological, and affective responses, leading to allostatic and adaptation. However, over time allostatic load can accumulate, and have negative implications for physical health (from McEwen, 1998; McEwen & Seeman, 1999).
Pressure Education Program (Chobanian et al., 2003; Whitworth & Chalmers, 2004; WHO & ISH, 2003).

2.2. Physiological responses to stress

When a situation is perceived as stressful, a cascade of co-ordinated physiological responses is elicited, for example, the activation of the autonomic sympatho-adrenal-medullary (SAM) axis and the HPA axis; as demonstrated in Figure 1.4. The SNS is immediately activated, stimulating the release of epinephrine and norepinephrine from the adrenal medulla (the SAM system), and stimulating the HPA axis. The SAM system has a more immediate effect compared to the HPA axis as it is activated by nerve impulses that stimulate the adrenal medulla. The release of norepinephrine results in lower blood flow to some organs, such as those in the gastrointestinal track and kidneys, whereas the epinephrine increases blood flow to areas such as the brain, heart, and muscles (resulting in increases in HR, blood pressure, glucose release), allowing an effective fight-or-flight response. The release of catecholamines, and their effects on blood pressure and HR allow an objective assessment of the stress response (for a review see; Klein & Corwin, 2002). Typically, SAM activity is assessed non-invasively via salivary enzyme α-amylase (for a review see; Granger et al., 2016). Activation of the HPA axis on the other hand is assessed via cortisol. Approximately, 15-20 minutes after the onset of a stressor cortisol levels rise; the stressor elicits the release of corticotrophin-releasing hormone (CRH) which stimulates the pituitary gland; as a result, adrenocorticotrophin hormone (ACTH) and arginine vasopressin are released. Adrenocorticotrophin circulates to the adrenal glands stimulating the release of corticosteroids, including cortisol. This creates a negative feedback loop whereby the increase in cortisol hinders the production of more ACTH (for reviews see; Klein &
Corwin, 2002; Kudielka & Kirschbaum, 2005). While HPA activation is adaptive in the short-term, in the long-term prolonged activation can lead to dysregulation of the feedback loop and have detrimental effects on health (e.g., Libby, Ridker, & Maseri, 2002; McEwen, 1998; Sapolsky, 2000).

2.3. The Cardiovascular Reactivity Hypothesis

Individual differences exist in the magnitude of the physiological stress response elicited, depending in part on a person’s appraisal of the stimulus, as eluded to earlier under the extended transactional model of stress (Folkman, 1997; Lazarus & Folkman, 1987). The same stressful situation may elicit quite a large stress response in one person (e.g., greater blood pressure, increases in HR) but have little effect on another person. As mentioned previously, the CVR hypothesis proposes that individuals who exhibit persistent and exaggerated cardiovascular responses to stress are at risk of negative physical health outcomes. Likewise, consistent with the principles of homeostatic regulation (McEwen, 1998; McEwen & Seeman, 1999), individuals who exhibit CVR below normal operating ranges are thought to be at risk of negative health outcomes. Indeed, lower or blunted CVR to stress has been linked to a range of negative health outcomes including depression, obesity, and eating disorders (Carroll, Phillips, & Der, 2008; Carroll, Phillips, Hunt, & Der, 2007; de Rooij, 2013; Ginty, Phillips, Higgs, Heaney, & Carroll, 2012; Phillips, 2011).

Exaggerated CVR, in particular, has been highlighted as a risk factor for the development of CVD (e.g., Blascovich & Katkin, 1993; Lovallo, 2005, 2015; Manuck, 1994; Obrist, 1981). CVD refers to a number of different cardiac conditions including CHD, coronary artery disease, artherosclerosis, heart attacks, stroke, and ischemic heart disease (Labarthe, 2011). Exaggerated CVR is hypothesised to lead to the development
Figure 1.4. Schematic overview of the stress response system (adapted from Murison, 2016, page 33). Dashed lines represent inhibitory pathways. BNST = bed nucleus of the stria terminalis.
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of CVD through a number of mechanisms. It is argued that exaggerated or persistent reactivity to stress results in wear and tear of the body’s organs and vasculature; this elevated reactivity contributes to long-term changes in autoregulation (overperfusion of skeletal muscle), resulting in greater arterial resistance and long-term changes in resting blood pressure (e.g., Carroll, 1992). Over time this heightened pressure results in hypertrophy of the vascular muscles (Folkow & Neil, 1971; Obrist, 1981). Such structural abnormalities are believed to encourage the deposit of arterial plaque on the vessel walls (Treiber et al., 2003). Additionally, excessive CVR is believed to lead to structural and functional changes in neuro-hormonal systems which influence cardiovascular responding (brain and immune system), as well as the cardiac system (Manuck, 1994; McEwen, 2000). A combination of structural changes and autoregulation are argued to link CVR to CVD due to associated cardiac and vascular hypertrophy (Lovallo, 2005; Lovallo & Gerin, 2003).

One way to study the relationship between CVR to stress and future CVD risk is to measure cardiovascular responses to laboratory stressors and then observe incidence of CVD longitudinally. CVR is generally calculated by subtracting resting blood pressure from blood pressure responding measured during a stress task. It is assumed that laboratory stressors will increase SNS activation, and the magnitude of this response is important in elucidating the pathogenesis of CVD (Krantz & Manuck, 1984). In fact, a range of prospective studies have demonstrated the utility of CVR in predicting future disease risk (for a review see; Lovallo, 2005; Treiber et al., 2003). Indeed, exaggerated CVR to acute stress (stress encountered in the laboratory/short-term stress) has been identified as great a risk factor for CVD as elevated cholesterol levels (Dimsdale, 2008).
2.2.1. Evidence for the CVR hypothesis. Longitudinal studies have provided evidence for the predictive ability of CVR to acute stress on cardiac health outcomes, including elevated resting blood pressure, atherosclerosis, increased left ventricular mass (LVM), and incidence of cardiac events, such as MI and stroke.

2.2.1.1. Predicting future elevated blood pressure and hypertension. Elevated resting blood pressure is a major risk factor for the development of hypertension and CHD (e.g., Fiebach et al., 1989; Stamler, Stamler, & Neaton, 1993; Whelton, 1994). Even heightened blood pressure within the normative range (<140/90, SBP/DBP) has been associated with increased risk of CVD (Kshirsagar, Carpenter, Bang, Wyatt, & Colindres, 2006; Vasan et al., 2001). Indeed, it has been argued that reducing resting blood pressure is key in CVD management (Czernichow et al., 2011).

Evidence has demonstrated that CVR to stressors predicts subsequent increases in blood pressure and future hypertension status. Early research focused on CVR to the cold pressor task and risk of hypertension (Hines, 1940; Hines & Brown, 1936). During the cold pressor task participants are asked to immerse a body part in cold water for a period of time; the magnitude of the cardiovascular response to this task has been found to predict hypertension status in adolescence and adult samples, with follow-up periods ranging from 20 to 45 years (Kasagi, Akahoshi, & Shimaoka, 1995; Menkes et al., 1989; Wood, Sheps, Elveback, & Schirger, 1984). Similarly, blood pressure reactivity to the cold pressor task predicted subsequent future resting blood pressure in child and adolescence samples, with 1-year and 5-year follow-ups (Malpass et al., 1997; Treiber et al., 1994; Treiber, Turner, Davis, & Strong, 1997). However, other research has found no relationship, or only a weak relationship between CVR to the cold pressor task and subsequent elevated resting blood pressure or hypertension development (Armstrong &
Research employing psychological stress tasks provide more convincing evidence for the association between CVR and future CVD risk. These tasks rely on cognitive effort and active coping, as opposed to requiring physical effort or eliciting pain (as done in the cold pressor task). Psychological tasks are thought to induce states that are more similar to those experienced in everyday life, thus are argued to be more important in elucidating mechanisms of diseases (Light et al., 1992). Other research has argued that the cold pressor task has suboptimal ability to predict the long-term consequences of stress on the cardiovascular system (Fredrikson & Matthews, 1990; Manuck, Kasprowicz, & Muldoon, 1990). In one of the CARDIA studies, which examined CVR to a video-game stressor, mirror-image tracing task, and the cold pressor task, only SBP reactivity to the video-game stressor (psychological stressor) was predictive of future resting SBP at a five-year follow-up in men (Markovitz, Raczynski, Wallace, Chettur, & Chesney, 1998). Using a subset of this data (a US-based sample) Matthews, Woodall, and Allen (1993) examined the predictive ability of a psychological stressor (mental arithmetic task) and physical stressor (isometric handgrip task) on resting blood pressure 6.5 years later. In both adults and children SBP and DBP reactivity to the mental arithmetic task was more successful as a predictor of subsequent resting blood pressure. Similarly, Del Rosario, Treiber, Harshfield, Davis, and Strong (1998) found SBP responding to a video game stressor explained an additional 4% of the variance in ambulatory SBP 2.5 years later, while responses to the cold pressor task did not predict future blood pressure. The results of this research suggest that CVR to psychological
stressors is a more reliable predictor of subsequent CVD risk than responses to physical stressors.

Indeed, research employing a range of psychological stressors have provided evidence for the utility of CVR in predicting future health outcomes. The results of a meta-analysis of 36 studies examining CVR to psychological stress demonstrated that exaggerated CVR was associated with more adverse future health outcomes, including elevated resting blood pressure, hypertension, left ventricular mass, and clinical cardiac events (Chida & Steptoe, 2010). In terms of future resting blood pressure, prospective research using multiple psychological stressors found blood pressure reactivity in middle-aged men was predictive of resting blood pressure at a 5-year follow-up (Manolio et al., 1994) and a 9-12 year follow-up (Tuomisto et al., 2005). Light et al. (1992) found individuals exhibiting exaggerated CVR to a reaction time task were more likely to have elevated resting blood pressure 10.5 years later. Likewise, blood pressure reactivity to mental stress (matrix problems) was positively correlated with resting blood pressure 5 years later (Carroll, Smith, Sheffield, Shipley, & Marmot, 1995) and 10 years later (Carroll et al., 2001). It is worth noting that in the latter cohort, only modest support for the CVR hypothesis was found; blood pressure reactivity to the task accounted for less than 1% of the variance in future blood pressure.

Research examining hypertension as an outcome has reported similar findings when using psychological stressors. Tuomisto et al. (2005) found that blood pressure responses to a series of psychological tasks, as well as the cold pressor task, were predictive of hypertension status at a 9 to 12 year follow-up; however, CVR to the psychological stressors were better predictors of hypertension development than the cold pressor task. Individuals demonstrating exaggerated blood pressure responding to a range
of stressors, including a mental arithmetic task (Falkner, Onesti, & Hamstra, 1981), an exercise challenge (Manolio et al., 1994), and in anticipation of an exercise challenge (Everson et al., 1997), were more likely to have developed hypertension 4-5 years later. Similar to research examining future resting blood pressure as an outcome, associations between CVR and subsequent hypertension development are often modest. For example, research from the CARDIA study found CVR to a problem-solving task had minimal predictive value regarding future incidence of hypertension development at a 5- and 10-year follow-up (Carroll et al., 1995; Carroll et al., 2001).

2.2.1.2. Prediction of other pre-clinical disease states. Research focusing on other cardiac health outcomes such as carotid atherosclerosis and LVM have provided support for the CVR hypothesis. Increased LVM is a well-established risk factor for CVD and left ventricular hypertrophy, and has been linked to cardiovascular morbidity as a result of MI, arrhythmia, stroke, and congestive heart failure (e.g., Bikkina et al., 1994; Kannel, 1999; Kannel, Doyle, McNamara, Quickeenton, & Gordon, 1975; Levy, Garrison, Savage, Kannel, & Castelli, 1989). Despite this only a few studies have examined CVR as a predictor of future LVM. In a sample of borderline hypertensive adults, Georgiades, Lemne, DeFaire, Lindvall, and Fredrikson (1997) found that although aggregated SBP and DBP reactivity to a mental arithmetic and isometric muscle contraction task were related to LVM at a three-year follow-up, only mean arterial blood pressure was predictive of future LVM, explaining 15% of the variance in LVM. In two independent samples of children and adolescents with a family history of hypertension, blood pressure reactivity to a range of tasks was related to future LVM at a 2.6 year follow-up (Murdison et al., 1998) and 2.3 year follow-up (Kapuku et al., 1991). However, when
controlling for other risk factors of LVM, CVR was only predictive of future LVM in the study by Murdison et al. (1998).

Likewise, only a handful of studies have examined the predictive role of CVR in plaque development, incidence of atherosclerosis (a build-up of plaque in the body’s arteries), and incidence of carotid atherosclerosis (plaque build-up specifically in the carotid arteries), but research is generally in support of the CVR hypothesis. For example, Barnett, Spence, Manuck, and Jennings (1997) found SBP reactivity to the Stroop Colour Word Interference Task was the best predictor of plaque thickness at a 2-year follow-up, explaining 7% of the variance. Matthews et al. (1998) found pulse pressure reactivity (increases in the magnitude between SBP and DBP responding) to a public speaking task and mirror tracing task predicted future carotid plaque thickness. Other studies have found support for the predictive ability of SBP reactivity on carotid artery disease, but only in combination with other factors such as greater workplace demands (Everson et al., 1997) or lower socio-economic status (Lynch, Everson, Kaplan, Salonen, & Salonen, 1998).

Other research has found evidence linking CVR to coronary calcification at a 13 year follow-up (Matthews, Zhu, Tucker, & Whooley, 2006); an established predictor of CVD events and mortality (e.g., Detrano, Wong, Doherty, & Shavelle, 1997; Pletcher, Tice, Pignone, & Browner, 2004). Everson et al. (2001) found middle-aged men demonstrating exaggerated SBP in anticipation of an exercise test had 72% greater risk of stroke and 87% greater risk of ischemic stroke compared to less reactive men, even after adjusting for known risk factors for stroke.

2.2.2. Reliability of CVR. Cardiovascular responses to laboratory stressors are argued to reflect responses to stressful experiences encountered in day-to-day life. It is
therefore assumed that the same person would demonstrate a similar magnitude of CVR to tasks over time, and in response to a range of stressors. Empirical evidence suggests this is the case, and has demonstrated the temporal stability of cardiovascular responses over time and across tasks (e.g., Kelsey, Ornduff, & Alpert, 2007; Smith & O'Keefe, 1988; J. R. Turner, 1994). Likewise, an underlying assumption of the CVR hypothesis is that CVR observed in the laboratory relates to cardiovascular responses to more naturalistic stressors, and to cardiovascular measures attained through ambulatory blood pressure monitoring. Indeed, individuals with greater SBP reactivity to laboratory tasks also showed greater SBP responding during times of high daily stress, as assessed by ambulatory blood pressure (Kamarck, Schwartz, Janicki, Shiffman, & Raynor, 2003). Similarly, blood pressure reactivity to a range of tasks was associated with CVR to in-class presentations (e.g., Kamarck, Debski, & Manuck, 2000; J. R. Turner, Girdler, Sherwood, & Light, 1990). However, other research has found weak correlations between laboratory CVR and CVR in real life settings (e.g., Pickering & Gerin, 1990; J. R. Turner et al., 1994).

2.2.3. Extending the reactivity hypothesis. The CVR hypothesis has been extended in recent years, whereby the focus is no longer on simply examining, and reporting, patterns of exaggerated reactivity to a single stressor. A number of developments have occurred due to some criticisms of the traditional CVR hypothesis, such as the modest relationship between CVR to laboratory stress and daily stressors (for reviews see; Kamarck & Lovallo, 2003; Linden, Gerin, & Davidson, 2003; Schwartz et al., 2003; Zanstra & Johnston, 2011), and the modest associations between blood pressure reactivity and subsequent future resting blood pressure (Carroll et al., 1995; Carroll et al., 2001). Therefore, to elucidate the relationship between CVR and future
disease risk the CVR hypothesis has been extended to include a second stress exposure; this is thought to reflect habituation-sensitization to reoccurring stress, one of the markers of disease risk identified by McEwen (1998). Likewise, research has acknowledged, and provided evidence for, the associations between blunted CVR and health outcomes (for reviews see; Carroll, 2011; Phillips, Ginty, & Hughes, 2013). Furthermore, it is hypothesized that an examination of the underlying determinants of blood pressure responding, CO and total peripheral resistance (TPR), can improve on the modest associations between stress reactivity and cardiac health outcomes (Kamarck & Lovallo, 2003; Manuck et al., 1990; Sherwood et al., 1986).

2.3. Habituation-Sensitization

While the theory of allostatic load implicates exaggerated CVR in the development of disease states, it also posits that an inability to habituate to reoccurring stress contributes to disease risk (e.g., Kelsey, 1993; McEwen & Seeman, 1999). By extending the traditional CVR laboratory protocol to include a second stress exposure it allows for examination of habituation-sensitization patterns to recurrent stress. Indeed, cardiovascular responding to recurrent stress is thought to be more reflective of responses to stress outside of the laboratory; as daily stressors are typically reoccurring, whereas laboratory tasks tend to be novel.

A recent review of studies which involved a habituation paradigm identified four types of stress responders, shown in Figure 1.5.; persistent reactors, persistent blunters, habituators, and sensitizers (Hughes, Lu, & Howard, 2018). Persistent reactors are individuals who demonstrate a cardiovascular response to the first stress exposure and exhibit a similar magnitude of CVR to the second task. In contrast, blunters are individuals who fail to mount a stress response to the first and second task. Habituators
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exhibit a marked increase in CVR to the first task and in response to the second task demonstrate lower CVR (in comparison to the first task). Sensitizers exhibit a cardiovascular response to the first task, but then exhibit a much higher response to the second task. These typologies may elucidate differences in CVD risk that would not be observable when only CVR to one stress exposure is considered. For example, habituators are hypothesized to demonstrate an optimal stress response in terms of physical health (Dienstbier, 1989). These individuals successfully mount a stress response, and this dissipates on the second exposure; suggesting adaptation to reoccurring stress. In this way the body prepares to cope with stress (e.g., increased blood flow to the muscles, preparation of the fight-or-flight response, increased immune function), and this response decreases when the stressor is experienced again, as such the cardiovascular system does not experience prolonged activation. In contrast, a sensitized response is thought to indicate compromised coping ability (Howard & Hughes, 2013; Hughes et al., 2018), which overtime may result in damage to the cardiovascular system (e.g., Kelsey, 1993; McEwen & Stellar, 1993).

Evidence from laboratory studies has accumulated to demonstrate how higher scores on certain “adaptive” personality traits such as extraversion, resilience, and openness are associated with more pronounced habituation to repeated stress (Lu & Wang, 2017; Lu, Wang, & Hughes, 2016; Lu, Wang, & You, 2016). On the other hand, a number of more “maladaptive” psychological traits have been associated with sensitization or a lack of habituation, for example trait dominance (Lee & Hughes, 2014), rumination (J. A. Johnson, Lavoie, Bacon, Carlson, & Campbell, 2012), stress vulnerability (O’Súilleabháin, Hughes, Oommen, Joshi, & Cunningham, 2019), and Type D personality (Howard & Hughes, 2013). These patterns of responding are not
observable when only CVR to the first stress exposure is considered, underlining the importance of examining patterns of habituation-sensitization in CVR research.

![Figure 1.5.](image)

**Figure 1.5.** The proposed typologies of stress responders; persistent reactors, persistent blunter, habituators, or sensitizers (taken from Hughes et al., 2018).

### 2.4. Hemodynamic Profile of Responses

The way in which the two parameters underlying blood pressure responding, CO and TPR, respond is known as the hemodynamic profile. Cardiac output refers to the number of litres of blood pumped through the arterial system per minute, while TPR refers to the resistance on arterial blood flow as a result of vessel dilation or constriction. These parameters have a compensatory relationship. As CO increases the arterial wall must allow extra blood to flow, thus the pressure on the artery wall is reduced, marked by a reduction in TPR. In contrast, when blood flow is lower (lower CO), TPR should increase to constrict the vessels in order to create an adequate amount of elasticity for blood flow velocity. As one parameter increases, the other should decrease (compensate).
The importance of examining CO and TPR becomes apparent when examining blood pressure responses. Individuals may demonstrate similar blood pressure responses (similar SBP and DBP reactivity to a task); however, these individuals may be exhibiting different hemodynamic responses; myocardial, vascular, or mixed (e.g., Light & Sherwood, 1989; Light, Turner, Hinderliter, & Sherwood, 1993; Manuck et al., 1990). Typically, an increase in CO accompanied by a decrease in TPR is described as a myocardial response and is a result of β-adrenergic activation. Myocardial responses are typically exhibited in response to active stress tasks, such as speech tasks and mental arithmetic tasks. When TPR increases and CO decreases, alpha-adrenergic activation occurs; this is known as a vascular response and is hypothesised to be elicited by passive coping contexts, such as the cold pressor task. A mixed response occurs when a synergistic increase or decrease in TPR and CO occurs.

Early research used a variety of methods to classify individual differences in hemodynamic responses, often employing arbitrary cut-offs which categorised responders as myocardial or vascular (Eliot, Buell, & Dembroski, 1982; Girdler, Turner, Sherwood, & Light, 1990; Sherwood, Dolan, & Light, 1990). However, this method has received a number of criticisms (for a review see; Gregg, Matyas, & James, 2002). First, by changing continuous measures of CO and TPR into categorical variables (myocardial or vascular responders) the quality of the data was reduced. Second, a group of responders who did not qualify as myocardial or vascular were often excluded from analyses; essentially lost data. Third, early computations did not take the reciprocal relationship between CO and TPR into consideration. Furthermore, these computations confounded HP (hemodynamic profile) values and compensation deficit (CD) values. HP refers to the way in which CO and TPR compensate (whether CO predominates, TPR
predominates, or both respond equally) and CD refers to the degree to which they compensate (Gregg et al., 2002).

To address such limitations Gregg et al. (2002) developed the hemodynamic-profile compensation-deficit (HP-CD) model, shown in Figure 1.6. This orthogonal model computes continuous measures for HP and CD; extending on research which previously categorised people into either vascular or myocardial responders and excluded individuals who did not fit these groups. The validity of this model has been confirmed by a number of studies (e.g., Howard, Hughes, & James, 2011; Hughes, Howard, James, & Higgins, 2011; James & Gregg, 2004; James, Gregg, Matyas, Hughes, & Howard, 2012; O'Leary, Howard, Hughes, & James, 2013; Ottaviani, Shapiro, Goldstein, James, & Weiss, 2006; Ottaviani, Shapiro, Goldstein, & Mills, 2007).

2.4.1. Hemodynamic profile and future disease states. Hemodynamic responses to stress have good temporal stability (Sherwood et al., 1990). From the reported evidence, vascular response profiles appear the most damaging in terms of cardiovascular health. Prolonged vascular responses to stress have been associated with increased vascular resistance, leading to vascular hypertrophy (Folkow, 1982; Obrist, 1982, 1985; Palatini & Julius, 2009). Individuals diagnosed with hypertension typically demonstrate elevated TPR and normal CO, at rest and in response to stress (e.g., S. Julius, 1988; Lund-Johansen, 1991; Manuck, 1994). Furthermore, individuals who exhibited a vascular response profile during an active task (mental arithmetic) showed greater ambulatory pulse pressure (a known risk factor for CVD) and greater ambulatory blood pressure outside of the laboratory (Gregg, Matyas, & James, 2005). However, other research has also suggested that a mixed response profile may play a role in CVD risk. A mixed profile is thought to indicate an abnormality in the homeostatic balance.
between CO and TPR (Hejl, 1957). It is worth noting that in this study a mixed response was most pronounced in late stage hypertensives. Therefore, it is not known if a mixed response profile led to the development of hypertension, or if this response is a by-product of hypertension due to changes in the vasculature.

Figure 1.6. An orthogonal model of HP and CD. Points A and C have a CD of zero as compensation occurred between CO and TPR (Point A; TPR is more negative, Point C; CO is more negative). Points B and C have equal, but positive, CD, however their location on the HP axis defines B as being myocardial and D as vascular (taken from Gregg et al., 2002).

Indeed, one of the predominant models of hypertension development implicates hemodynamic responses in the development of CVD. The hyperkinetic circulatory model (S. Julius, 1988; S. Julius, Esler, & Randall, 1975) proposes higher resting CO
unaccompanied by an appropriate decrease in TPR results in excessive stress on vessels resulting in thickened vessel walls and narrower diameters; eventually leading to permanently elevated TPR (Folkow, 1982; Obrist, 1985). This is hypothesised to result in endothelial injury, which makes arteries more susceptible to platelet deposition; support for this stems from a longitudinal study by Lund-Johansen (1991). At the beginning of the study hypertensive men (under age 30) demonstrated higher resting CO and similar resting TPR compared to a group of age-matched normotensive men. During exercise hypertensives demonstrated exaggerated TPR responding relative to normotensives. Interestingly, at a 10- and 20-year follow-up the resting hemodynamic profile of hypertensives had changed; hypertensives had elevated TPR and lower CO. This demonstrated that over time individuals with essential hypertension developed an abnormal hemodynamic response pattern, marked by a high resistance pattern.

It is unclear which response pattern, if any, plays a causal role in the development of future disease states. However, a consideration of the type of coping context elicited by the stressor (whether active or passive) may help elucidate this relationship. Manuck et al. (1990) highlights the complexities of this relationship. Hemodynamic response patterns demonstrate some cross-stressor reliability (individual-response specificity); however, active tasks and passive tasks are associated with different variations of hemodynamic responding (stimulus-response specificity). It may be the case that when the hemodynamic response pattern elicited is incongruent with that typically elicited by the task this results in a more “harmful” response pattern. Overall, the available research appears to implicate elevations in TPR, without a marked decrease in CO, in disease mechanisms.
2.4.2. Stimulus-response specificity. Active and passive stressors are hypothesised to elicit myocardial and vascular responses, respectively (Schneiderman & McCabe, 1989). As previously mentioned, active tasks are associated with a myocardial response as beta-adrenergic stimulation is elicited during active coping contexts (Obrist, 1981). During such tasks, responders have control over the real or imagined outcomes of the situation. For example, during a mental arithmetic or speech task participants can alter their effort and performance to change the experience of the task. In contrast, passive tasks typically elicit alpha-adrenergic stimulation resulting in a vascular response pattern (Brownley, Hurwitz, & Schneiderman, 2000; Sherwood et al., 1986). Passive tasks provide participants with little control over the task; participants simply endure the experience (e.g., watching emotion-eliciting film-clips or viewing negative imagery). A number of studies have provided support for stimulus-response specificity, where passive tasks elicit an increase in TPR with little or no change in CO, and active tasks elicit increases in CO and decreases in TPR (e.g., Allen, Obrist, Sherwood, & Crowell, 1987; Bolli et al., 1981; Girdler et al., 1996; Gregg, James, Matyas, & Thorsteinsson, 1999; Obrist et al., 1979; Saab et al., 1993; Winzer et al., 1999).

More recently studies employing the HP-CD computational model (Gregg et al., 2002) have provided further support for stimulus-response specificity. Mental arithmetic tasks have been found to elicit myocardial responses (Gregg et al., 2002), and physical passive stressors (the cold pressor task and the handgrip exercise task) have been found to elicit vascular responses (Gregg et al., 2002; Ottaviani et al., 2006). However, the handgrip task was only associated with a vascular response in women; a mixed response was demonstrated by men (Ottaviani et al., 2006). One study tested the effects of a behavioural passive stressor – sleep restriction; sleep restriction produced a vascular
response pattern despite no differences in blood pressure between individuals who were sleep restricted and rested (James & Gregg, 2004). The temporal stability of HP responses has been demonstrated (Gregg et al., 2002). HP responses to laboratory tasks, in particular active stressors, have also been shown to predict ambulatory blood pressure (Gregg et al., 2005; Ottaviani et al., 2006).

To date only a few studies have examined how personality may affect hemodynamic responses. Both these studies used a mental arithmetic stressor and provided support for stimuli-response specificity; the mental arithmetic task was associated with a myocardial response. However, examination of two personality variables, linked to CVD risk, elicited distinct patterns of hemodynamic responding. Hughes et al. (2011) demonstrated that higher scores in neuroticism were associated with a more vascular response to the task. Likewise, Howard et al. (2011) found that participants with the Type D personality trait demonstrated a vascular response profile. Both these personality types have been linked to greater CVD risk (e.g., Denollet et al., 1996; Denollet, Vaes, & Brutsaert, 2000; Smith & MacKenzie, 2006; Suls & Bunde, 2005). Evidence from these studies highlight how personality factors can interact with situational demands to elicit distinct patterns of responding; which is important considering vascular response patterns have been implicated in CVD risk (Lund-Johansen, 1991; Palatini & Julius, 2009).

2.5. Challenge and Threat Appraisals

Cognitive appraisals of one’s perceived ability to manage performance-based stressful situations, such as speech tasks, are argued to influence the hemodynamic response pattern elicited; and mediate the effects of stimulus-response specificity. The biopsychosocial model of challenge and threat (Blascovich & Tomaka, 1996; Tomaka,
Blascovich, Kelsey, & Leitten, 1993) extends appraisal theories (Folkman, 2008; Lazarus & Folkman, 1987) and argues that motivational states evoked by the task elicit either a challenge or a threat cardiovascular response. A cardiovascular challenge response is elicited when perceived resources meet, or exceed, the demands of the situation. In contrast, when the demands of a stressor exceed perceived resources a threat response is elicited (for reviews see; Blascovich & Mendes, 2000, 2010). For example, Tomaka et al. (1993) found participants reporting greater pre-task challenge states to a mental arithmetic task demonstrated a more challenge-oriented cardiovascular response, while participants with a higher ratio of perceived threat demonstrated a more threat-oriented cardiovascular response. Experimentally manipulated challenge and threat states, such that participants were instructed to overcome the challenge of the task (challenge manipulation) or given instructions highlighting the evaluative aspect (threat manipulation), have also been found to result in these distinct response profiles (Tomaka, Blascovich, Kibler, & Ernst, 1997).

2.5.1. The challenge-threat index. Much of the research reporting on cardiovascular indices of challenge/threat responses employ a measure known as the challenge-threat index. Essentially this index transforms CO and TPR values to provide a continuous measure of a physiological challenge response (e.g., Blascovich, Seery, Mugridge, Norris, & Weisbuch, 2004; Shimizu, Seery, Weisbuch, & Lupien, 2011), with higher scores indicative of a more challenge-oriented response. The challenge-threat index appears to employ a similar computation to the HP-CD model; the latter classifying patterns of CO and TPR responding as myocardial, vascular, or mixed, and the former classifying patterns of responding as more challenge-oriented, or more threat-
oriented. While given different names, these computations essentially reflect the same construct.

Much research employing the challenge-threat index focuses on performance. For example, exhibiting a greater physiological challenge response has been shown to predict better performance on a range of laboratory tasks, such as practice Graduate Record Examination questions (Chalabaev, Major, Cury, & Sarrazin, 2009), and a Stroop and basketball task (M. J. Turner, Jones, Sheffield, & Cross, 2012). Likewise, exhibiting a greater cardiovascular challenge response when giving a speech task about a specific motivated performance situation was associated with better performance during that situation outside the laboratory, for example performance on a college course (Seery, Weisbuch, Hetenyi, & Blascovich, 2010) or athlete’s performance during the baseball and softball season (Blascovich et al., 2004).

2.5.2. HP-CD model and challenge-threat index: Conceptual overlap.

Considering that both models essentially transform CO and TPR values it may be the case that they are attempting to measure the same construct. Similar to earlier models that attempted to classify hemodynamic response patterns as indicative of myocardial or vascular responses, the challenge-threat index has a similar aim, but provides a continuous measurement of challenge/threat. This is indeed quite similar to HP-CD computational model; however, the HP-CD model considers the degree to which CO and TPR compensate and essentially, classifies responses as myocardial or vascular (or indeed a mixed response). It appears that responses classified as myocardial via the HP-CD model are classified as challenge-oriented by the challenge-threat index, and vascular responses are indicative of threat-oriented responses.
2.6. Emotion Regulation and Challenge/Threat Responding

The importance of hemodynamic response patterns becomes pertinent when examining the physiological consequences of habitual, and instructed, emotion regulation use. The most consistent evidence for the relationship between emotion regulation style and CVR to stress stems from research examining the influence of reappraisal on patterns of CO and TPR responding. Individuals scoring high in trait reappraisal demonstrated a more challenge-orientated cardiovascular profile in response to a mental arithmetic stressor; indexed by increased CO and decreased TPR (Mauss, Cook, Cheng, et al., 2007). Likewise, in experimental research participants instructed to reappraise arousal as adaptive when completing the TSST demonstrated increased CO and decreased TPR, compared to individuals instructed to ignore feelings of stress, or given no instructions (Jamieson et al., 2012, 2013). Other research in this area has employed the challenge-threat index to classify the underlying hemodynamic response. During the TSST and a competitive math task, individuals instructed to use reappraisal demonstrated greater challenge responses (Hangen, Elliot, & Jamieson, 2019; Jamieson et al., 2013). To our knowledge, no research to-date has applied the HP-CD model to examine the underlying response pattern of reappraisal, nor has research examined the relationship between suppression, or emotion regulation difficulties, and these parameters.

3. Conclusion and Thesis Outline

Evidence from cross-sectional and experimental research advocate reappraisal as a process which buffers the negative emotional and physiological, consequences of stress. In response to acute stress, instructed use of reappraisal, as well as a greater tendency to engage in this strategy habitually, is associated with more positive emotion,
less negative emotion, and a challenge-orientated cardiovascular response. On the other hand, suppression does little to mitigate negative emotional responses during stress, and generally results in exaggerated SNS activity (indexed by blood pressure or skin conductance responding). While instructed use of reappraisal and suppression typically demonstrates distinct patterns of SNS activity, it is difficult to claim that the propensity to use these strategies shows the same pattern of cardiovascular responding. Future research needs to address this gap, in particular, as individual differences would reflect how individuals naturally respond to, and cope with, daily stressors; examining the psychophysiological consequences of trait emotion regulation would therefore offer insights into the long-term implications of reappraisal and suppression use. Furthermore, research in the area can be strengthened by addressing methodological limitations with past research and by examining emotional regulation within an extended cardiovascular reactivity paradigm.

Knowledge and expertise from stress reactivity research can make a substantial contribution to the emotion regulation literature. Past research examining the physiological outcomes of emotion regulation is limited by a number of methodological shortcomings. Although many of these experiments are well-controlled in terms of counterbalancing the order of stimuli, they lack key stages inherent in CVR research. For example, within the stress reactivity literature it is common to include a pre-baseline acclimatization period (e.g., Hogan et al., 2012; Howard et al., 2011; van Stegeren, Wolf, & Kindt, 2008) to allow participants to become adjusted to the laboratory environment and ensure participants are fully rested prior to recording physiological parameters. Emotion regulation studies rarely include such a period. Furthermore, baseline periods tend to be short, usually one minute (e.g., Gross, 1998b; Gruber et al., 2014; Richards &
Gross, 1999; Shiota & Levenson, 2009), three minutes (Dunn et al., 2009), or five 
minutes (e.g., Jamieson et al., 2012, 2013) with instructions to simply “sit quietly”;
“clear your mind” or “relax”. This conflicts with the recommended 10-minute Vanilla 
resting baseline (Jennings, Kamarck, Stewart, Eddy, & Johnson, 1992), where the 
inclusion of a minimally taxing task is recommended in order to avoid arousal from task 
anticipation, boredom, or rumination. A study by Ray, Wilhelm, and Gross (2008) is an 
exception to this; baseline measures were taken for five minutes while participants 
watched a 5-minute neutral film. Considering that physiological baseline measurements 
often act as a comparison against reactivity elicited during a task, the implementation of 
a clear, methodologically sound baseline period is essential.

Furthermore, previous research tends to measure physiological responding in 
terms of skin conductance responsivity or HR, rather than more sophisticated indices of 
CVR, such as blood pressure, CO, and TPR. This is a clear limitation of the emotion 
regulation literature considering the utility of blood pressure reactivity in predicting the 
development of CHD (Carroll et al., 2001; Light et al., 1992). In particular, a focus on 
CO and TPR will help extend research in this area. Reappraisal has already been linked 
to a more challenge-oriented response to stress in terms of CO and TPR; but the 
relationship between these parameters and trait suppression needs to be examined. It 
seems appropriate to assess CO and TPR in future research, but also to apply the HP-CD 
model to confirm the type of hemodynamic response elicited (Gregg et al., 2002).

The relationship between emotion regulation and stress reactivity is also limited 
by the focus of past research on stimuli that are considered “passive” tasks such as film-
clips and negative imagery. Conversely, active tasks, such as speech tasks, require 
participants to be actively engaged during the task and participants can exert some
control over their performance. Within the stress reactivity literature, active tasks are argued to be more representative of everyday stressors, better predictors of future blood pressure, and are associated with a pattern of physiological responding distinct from passive tasks (e.g., Markovitz et al., 1998). In particular, the use of speech tasks has been shown to successfully elicit CVR in the laboratory, and the magnitude of this response has been shown to be generalizable from laboratory to field (Johnston, Tuomisto, & Patching, 2008). Within the emotion regulation literature limitations of passive tasks have also been highlighted. Passive tasks have been argued not to elicit the same internal motivational and socio-evaluative states characteristic of active tasks (Aldao, 2013), while active tasks are posited to prompt stronger emotional response tendencies which result in changes in affect and physiological arousal (Egloff, Schmukle, Burns, & Schwerdtfeger, 2006). It may be the case that active stressors are more reflective of coping responses elicited by daily stressors, compared to simply watching film-clips or negative imagery. Furthermore, the biopsychosocial model postulates that challenge/threat responses are only elicited in motivated performance contexts, such as speech tasks. In a passive context the influence of emotion regulation style on physiological patterns of challenge/threat may not be observable.

Overall, evidence from laboratory and cross-sectional research strongly suggest that reappraisal can promote adaptive stress responding, in terms of affect and physiological arousal. Conversely, use of suppression appears to exacerbate the stress response. While, in the short-term this may not negatively impact health, over time repeated activation of the SNS (for high suppressors) may confer a risk for CHD. Although the reviewed studies present evidence for a relationship between trait emotion regulation and stress responsivity, it must be acknowledged that emotion regulation
research examining CVR is in its early stages. Despite the potential importance of trait emotion regulation in elucidating the effects of stress on physical health few studies have adopted a stress reactivity research perspective. Combining strengths from these overlapping, yet separate, literatures can extend our understanding of this relationship. This thesis addresses the methodological issues outlined; namely the inclusion of an official acclimatization period and a Vanilla resting baseline, the use of active tasks, and the use of more sophisticated measures of cardiovascular responding. In doing so, this research can help elucidate how individual differences in emotion regulation influence responses to stress. There now follows a brief overview of the empirical research reported in the remainder of the thesis.

3.1. Suppression and Emotion Dysregulation are associated with Perceived Stress and Poor Psychological Health: A Cross-Sectional Study

Habitual use of reappraisal is associated with indices of well-being, such as greater PA, lower NA, lower anxiety, and depression. In contrast, greater habitual use of suppression and greater difficulties in emotion regulation are associated with indices of poorer psychological health. The aim of Study 1 (Chapter 2) was to examine if individual differences in emotion regulation style were associated with greater perceived stress, and if this in turn was associated with indices of distress (anxiety and depression symptoms). A total of 170 participants were recruited via MTurk and completed a battery of questionnaires assessing emotion regulation style, distress, and perceived stress.
3.2. Individual Differences in Emotion Regulation and Cardiovascular Reactivity to an Active Stressor

The aim of Study 2 (Chapter 3) was to examine if individual differences in emotion regulation influenced stress responsivity to an acute active stress task. Forty-eight participants completed a well-established cardiovascular laboratory paradigm; acclimatization, baseline, task, inter-task rest period, and task. Habitual use of reappraisal and suppression were measured using the ERQ. Emotion regulation difficulties were measured using the DERS. The main aim of this study was to examine if trait emotion regulation influenced CVR to an active stressor, with a focus on the underlying hemodynamic profile.

3.3. Establishing the Validity of a Novel Passive Stress Task

Study 3 (Chapter 4) focuses on establishing the validity of a novel passive stress task. Cardiovascular responses to typical passive stress tasks, such as watching emotional video-clips or the cold pressor task, may not be generalisable to responses exhibited to stressors encountered in everyday life. This study adapts a more novel stress task and invited participants from Study 2 to return to the laboratory and watch the video-clip of themselves completing the speech task. A total of 26 participants completed the second phase of this research. The aim of the study was to examine if the passive stress task elicited a cardiovascular response distinct from the active task, and if this response was typical of passive coping (increased TPR with little or no change in CO).
3.4. Individual Differences in Emotion Regulation and Cardiovascular Reactivity to a Passive Stressor

Study 4 (Chapter 5) reports on data from Study 2 and Study 3. To date much of the research examining the physiological consequences of emotion regulation have employed passive coping contexts. This study aimed to extend this research by examining the role of trait emotion regulation on CVR to a valid passive stress task. This study reports on the relationship between individual differences in emotion regulation and CVR during a passive coping context.

3.5. Experimentally Manipulating Emotional Regulation Style: Reappraisal and Cardiovascular Habituation to Active Stress

The focus of Study 5 (Chapter 6) was to examine the effects of instructed reappraisal on cardiovascular adaptation to recurrent stress. The active task reported upon in Study 2 was employed. Previous research has demonstrated that instructions to reappraise stress as beneficial and aiding performance results in a more challenge-oriented response during novel stress (indexed by greater CO and lower TPR responding). This study therefore sought to examine if reappraisal would aid cardiovascular adaption to recurrent stress. Participants $(N = 106)$ completed a typical cardiovascular laboratory protocol; acclimatization, baseline, first stress exposure, inter-task rest period, emotion regulation instruction period, and second stress exposure. Participants were randomly assigned to receive reappraisal or control instructions prior to completing the speech task a second time. This study also aimed to examine how trait emotion regulation style interacted with instructions to use reappraisal.
CHAPTER 2

Suppression and Emotion Dysregulation are associated with Perceived Stress and Poor Psychological Health: A Cross-Sectional Study

Introduction

Emotion regulation style and health

A wealth of research has documented the association between individual differences in emotion regulation and psychological health outcomes. Typically, individuals who report greater habitual use of reappraisal, a hypothesised healthful emotion regulation strategy, also report lower anxiety and depressive symptoms (Appleton et al., 2013; Dennis, 2007; Lam et al., 2009; Rogier et al., 2017), more satisfaction with life (Gross & John, 2003), lower self-reported NA, and greater PA (Balzarotti et al., 2010; J. Johnson et al., 2016). In contrast, a greater tendency to engage in suppression is associated with higher anxiety and depression symptoms (Appleton et al., 2013; Chervonsky & Hunt, 2018; Dennis, 2007; Fresco et al., 2007; Nolen-Hoeksema & Aldao, 2011; Rogier et al., 2017), more self-reported NA and less PA (Balzarotti et al., 2010; J. Johnson et al., 2016), as well as lower life satisfaction (Gross & John, 2003). The links between reappraisal, suppression, and psychological health outcomes have been confirmed by a number of meta-analytic studies examining adolescent (Schafer et al., 2017) and adult populations (Aldao & Nolen-Hoeksema, 2010). However, some research has reported no relationship between trait suppression and anxiety (Lam et al., 2009), or between trait reappraisal and depressive symptoms (Chervonsky & Hunt, 2018; Dennis, 2007; Nolen-Hoeksema & Aldao, 2011). A separate
literature, focusing on difficulties in emotion regulation (rather than emotion regulation strategy use), has documented that individuals with greater difficulties in emotion regulation also report greater NA (Pollock et al., 2016) and score higher on measures of depression, anxiety, chronic worry, and perceived stress (e.g., Allan et al., 2015; Bardeen et al., 2012; Ritschel et al., 2015; Roemer et al., 2009; Ruganci & Gencoz, 2010; Salters-Pedneault et al., 2006). Together, this research indicates that emotion regulation style and difficulties in emotion regulation affect psychological health.

Similarly, individual differences in emotion regulation have been implicated in physical health outcomes. For example, greater habitual use of reappraisal has been associated with lower levels of inflammation, while habitual use of suppression has been associated with greater inflammation (Appleton et al., 2013). In particular, inhibition of emotions has been linked to heightened CVD risk, including greater incidence of heart disease and all-cause mortality (e.g., Denollet et al., 1996; Gallacher, Yarnell, Sweetnam, Elwood, & Stansfeld, 1999; Grossarth-Maticek, Bastiaans, & Kanazir, 1985; Jorgensen, Johnson, Kolodziej, & Schreer, 1996; M. Julius, Harburg, Cottington, & Johnson, 1986; Kupper & Denollet, 2018). There is also evidence to suggest greater difficulties in emotion regulation are associated with greater CVD risk. In a sample of individuals reporting high levels of chronic stress, only those reporting greater emotion regulation difficulties had a higher risk of CVD (indexed by greater BMI, greater insulin resistance, and greater resting blood pressure). Individual differences in emotion regulation appear to be associated with physical and psychological health outcomes, but the mechanisms behind these associations are unknown.
Stress appraisals as a mechanism

The way in which individuals appraise and cope with stress may be a pathway through which individual differences in emotion regulation are linked with health outcomes. Indeed, greater perceived stress in daily life, as assessed by the Perceived Stress Scale (PSS; S. Cohen, Kamarck, & Mermelstein, 1983) is associated with a range of negative health outcomes. For example, individuals who report more perceived stress also report lower subjective physical health and mental health, more psychosomatic complaints (Dao-Tran, Anderson, & Seib, 2017; Wiklund, Malmgren-Olsson, Ohman, Bergstrom, & Fjellman-Wiklund, 2012), greater anxiety and depressive symptoms, and lower satisfaction with life (e.g., Klein et al., 2016). Cross-sectional and longitudinal research demonstrate greater perceived stress is associated with markers of CVD risk, such as higher BMI, higher resting blood pressure, greater waist circumference, higher cholesterol, and elevated CRP, as well as greater incidence of hypertension, diabetes, and obesity (e.g., Chiang et al., 2019; Gebreab et al., 2012; Glei et al., 2013; Kashani, Eliasson, & Vernalis, 2012; McDade, Hawkley, & Cacioppo, 2006).

Only a few studies have examined the relationship between emotion regulation style and perceived stress, but results are in the expected directions. Greater habitual use of reappraisal has been associated with lower perceived stress (Miklósi et al., 2014; Troy, Ford, McRae, Zarolia, & Mauss, 2017), while, greater emotion regulation difficulties have been linked to greater perceived stress (Roy et al., 2018; Stalder et al., 2011). Preliminary evidence that stress appraisals may influence the relationship between emotion regulation style and indices of well-being stems from research employing daily diary studies. One study, which assessed self-reported affect and stress over a one-week period, reported that greater reappraisal use was associated with greater PA and lower
NA, while greater use of suppression was associated with lower PA; as expected. However, during days where participants reported high levels of stress, those who habitually engaged in suppression reported less PA (Richardson, 2017). Despite experiencing similar levels of stress, only individuals who habitually engaged in suppression felt lower PA. In this study there were no significant interactions between reappraisal and reported stress. However, in another study assessing mood and stress over a two-week period, trait reappraisal interacted with stress appraisals; on days with high levels of reported stress greater habitual use of reappraisal was associated with less negative mood, suggesting reappraisal buffered the negative effects of stress (J. Johnson et al., 2016).

It may be the case that emotion regulation style influences *appraisals* of stress, leading to the observed psychological and physiological associations. The theoretical underpinnings of the transactional model of stress and coping posits appraisals of stressful situations as key in determining the subsequent psychophysiological effects of stress (Lazarus & Folkman, 1984). If individuals who experience greater emotion regulation difficulties, or individuals who habitually engage in more maladaptive strategies such as suppression, perceive more stress in their environment this may be a mechanism through which emotion regulation style influences psychological and physical health. Similarly, the process model of emotion regulation (Gross, 1998a) would posit that individuals who habitually engage in reappraisal do so before the full emotional experience of the stressor is elicited, thereby changing appraisals of the situation and altering the emotional response. Perhaps these individuals perceive less stress as a result. When responses to stress are overwhelming or inappropriate it can have a multitude of negative consequences for the individual, including poorer psychological
and physical health (for reviews see; Chida & Steptoe, 2010; Lupien et al., 2009; Suls & Bunde, 2005). Emotion regulation ability may help elucidate the relationship between stress responsivity and future health outcomes.

The current study

The current study aimed to examine if individual differences in emotion regulation style (habitual use of reappraisal and suppression, and difficulties in emotion regulation) predicted perceived stress – that is, appraisals of stress in day-to-day life when no immediate threat is posed. We then wished to examine if perceived stress mediates the relationship between emotion regulation style and indices of well-being, including anxiety, depression, and satisfaction with life. It was hypothesised that habitual use of reappraisal would be associated with lower perceived stress and this in turn would be associated with lower distress (anxiety and depression) and greater satisfaction with life. In contrast, we hypothesized that habitual use of suppression, and greater emotion regulation difficulties, would be associated with greater perceived stress, greater distress, and lower satisfaction with life.

Method

Design

The current study employed a cross-sectional design. The main predictor variables were individual differences in emotion regulation (reappraisal, suppression, and difficulties). The outcome variables included distress (anxiety and depressive symptoms) and satisfaction with life. Perceived stress was included as a mediating variable.
Chapter 2, Study 1: Trait ER and Perceived Stress

**Procedure and participants**

Participants were recruited using Amazon Mechanical Turk (MTurk); an online crowdsourcing platform where participants complete research in exchange for financial compensation. To ensure the quality of the data collected, several steps were taken. First, the study was only available to participants with a review score greater than 95% and who were residents of English-speaking countries. Second, to minimize the effect of random responding or inattention, two instructional attention checks were embedded in the survey (e.g., “Select Always”). This is consistent with methods employed in other studies utilizing both online and paper surveys (e.g., Benfer, Bardeen, Fergus, & Rogers, 2018; Oppenheimer, Meyvis, & Davidenko, 2009). Third, along with these “catch questions”, a mathematical equation was also included (“Please add the following numbers and write your answer: 3 and 5 = _”) to screen out potential responses from computer program software. Numerous comparative studies have highlighted that results attained through the MTurk platform mirror results from laboratory-based studies (e.g., Bartneck, Duenser, Moltchanova, & Zawieska, 2015; Casler, Bickel, & Hackett, 2013; Paolacci, Straeter, & de Hooge, 2015); the measures employed above limit the likelihood that responses were random or a result of programming software.

A priori power analyses were conducted using the algorithm devised by Fritz and MacKinnon (2007). Past research has reported medium strength correlations between emotion regulation style and PSS (e.g., Mikolajczak, Nelis, Hansenne, & Quoidbach, 2008), and large correlations between distress and PSS (e.g., Klein et al., 2016). Therefore, to achieve 80% power, a sample size of 88 was needed to detect a small mediated effect, 59 participants were needed to detect a medium mediated effect, and 58 were needed to detect a large mediated effect.
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In total 246 participants completed the battery of questionnaires, including demographic information and psychometric questionnaires. Only participants who correctly responded to the two attention checks and the mathematical equation were included in the final analyses; resulting in a sample of 170 participants (83 women, 87 men), aged 19-86 years ($M = 37.47$ years, $SD = 12.62$).

**Sample demographics.** Participants were predominately residents of the United States of America (98.8%, $n = 168$), with two participants from Canada (1.2%). Most participants identified as Caucasian (76.5%), 7.1% as Black/African American, 4.1% as Asian, 2.9% as Hispanic/Latino, and 9.4% of participants did not report their ethnic group.

The current sample is quite educated; nearly half the respondents had completed an undergraduate level university degree (49.4%, $n = 84$), 14.1% had completed a university level master’s degree ($n = 24$), 0.6% held a PhD ($n = 1$), 14.1% had completed a technical course ($n = 24$), 10.6% had a diploma/certificate ($n = 18$), and 7.1% held a high school qualification ($n = 12$). Three participants responded “other” (1.8%).

**Materials**

**Emotion Regulation Questionnaire (ERQ).** The ERQ (Gross & John, 2003) is a 10-item measure of the emotion regulation strategies of cognitive reappraisal (6 items, e.g., “When I want to feel less negative emotion, I change the way I’m thinking about the situation”) and expressive suppression (4 items, e.g., “I keep my emotions to myself”). Responses are coded on a scale of 1 (strongly disagree) to 7 (strongly agree), such that higher scores represent a greater tendency to use the strategy. ERQ items are presented in Appendix A. Good internal reliability and test-retest reliability across a range of samples have been demonstrated (e.g., Gross & John, 2003; Perez & Soto, 2011). In the current
sample, both subscales demonstrated excellent reliability, with a Cronbach’s α of .83 for the reappraisal subscale and .81 for the suppression subscale.

**Difficulties in Emotion Regulation Scale (DERS).** The DERS (Gratz & Roemer, 2004) is a 36-item measure of emotion regulation deficits in six areas, when distressed; (i) impulse control difficulties (e.g., “When I’m upset, I have difficulty controlling my behaviours”), (ii) lack of emotional awareness (e.g., “I pay attention to how I feel” [reverse-scored]), (iii) lack of emotional clarity (e.g., “I have difficulty making sense out of my feelings”), (iv) nonacceptance of emotional responses (e.g., “When I’m upset, I become irritated with myself for feeling that way”), (v) difficulties engaging in goal-related behaviour (e.g., “When I’m upset, I have difficulty focusing on other things”), and (v) lower perceived access to effective emotion regulation strategies (e.g., “When I’m upset, I believe that there is nothing I can do to make myself feel better”). Higher scores indicate more difficulty in each of these emotional competencies. Responses are coded on a scale of 1 (almost never) to 5 (almost always). DERS items are presented in Appendix B. In the current sample, the DERS total score had excellent reliability; Cronbach’s α was .95. The six subscales all demonstrated a Cronbach’s α > .61 (nonaccept; .94, goals; .86, impulse; .89, aware; .80, strategies; .92 and clarity; .61). For the clarity subscale, the item “When I’m upset, I acknowledge my emotions” was removed to improve Cronbach’s α from .57 to .61.

**Distress.** Symptoms of anxiety and depression were assessed using the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983). The HADS contains two subscales each with seven items, assessing depressive (e.g., “I look forward with enjoyment to things”) and anxiety symptoms (e.g., “I get sudden feelings of panic”). Each item is rated on a four-point Likert scale, 0-3; higher scores indicate greater
symptoms of anxiety and depression. Consistent with previous approaches the anxiety and depression subscales were summed to create an overall distress score, which has demonstrated excellent internal reliability and test-retest reliability (Gough & Hudson, 2009; S. B. Roberts, Bonnici, Mackinnon, & Worcester, 2001; Singer et al., 2009; Spinhoven et al., 1997). The total scale had less-than-optimal reliability (Cronbach’s $\alpha = .59$), therefore two items were removed (“I have lost interest in my appearance” and “I feel tense or wound up”) to improve reliability to .71. The distress scale was computed omitting these two items.

**Perceived Stress Scale.** The 10-item version of the 14-item Perceived Stress Scale (PSS; S. Cohen et al., 1983) was employed to assess perceived stress experienced during the previous month; that is, the degree to which people appraise situations as stressful. The 10-item version is recommended due to its better factor structure and internal reliability (S. Cohen & Williamson, 1988). Items were rated on a five-point Likert scale of 0 (*never*) to 4 (*very often*), with higher scores indicative of greater perceived stress (e.g., “In the last month, how often have you found that you could not cope with all the things you had to do?”). The concurrent validity of this scale has been established (S. Cohen et al., 1983). In the current sample, Cronbach’s $\alpha$ was .75.

**Satisfaction with Life.** Satisfaction with one’s life as a whole, was assessed with the five-item satisfaction with life scale (SWLS; Diener, Emmons, Larsen, & Griffin, 1985). Items were scored on a 7-point scale from 1 (*disagree strongly*) to 7 (*agree strongly*) with higher scores taken to indicate greater life satisfaction (e.g., “I am satisfied with my life”). Numerous studies examining the psychometric properties of this scale have demonstrated its validity and reliability as a measure of well-being (Diener et al.,
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1985; Pavot & Diener, 1993). In the current sample, internal consistency was excellent, with a Cronbach’s α of .93.

Results

Overview of Analyses

Mediation analyses were conducted using the PROCESS macro for SPSS, model 4 (Hayes, 2013) to examine if perceived stress mediates the relationship between emotion regulation style and indices of well-being (distress, satisfaction with life). Indirect effects were estimated with \( n = 5,000 \) bootstrap resamples. Estimates are presented with 95% bootstrapped confidence intervals (CI). Indirect effects are considered to be statistically significant if the 95% CI does not include zero. Table 2.1 displays the means, standard deviations, and range of all self-report measures. Table 2.2 displays intercorrelations for these variables.

Table 2.1.
Means, standard deviations, and range for all variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>( M )</th>
<th>( SD )</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reappraisal</td>
<td>29.58</td>
<td>5.88</td>
<td>5-42</td>
</tr>
<tr>
<td>Suppression</td>
<td>17.24</td>
<td>5.24</td>
<td>4-28</td>
</tr>
<tr>
<td>DERS</td>
<td>88.34</td>
<td>26.29</td>
<td>36-153</td>
</tr>
<tr>
<td>PSS</td>
<td>17.53</td>
<td>7.14</td>
<td>1-34</td>
</tr>
<tr>
<td>SWLS</td>
<td>22.86</td>
<td>8.04</td>
<td>5-35</td>
</tr>
<tr>
<td>Distress</td>
<td>12.04</td>
<td>6.82</td>
<td>1-32</td>
</tr>
</tbody>
</table>
Table 2.2.

*Intercorrelations between emotion regulation and health outcomes*

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Distress</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. SWLS</td>
<td>-.37**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. PSS</td>
<td>+.72**</td>
<td>-.35**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Reappraisal</td>
<td>-.10</td>
<td>.37**</td>
<td>-0.89</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Suppression</td>
<td>+.16*</td>
<td>+.11</td>
<td>+.24*</td>
<td>+.12</td>
<td></td>
</tr>
<tr>
<td>6. DERS</td>
<td>+.68**</td>
<td>-.02</td>
<td>+.71**</td>
<td>-.04</td>
<td>+.37**</td>
</tr>
</tbody>
</table>

*Perceived Stress as a mediator*

There was a significant total effect of emotion regulation difficulties on reported distress, $b = 0.18$, $t = 11.73$, $p < .001$, 95% CI [0.15, 0.21], explaining 45.9% of the variance in distress. There was a significant indirect effect of emotion regulation difficulties on distress, via perceived stress, $b = 0.09$, [0.06, 0.12]. Figure 2.1 demonstrates people with greater emotion regulation difficulties perceived higher levels of stress, and this was associated with greater distress (depressive and anxiety symptoms). The mediation model was repeated with each of the DERS subscales entered as predictors. The results remained the same for all subscales with the exception of difficulties in emotional awareness. While difficulties in emotional awareness positively predicted distress ($p < .001$), there was no direct effect of awareness on perceived stress ($p = .270$), nor an indirect effect of awareness on distress via perceived stress, $b = 0.10$, [-0.10, 0.29].
Likewise, there was a significant total effect of suppression on distress, $b = 0.21$, $t = 2.01$, $p = .047$, $[0.01, 0.41]$, explaining 2.42% of the variance in distress. There was an indirect effect of suppression on distress, via perceived stress, $b = 0.19$, $[0.05, 0.35]$. Figure 2.2 demonstrates greater habitual use of suppression was associated with greater perceived stress, and this was associated with greater distress.

There was no total effect of reappraisal on distress, $b = -0.12$, $t = -1.30$, $p = .196$, $[-0.30, 0.06]$, nor an indirect effect through perceived stress, $b = -0.06$, $[-0.20, 0.09]$.

**Satisfaction with Life**

Although there was no total effect of emotion regulation difficulties on satisfaction with life, $b = 0.003$, $t = 0.13$, $p = .899$, $[-0.04, 0.05]$, there was a significant indirect effect through perceived stress, $b = -0.16$, $[-0.21, -0.11]$. Figure 2.3 demonstrates that individuals with greater difficulties in emotion regulation reported greater perceived stress, and this was associated with lower life satisfaction. These results were replicated using the subscales of the DERS, with the exception of the awareness subscale. There was no direct effect of awareness on perceived stress ($p = .383$). However, there was a direct effect on life satisfaction, $b = -0.60$, $t = -4.64$, $p < .001$, $[-0.86, -0.35]$. Greater difficulties in emotional awareness were associated with lower reported life satisfaction, but perceived stress did not mediate this relationship, $b = -0.04$, $[-0.16, 0.06]$.

Similarly, there was no total effect of suppression on life satisfaction, $b = 0.18$, $t = 1.55$, $p = .123$, $[-0.05, 0.41]$; however, there was a significant indirect effect through perceived stress, $b = -0.01$, $[-0.28, -0.43]$. Figure 2.4 demonstrates people reporting greater habitual use of suppression also reported more perceived stress, and this was associated with lower life satisfaction.
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While there was a total effect of reappraisal on life satisfaction, $b = 0.49$, $t = 4.96$, $p < .001$, $[0.29, 0.68]$ explaining 12.9% of the variance in life satisfaction, there was no indirect effect of reappraisal via perceived stress on life satisfaction, $b = 0.04$, $[-0.03, 0.12]$. Although habitual use of reappraisal was associated with greater life satisfaction, perceived stress did not influence this relationship.

Figure 2.1. Individuals with greater difficulties in emotion regulation reported more perceived stress, and this was associated with greater anxiety and depression symptoms.

Figure 2.2. Individuals who habitually engage in suppression reported more perceived stress, and this was associated with greater anxiety and depression symptoms.
Figure 2.3. Individuals with greater difficulties in emotion regulation reported more perceived stress, and this was associated with lower life satisfaction.

Figure 2.4. Individuals who habitually engage in suppression reported more perceived stress, and this was associated with lower life satisfaction.

**Discussion**

The results demonstrate that greater habitual use of suppression, and greater difficulties in emotion regulation, are associated with greater perceived stress and greater anxiety and depressive symptoms (distress). The mediation model suggests perceived stress may be a pathway through which emotion regulation style is associated with
greater distress. These individuals perceive more stress in their day-to-day life, and this appears to have consequences for psychological health.

This is the first study to suggest individual differences in habitual use of suppression and difficulties in emotion regulation influence psychological health via perceived stress. It appears that individuals who score high on these constructs not only experience higher levels of anxiety and depressive symptoms, as reported by a wealth of research (e.g., Allan et al., 2015; Appleton et al., 2013; Chervonsky & Hunt, 2018; Dennis, 2007; Fresco et al., 2007; Nolen-Hoeksema & Aldao, 2011; Ritschel et al., 2015; Rogier et al., 2017), but also perceive more stress in their lives. If individuals scoring higher in suppression and emotion regulation difficulties typically report more stress in day-to-day life it is likely this would translate to responses during acute stress. Greater perceived stress when faced with acute stress is theorised to be reflected in physiological responses to acute stress (Lazarus & Folkman, 1984); with exaggerated CVR leading to future negative health outcomes (e.g., Treiber et al., 2003). Indeed, instructions to suppress responses during acute stress results in greater SNS reactivity (e.g., Gross, 1998b; Harris, 2001; N. A. Roberts et al., 2008). The relationship between emotion regulation style and stress responsivity during acute stress needs to be tested in a controlled laboratory environment. By examining if a direct psychosomatic pathway exists between emotion regulation style and CVR this may explain the observed relationship between suppression use and greater inflammation (Appleton et al., 2011) and greater difficulties in emotion regulation with increased CVD risk (Roy et al., 2018).

Interestingly, habitual use of reappraisal was neither related to perceived stress nor self-reported distress, contrasting with some reported research (e.g., Appleton et al., 2013; Dennis, 2007; Mikolajczak, Nelis, Hansenne, & Quoidbach, 2008; Troy et al.,
While greater habitual use of reappraisal was associated with greater life satisfaction, perceived stress did not influence this relationship. It appears the reported benefits of reappraisal use, in terms of greater well-being, occurs through some other mechanism, rather than via perceived stress. However, it is not unusual for trait reappraisal to show weak associations with indices of poorer well-being. For example, a number of studies have reported no relationship, or modest relationships, between habitual reappraisal and indices of psychopathology (e.g., Chervonsky & Hunt, 2018; Dennis, 2007; Nolen-Hoeksema & Aldao, 2011). Likewise, a number of studies report stronger associations between maladaptive strategies and negative psychological health outcomes (Aldao & Nolen-Hoeksema, 2010, 2012; Aldao, Nolen-Hoeksema, & Schweizer, 2010).

It is worth noting that the present study tested associations between trait reappraisal and perceptions of stress in daily life, not in response to an acute stressor. Perhaps reappraisal influences stress responsivity when faced with acute stress, which, over-time, leads to better health outcomes. Indeed, laboratory studies instructing reappraisal use show that reappraisal is associated with lower NA, greater PA, and lower SNS during acute stressors (e.g., Gross, 1998b; Gross & John, 2003; Mauss, Cook, Cheng, et al., 2007). However, few studies have examined if habitual use of this strategy leads to similar outcomes.

A strength of this study is the large number of respondents, and the heterogeneous sample with the inclusion of participants across different ethnic groups and age ranges. This would not have been achieved by sampling a student population. However, the sample predominantly resided in the USA and over three-quarters of participants identified as Caucasian. Furthermore, these individuals were, in general, well-educated,
and given the recruitment method, technologically literate. This somewhat limits the
generalisability of the results to other cultures and different socio-economic groups. As
an online recruitment tool was employed this meant that participants completed the
survey alone, therefore we cannot be confident that participants fully engaged with the
material. However, the inclusion of attention checks overcomes this limitation; this
ensured the sample included in the reported analyses engaged with the survey. The
observed results are also strengthened by the exclusion criteria employed; the survey was
only available to participants from English speaking countries, and participants who had
a responder rating of above 95%, improving the quality of collected data. The cross-
sectional nature of the data prevents us from observing causal relationships; future
research needs to examine the relationship between emotion regulation style, perceived
stress, and indices of well-being longitudinally.

While cross-sectional, the results offer an insight into how emotion regulation
style may alter stress appraisals. Given the role of stress appraisals in the cardiovascular
reactivity hypothesis it seems worthwhile to test the role emotion regulation plays in
cardiovascular responding to stress in a laboratory session. The next chapter will
therefore examine how individual differences in emotion regulation influence stress
responsivity; in terms of stress appraisals and CVR to acute stress.
CHAPTER 3

Individual Differences in Emotion Regulation and Cardiovascular Reactivity to an Active Stressor

Introduction

The results of Study 1 implicate perceived stress as a pathway through which emotion regulation style influences well-being. Indeed, within the stress reactivity literature, appraisals of stress-eliciting situations are posited to determine the affective and physiological responses to stressors (Lazarus & Folkman, 1987). It follows that if individual differences in emotion regulation style alter appraisals of acute stress, this will influence CVR. Indeed, empirical studies have provided evidence that instructed use of reappraisal and suppression influences physiological responses to stressful stimuli in a laboratory setting.

Reappraisal and Suppression

Typically, instructed reappraisal results in lower SNS activity, as indexed by lower mean arterial pressure and skin conductance (e.g., Ayduk & Kross, 2008; Gruber et al., 2014). Reappraisal is hypothesised to result in lower physiological responding as it involves changing the appraisals of a situation; if a stress-eliciting situation is reappraised as not “stressful” or non-threatening this should be reflected by lower physiological responding, consistent with the transactional model of stress and coping (Lazarus & Folkman, 1984). In contrast, instructed suppression leads to greater SNS activity; as indexed by greater skin conductance, HR, and blood pressure responses (e.g., Butler et al., 2003; Gross, 1998b; Harris, 2001; Hofmann et al., 2009; Quartana & Burns,
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2010; N. A. Roberts et al., 2008). Suppression involves continuously inhibiting the emotional response and this is thought to incur high cognitive and physiological costs (Appleton & Kubzansky, 2014), resulting in poorer memory for emotion-eliciting stimuli and exaggerated SNS responding (e.g., Richards & Gross, 1999). Results from experimental studies have corroborated the argument that reappraisal can be considered an adaptive strategy; it is associated with lower physiological arousal which may offer a protective function in terms of physical health. While use of instructed suppression appears to exacerbate physiological responding.

However, past research largely instructs the use of emotion regulation. Examining the consequences of instructed emotion regulation tells us little about how habitual use of these strategies (how individuals regulate their emotions in their day-to-day life) may influence CVR. Furthermore, research to-date has focussed on stimuli that are considered “passive” stressors, such as asking participants to view emotion-eliciting video-clips or images. During such tasks participants simply endure the experience and have little control over the task (e.g., Obrist, 1981; Obrist et al., 1978). Conversely, active tasks require engagement, are argued to be more representative of everyday stressors, better predictors of future blood pressure, and are associated with a pattern of physiological responding distinct from passive tasks (Markovitz et al., 1998).

Only a handful of studies have examined the influence of emotion regulation style on CVR during active stress. Reported results suggest trait reappraisal moderates CVR, but there is little evidence to suggest trait suppression influences CVR. 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). Other research has focused on the two
Chapter 3, Study 2: Trait ER and CVR to Active Stress

compensatory cardiovascular parameters underlying blood pressure responses, CO and TPR. Individuals scoring high in reappraisal demonstrated a challenge-oriented cardiovascular response during a mental arithmetic task; indexed by increased CO and decreased TPR (Mauss, Cook, Cheng, et al., 2007). Likewise, a number of studies have shown that participants instructed to use reappraisal demonstrate a challenge-oriented cardiovascular response during active stress tasks (greater CO and lower TPR), compared to individuals instructed to ignore feelings of stress, or given no instructions (e.g., Jamieson et al., 2012, 2013). No research to-date has examined the relationship between suppression and hemodynamic response patterns during active stress.

**Difficulties in Emotion Regulation**

No published research has examined the influence of emotion regulation difficulties on stress responsivity. There is some evidence to suggest greater difficulties in emotion regulation are associated with lower resting HRV (Visted et al., 2017; Williams et al., 2015); taken to indicate a more inflexible parasympathetic nervous system (Appelhans & Luecken, 2006; Thayer et al., 2012). However, conflicting results have also been reported (Stalder et al., 2011). Despite no research directly examining difficulties in emotion regulation and responses to stress, cross-sectional research suggests a relationship. Only in adults reporting greater difficulties in emotion regulation was chronic stress associated with greater CVD risk (indexed by greater resting blood pressure, body mass index, and insulin resistance); chronic stress was not associated with CVD risk in individuals scoring low in emotion regulation difficulties (Roy et al., 2018). This suggests that emotion regulation ability may help buffer the physiological impact of chronic stress on CVD risk factors, and by association influence CVR to stress.
The Current Study

While instructed use of reappraisal and suppression is associated with lower, and greater, SNS responding respectively, less research has taken the individual difference approach, particularly when examining blood pressure reactivity. Drawing on the current research it is difficult to claim that the propensity to use reappraisal reliably results in lower stress reactivity, and suppression results in greater stress reactivity.

The current study will therefore employ a typical active stress task (a speech task) to examine the relationship between CVR and individual differences in emotion regulation within a standardized laboratory paradigm. This will be the first study 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 (e.g., Hangen et al., 2019; Jamieson et al., 2013). In Chapter 1, we argued that the challenge-threat index and HP-CD model essentially reflect the same construct. Therefore, in this study we will compare classifications of challenge/threat responders as suggested by challenge-threat index with myocardial/vascular responders as identified by the HP-CD computation.

The emotional aspect of the speech task will be manipulated; participants will speak about a block of neutral-emotion and negative-emotion words. Previous research has suggested that the emotional context of the task is important, at least for the use of suppression. It is hypothesized that suppression only results in greater SNS activity when individuals attempt to inhibit the expression of strong emotional responses; that is, suppressing a neutral-emotion event, or an event that does not elicit strong emotions,
should not result in exaggerated physiological arousal (Gross & Levenson, 1997; Wegner & Gold, 1995).

We expected that greater habitual use of reappraisal would be associated with lower blood pressure responses and a more myocardial response to the task. In contrast, we hypothesized a greater tendency to suppress would be associated with greater blood pressure responses and a vascular response; but this may only be evident in response to the negative task. We expected that greater emotion regulation difficulties would be associated with an increase in blood pressure, and this would be underlined by a more vascular hemodynamic profile.

Methodology

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 affect (NA, PA, stress, and anxiety) and mean SBP, DBP, HR, CO, and TPR.

Participants

A total of 51 participants reporting good health, no history of cardiovascular disease, and not taking medication known to affect blood pressure, completed the study. Participants were required to be between 18-25 years of age, non-smokers, and prior to participation refrain from consuming caffeinated products for six hours and alcohol for 12 hours. Such restrictions were employed due to the influence of caffeine (e.g., Hartley et al., 2000; James & Richardson, 1991; Savoca et al., 2005), smoking (e.g.,
Cruickshank, Neil-Dwyer, Dorrance, Hayes, & Patel, 1989; James & Richardson, 1991; Saladini et al., 2016), and alcohol (e.g., McFadden, Brensinger, Berlin, & Townsend, 2005; Potter, Watson, Skan, & Beevers, 1986) on blood pressure. 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 (Chobanian et al., 2003; Whitworth & Chalmers, 2004; WHO & ISH, 2003). A final sample of 48 healthy young adults, testing as normotensive, were included in the analyses (25 women and 23 men) aged 18-24 ($M = 19.77$, $SD = 1.39$).

A priori power analyses were conducted with G*Power (Faul, Erdfelder, Lang, & Buchner, 2007), using an alpha of .05, and power of .80. In order to acquire sufficient power a minimum of 35 participants were needed to detect large effects ($f = .40$) and a minimum of 68 were needed to detect medium effects ($f = .25$).

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.* Cardiovascular parameters (SBP, DBP, HR, CO, and TPR) were measured non-invasively using the Finometer PRO (Finapres Medical Systems BV, BT Arnhem, The Netherlands). The Finometer provides beat-to-beat measures of blood pressure and hemodynamic monitoring, based on the volume-clamp method (Peñaz, 1973). A finger cuff is attached to participant’s middle finger. Inside the cuff is an infrared transmission plethysmograph which maintains the arterial walls at a set diameter.
The Finometer has been used extensively in cardiovascular and clinical research; and meets the validation criteria of the Association for the Advancement of Medical Instrumentation and the revised protocol of the British Hypertension Society (Schutte, Huisman, Van Rooyen, Oosthuizen, & Jerling, 2003). CO and TPR indicators are provided based on the validated Modelflow modelling method (Wesseling, De Wit, Van der Hoeven, Van Goudoever, & Settels, 1995; Wesseling, Jansen, Settels, & Schreuder, 1993).

**Psychological Measures.**

**Individual differences in emotion regulation.** The ERQ (Gross & John, 2003) and DERS (Gratz & Roemer, 2004), as described in Study 1, assessed individual differences in emotion regulation. In the current sample, the scales showed good reliability with a Cronbach’s $\alpha$ of .85, .65, and .95 for the reappraisal, suppression, and DERS, respectively.

**Self-reported/spontaneous use of emotion regulation strategies.** A four-item scale was employed to assess self-reported use of emotion regulation strategies during each stress task; this contained a two-item reappraisal subscale and a two-item suppression subscale. Items were adapted from similar measures (Egloff et al., 2006; Ehring, Tuschen-Caffier, Schnulle, Fischer, & Gross, 2010). Participants indicated the extent to which they used reappraisal (e.g., “I tried to change the way I thought about the task to stay calm”) and suppression (e.g., “I tried not to let my feelings show during the task”) on a 6-point scale ranging from 1 (disagree strongly) to 6 (agree strongly); higher scores indicated greater use of the strategy. Spearman-Brown coefficient indicated that the scales had good internal consistency, all $\alpha$’s > .76. However, the self-reported use of
reappraisal scale had less-than-ideal internal consistency in response to the negative-emotion task, $\alpha = .60$.

**Affect measures.**

*Positive Negative Affect Schedule (PANAS).* The PANAS (Watson, Clark, & Tellegen, 1988) is a 20-item scale measuring state positive and negative affect. This measure asks participants to indicate the extent to which they agree with a number of statements from 1 (*very slightly/not at all*) to 5 (*extremely*). 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 $\alpha$ was $\geq .79$ for each scale.

*Self-report questionnaires.* Participants were asked, pre- and post-task, to rate on a ten-point Likert scale from 1 (*not at all*) to 10 (*extremely*) the extent to which they felt stressed or anxious in that moment.

**Stressor task.** A socio-evaluative speech task was employed, using standardized presentation of word stimuli. A block of forty negative-emotion words and forty neutral-emotion words were presented on Superlab (Version 2.02; Cedrus Corporation; San Pedro, CA). Words were chosen from the Affective Norms for English Words (ANEW; Bradley & Lang, 1999); a set of words rated in terms of valence (negative/positive emotions elicited) and arousal (high, low) with the aim to provide a verbal version of the existing IAPS (Lang, Bradley, & Cuthbert, 2008). Words for the current stress task were selected using previously reported cut-offs for arousal and valence (Scott, O'Donnell, Leuthold, & Sereno, 2009). Words were deemed *negative* if they had an arousal value greater than 6.00 and a valence value less than 4.00, and *neutral* if the arousal value was less than 5.45 and the valence value was between 4.00 and 6.00. The list of words
included are presented in Appendix C. This format of speech task (block of specific
words presented) has previously been shown to reliably elicit activity of the
cardiovascular system (e.g., Hughes & Callinan, 2007; O'Súilleabháin, Howard, &
Hughes, 2018). The words selected in these studies, however, were generic nouns taken
from the MRC Psycholinguistic database (Coltheart, 1981). For the purpose of this study,
and to alter the emotional content of the stimuli, we choose to employ words from the
ANEW.

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. Demographic information was recorded, and
participants completed a 20-minute acclimatization period; during which participants
completed a series of psychometric scales and were given neutral reading material.
Participants were then connected to the Finometer PRO. The Finometer cuff was attached
to the middle finger of the non-dominant hand. Resting cardiovascular measures were
taken during an official 10-minute baseline period while participants completed the
affective scales. Again, reading material was provided to lower the risk of potential
boredom and/or rumination arousal, employing the Vanilla resting baseline as
recommended by Jennings et al. (1992). Following this, participants were verbally
informed that words would appear on the computer screen, and to talk about each word
for as long as possible. The experimenter evaluated when to change the word presented;
after three seconds of silence (as measured by a stopwatch), or repetition. Participants
were instructed to look at the camcorder as much as possible during the task as the
recorded video-clips would later be analysed for body language; this instruction was given in order to heighten the socio-evaluative aspects of task. The task lasted for five minutes. Participants were asked to fill out affective rating scales. After a ten-minute inter-task rest period, participants completed the task for a second time. Presentation of the block of neutral-emotion and negative-emotion words was counterbalanced. Following completion of the laboratory session participants 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 3.1. Internal consistency for each cardiovascular variable was excellent with Cronbach’s $\alpha > .98$ for all phases. Reactivity scores to each task were calculated for each parameter by subtracting mean baseline values from the mean of the task.

To investigate the association between stress responsivity and trait emotion regulation (reappraisal, suppression, and difficulties) a series of custom-built $3 \times 1$ repeated measures ANCOVAs were conducted. The within-subjects factor was phase with three levels; baseline, negative-emotion task, and neutral-emotion task. Continuous scores on trait emotion regulation were entered as a covariate. The dependent variables were cardiovascular parameters and self-reported affect.

HP and CD scores in response to each task were calculated using the method proposed by Gregg et al. (2002). Consistent with this approach one-sample $t$-tests were
conducted to examine if HP and CD scores differed from zero. A significant result for CD indicates a change in blood pressure. A significant result for HP indicates a vascular response pattern if the \( t \)-value is positive, and myocardial response if the \( t \)-value is negative. A significant CD result, but no significant HP result signals a mixed response.

The challenge-threat index was calculated using the recommended approach (e.g., Blascovich et al., 2004; Shimizu et al., 2011). Cardiac output and TPR scores were standardised and weighted; TPR values were given a weight of -1, and CO values a weight of +1. These values were summed to produce a challenge-threat index for each task. HP and challenge-threat values were transformed to provide a nominal challenge/threat categorisation; a chi-square test of association was conducted to examine the association between myocardial/vascular responses (as categorised by the HP computation) and challenge/threat responses (as categorised by the challenge-threat index).

Effect sizes are presented as partial \( \eta^2 \) for ANOVA analyses with values of .04, .25, and .64 taken to demonstrate small, medium, and large effects, respectively (J. Cohen, 1992). Partial \( \eta^2 \) has been recommended for ANOVA designs (Tabachnick & Fidell, 1989). Effect sizes for correlations are presented as \( r \), with values of .10, .30, and .50 taken to be indicative of small, medium, and large effect sizes respectively (J. Cohen, 1988, 1992). Effect sizes for one-sample \( t \)-tests were calculated using G*Power, and are presented as \( d \), with values of .20, .50, and .80 indicative of small, medium, and large effect sizes.

**Emotion Regulation Scores**

Table 3.2 displays means, standard deviations, and the range of scores on each emotion regulation subscale.
### Stress manipulation check

**Confirmation of reactivity.**

**Baseline to task.** Table 3.1 displays means and standard deviations for each phase. A series of repeated measures ANOVAs confirmed that the negative-emotion and neutral-emotion tasks elicited a physiological stress response. SBP, DBP, HR, and CO increased from baseline to task, all *p* < .006. For TPR, there was no main effect of phase (*p* = .118). Pairwise comparisons found that there were no differences in SBP, DBP, HR, CO, or TPR between the two tasks, all *p* > .085. Each task, regardless of emotion content, elicited a significant cardiovascular stress response.

### Psychological experience of the stressor task

There was a significant effect of phase on self-reported stress, *F*(2, 92) = 4.24, *p* = .017, partial *η*² = .084, and PA, *F*(2, 94) = 5.48, *p* = .006, partial *η*² = .104. Pairwise comparisons confirmed participants reported more stress after the negative-emotion task (M = 3.40, SD = 2.17) compared to baseline (M = 2.67, SD = 1.45), *p* = .049, 95% CI [-1.57, -.002], and less PA after the negative (M = 30.19, SD = 7.70) and the neutral task (M = 30.08, SD = 7.94) compared to baseline (M = 32.08, SD = 7.31), *p* = .017, and *p* = .015, respectively. There were no differences in self-reported stress and PA, between post-neutral and post-negative task periods. There was no significant effect of phase for ratings of anxiety, *F*(2, 92) = 1.06, *p* = .353 or self-reported NA, *F*(2, 94) = 1.34, *p* = .266, partial *η*² = .028. Overall, participants reported less PA after each task, less stress after the negative-emotion task, and there were no differences in self-reported affect between the two tasks.
Chapter 3, Study 2: Trait ER and CVR to Active Stress

Trait Emotion Regulation

**Physiological outcomes.** There was a significant Phase × Reappraisal interaction effect for TPR ($p = .005$); however, no significant Phase × Reappraisal interaction effect for CO ($p = .077$). Table 3.3 displays the full results. Figure 3.1 shows that greater habitual use of reappraisal was associated with lower TPR and greater CO reactivity in response to the negative task ($r = -.426, p = .005$, and $r = +.311, p = .045$, respectively), but was unrelated to CO and TPR reactivity during the neutral task ($r = -.070, p = .685$, and $r = +.105, p = .507$, respectively). Table 3.3 shows there were no significant interaction effects for SBP, DBP, or HR (all $p$s $>.118$).

There was a significant Phase × Suppression interaction effect for SBP ($p = .011$) and DBP ($p = .007$). Figure 3.2 demonstrates that greater habitual use of suppression was associated with greater SBP and DBP reactivity to both the neutral task (SBP, $r = +.336, p = .029$; DBP, $r = +.323, p = .037$) and negative task (SBP, $r = +.370, p = .016$; DBP, $r = +.437, p = .004$). There were no significant Phase × Suppression interaction effects for HR, CO, or TPR (all $p$s $>.269$). Table 3.3 displays the full results. Overall, the results suggest that greater habitual use of suppression is associated with exaggerated blood pressure reactivity, regardless of the emotional nature of the task. In response to the negative task, a greater tendency to engage in reappraisal was associated with lower TPR and greater CO reactivity, indicative of a myocardial (challenge-oriented) response.

**Psychological outcomes.** Trait reappraisal and trait suppression were not associated with self-reported affect during the baseline period, all $p$s $>.114$. Likewise, there was no significant Task × Reappraisal, or Task × Suppression interactions for any of the self-report measures of affect, all $p$s $>.146$. The results suggest that trait emotion regulation had no influence on self-reported affect.
Table 3.1.

Mean (with SDs) cardiovascular parameters at each phase

<table>
<thead>
<tr>
<th>Phase</th>
<th>Baseline</th>
<th></th>
<th>Negative Task</th>
<th></th>
<th>Neutral Task</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>119.10</td>
<td>10.81</td>
<td>131.84</td>
<td>16.69</td>
<td>129.95</td>
<td>16.44</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>70.88</td>
<td>9.01</td>
<td>79.63</td>
<td>10.20</td>
<td>78.83</td>
<td>10.98</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>75.88</td>
<td>11.42</td>
<td>80.17</td>
<td>10.42</td>
<td>79.91</td>
<td>11.08</td>
</tr>
<tr>
<td>CO (lpm)</td>
<td>6.22</td>
<td>1.68</td>
<td>6.54</td>
<td>1.93</td>
<td>6.40</td>
<td>1.73</td>
</tr>
<tr>
<td>TPR (pru)</td>
<td>0.94</td>
<td>0.28</td>
<td>1.05</td>
<td>0.44</td>
<td>1.01</td>
<td>0.33</td>
</tr>
</tbody>
</table>

Note. Task order was counterbalanced. SBP, DBP, HR, and CO significantly increased from baseline to each task. TPR did not change.

Table 3.2.

Means, standard deviations, and range of scores for each emotion regulation scale

<table>
<thead>
<tr>
<th>Scale</th>
<th>M</th>
<th>SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reappraisal</td>
<td>30.67</td>
<td>5.96</td>
<td>17-42</td>
</tr>
<tr>
<td>Suppression</td>
<td>11.10</td>
<td>3.48</td>
<td>4-18</td>
</tr>
<tr>
<td>Total DERS</td>
<td>84.70</td>
<td>23.91</td>
<td>50-135</td>
</tr>
<tr>
<td>Impulse Control</td>
<td>11.77</td>
<td>5.54</td>
<td>6-26</td>
</tr>
<tr>
<td>Nonacceptance</td>
<td>14.11</td>
<td>5.59</td>
<td>6-27</td>
</tr>
<tr>
<td>Goal-directed behaviour</td>
<td>15.63</td>
<td>4.99</td>
<td>5-25</td>
</tr>
<tr>
<td>Clarity</td>
<td>11.56</td>
<td>3.69</td>
<td>5-21</td>
</tr>
<tr>
<td>Awareness</td>
<td>14.63</td>
<td>4.43</td>
<td>8-24</td>
</tr>
<tr>
<td>Strategies</td>
<td>17.05</td>
<td>7.09</td>
<td>8-34</td>
</tr>
</tbody>
</table>
Figure 3.1. Greater habitual use of reappraisal was associated with greater CO and lower TPR reactivity in response to the negative task. A median spilt was applied to reappraisal scores for demonstration purposes.
Figure 3.2. Greater habitual use of suppression was associated with greater SBP and DBP responding to both tasks. A median split was applied to suppression scores for demonstration purposes.
Difficulties in Emotion Regulation

**Physiological outcomes.** There was a significant Phase × DERS interaction effect for CO ($p = .014$) and TPR ($p = .027$). Figure 3.3. demonstrates greater emotion regulation difficulties were associated with lower CO reactivity ($r = -.312, p = .042$) and higher TPR reactivity ($r = +.314, p = .040$) to the negative task, but were not related to reactivity to the neutral task: CO reactivity, $r = +.143, p = .360$; TPR reactivity, $r = -.123, p = .432$. There were no significant Phase × DERS interaction effects for SBP, DBP, or HR (all $p$s > .243). Table 3.3 displays the full results. Overall, individual differences in emotion regulation difficulties did not influence blood pressure responses. However, individuals with greater emotion regulation difficulties demonstrated greater TPR and lower CO responses to the negative task, indicative of a vascular (threat-orientated) response.

**Psychological outcomes.** At baseline, greater emotion regulation difficulties were associated with greater self-reported NA ($r = +.641, p < .001$), stress ($r = +.432, p = .004$) and anxiety ($r = +.423, p = .005$), but unrelated to PA ($r = -.097, p = .537$). There were no Phase × DERS interaction effects for any of the affective measures, all $p$s > .075. Greater emotion regulation difficulties were associated with greater negative emotions in the absence of stress but were not related to the emotional experience of the tasks during acute stress.

**Further Analyses of the DERS**

The ANCOVA analyses were repeated with the subscales of the DERS entered as covariates to examine which facets were underlying the observed vascular response to the negative-emotion task. The subscales of emotional clarity, engaging in goal-directed behaviour, and emotional awareness had no influence on cardiovascular parameters, all
For the subscales of impulse control (Impulse), acceptance of emotional responses (Accept), and accessing emotion regulation strategies (Strategies), there were significant Phase × Difficulties interaction effects on CO and TPR (with the exception of Accept and CO responding). There were no significant interaction effects for SBP, DBP, or HR, all ps > .056. Table 3.4 presents these results. Correlational analyses presented in Table 3.5, confirmed that, similar to the overall DERS scores, greater scores on the Impulse, Accept, and Strategies subscales were associated with lower CO reactivity and greater TPR. These three subscales appear to be driving the relationship between emotion regulation difficulties and a threat-oriented response to the negative task.

Examination of the underlying hemodynamic profile

One sample $t$-tests found that both the negative and neutral tasks elicited a mixed pattern of hemodynamic responding. HP values were not significantly different from zero: 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 CD values were significantly different from zero: negative task, $t(47) = 6.24, p < .001, d = 0.90, [0.03, 0.05]$; neutral task, $t(47) = 9.68, p < .001, d = 1.40, [0.02, 0.04]$.

Reappraisal. There was a significant Phase × Reappraisal interaction effect on HP scores, $F(1, 40) = 7.44, p = .009, \text{ partial } \eta^2 = .157$. Figure 3.4 demonstrates that greater habitual use of reappraisal was associated with a more myocardial response profile to the negative task ($r = -.418, p = .006$), and unrelated to hemodynamic responses to the neutral task ($r = -.094, p = .553$). Likewise, there was a significant Phase × Reappraisal interaction effect on CD scores, $F(1, 40) = 7.50, p = .009, \text{ partial } \eta^2 = .158$.

Higher scores on trait reappraisal were associated with less CD to the negative task ($r =
Examination of the underlying hemodynamic profile confirmed that the observed patterns of CO and TPR responding for trait reappraisers during the negative task were indicative of a myocardial cardiovascular response.

**Suppression.** Habitual suppression use did not influence HP, $F(1, 40) = 0.35, p = .853$, partial $\eta^2 = .001$, or CD, $F(1, 40) = 0.41, p = .840$, partial $\eta^2 = .001$.

**Emotion Regulation Difficulties.** There was a significant Phase × DERS interaction effect on HP scores, $F(1, 41) = 7.91, p = .008$, partial $\eta^2 = .162$. Figure 3.5 demonstrates that 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 ($r = - .118, p = .450$). There was no significant Phase × DERS interaction effect on CD scores, $F(1, 41) = 2.93, p = .095$, partial $\eta^2 = .067$. Examination of the underlying hemodynamic profile confirmed that the observed patterns of CO and TPR responding for individuals with greater difficulties in emotion regulation, during the negative task, were indicative of a vascular cardiovascular response.

**Challenge-threat Index**

**Comparison with HP values.** A chi-squared test of association confirmed that vascular and myocardial responses as classified by the HP-CD computational model were associated with threat and challenge responses (respectively) as classified by the challenge-threat index: negative task, $\chi^2 = 32.36, p < .001, w = .830$; neutral, $\chi^2 = 31.88, p < .001, w = .824$. This confirms that the HP-CD model and challenge-threat index share considerable statistical overlap.
Chapter 3, Study 2: Trait ER and CVR to Active Stress

**Emotion regulation style and indices of challenge-threat.** There was a significant Phase × Trait Reappraisal interaction effect on challenge-threat scores, $F(1, 40) = 6.87, p = .012$, partial $\eta^2 = .147$. Greater habitual use of reappraisal was associated with a greater challenge response to the negative-emotion task ($r = +.416, p = .006$), but unrelated to challenge-threat responding to the neutral task ($r = +.094, p = .556$). There was also a significant Phase × DERS interaction effect on challenge-threat scores, $F(1, 41) = 6.91, p = .012$, partial $\eta^2 = .144$. Greater emotion regulation difficulties were associated with a lower challenge response, that is, a more threat-oriented response, during the negative-emotion task ($r = -.300, p = .051$). Emotion regulation difficulties were not related to challenge-threat responding to the neutral task ($r = +.141, p = .367$). There was no significant Phase × Trait Suppression interaction effect on challenge-threat responses, $F(1, 40) = 0.04, p = .850$, partial $\eta^2 = .001$.

Results using the challenge-threat index mirror findings from the HP-CD computational model; individuals with greater emotion regulation difficulties demonstrated a threat-oriented cardiovascular response during the negative emotion task, while greater habitual use of reappraisal was associated with a more myocardial/challenge response. Both models confirmed trait suppression was not associated with patterns of hemodynamic responding.
Table 3.3.

*Phase × Trait ER interaction effects on CVR*

<table>
<thead>
<tr>
<th></th>
<th>Reappraisal</th>
<th></th>
<th>Suppression</th>
<th></th>
<th>DERS</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>F</td>
<td>(\eta^2)</td>
<td>p</td>
<td>F</td>
<td>(\eta^2)</td>
<td>p</td>
</tr>
<tr>
<td>SBP</td>
<td>1.77</td>
<td>.042</td>
<td>.186</td>
<td>4.75</td>
<td>.106</td>
<td>.016*</td>
</tr>
<tr>
<td>DBP</td>
<td>0.95</td>
<td>.023</td>
<td>.372</td>
<td>5.81</td>
<td>.127</td>
<td>.007**</td>
</tr>
<tr>
<td>HR</td>
<td>2.20</td>
<td>.052</td>
<td>.118</td>
<td>0.92</td>
<td>.022</td>
<td>.915</td>
</tr>
<tr>
<td>CO</td>
<td>2.85</td>
<td>.066</td>
<td>.077</td>
<td>1.34</td>
<td>.032</td>
<td>.269</td>
</tr>
<tr>
<td>TPR</td>
<td>7.99</td>
<td>.167</td>
<td>.005**</td>
<td>0.14</td>
<td>.004</td>
<td>.738</td>
</tr>
</tbody>
</table>

*Note.* **\(p < .01\), *\(p < .05\)*
Figure 3.3. Greater difficulties in emotion regulation were associated with lower CO and greater TPR reactivity in response to negative-emotion task. A median split was applied to difficulties in emotion regulation for demonstration purposes.
Table 3.4.

**Phase × DERS subscales interaction effects on CVR**

<table>
<thead>
<tr>
<th></th>
<th>Impulse</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
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<td></td>
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<td>$p$</td>
<td>$F$</td>
<td>$\eta^2_p$</td>
<td>$p$</td>
<td>$F$</td>
<td>$\eta^2_p$</td>
<td>$p$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>2.55</td>
<td>.059</td>
<td>.092</td>
<td>0.37</td>
<td>.009</td>
<td>.693</td>
<td>1.96</td>
<td>.046</td>
<td>.154</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DBP</td>
<td>3.11</td>
<td>.071</td>
<td>.056</td>
<td>1.12</td>
<td>.027</td>
<td>.331</td>
<td>2.87</td>
<td>.065</td>
<td>.069</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR</td>
<td>0.45</td>
<td>.011</td>
<td>.635</td>
<td>1.50</td>
<td>.035</td>
<td>.230</td>
<td>0.56</td>
<td>.014</td>
<td>.568</td>
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<tr>
<td>CO</td>
<td>8.86</td>
<td>.178</td>
<td>.001**</td>
<td>2.79</td>
<td>.064</td>
<td>.080</td>
<td>4.15</td>
<td>.092</td>
<td>.027*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TPR</td>
<td>8.47</td>
<td>.171</td>
<td>.004**</td>
<td>7.51</td>
<td>.155</td>
<td>.006**</td>
<td>4.20</td>
<td>.093</td>
<td>.041*</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Chapter 3, Study 2: Trait ER and CVR to Active Stress
Table 3.5.

*Associations between DERS subscales and CVR*

<table>
<thead>
<tr>
<th></th>
<th>Negative Task</th>
<th>Neutral Task</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CO</td>
<td>TPR</td>
</tr>
<tr>
<td>Impulse</td>
<td>-0.464</td>
<td>0.002*</td>
</tr>
<tr>
<td>Accept</td>
<td>-0.261</td>
<td>0.091</td>
</tr>
<tr>
<td>Strategies</td>
<td>-0.305</td>
<td>0.047*</td>
</tr>
</tbody>
</table>

*Note.* Significant *p* values (< .05) denoted by *
Figure 3.4. Individuals scoring high in habitual reappraisal demonstrated a myocardial hemodynamic profile, while individuals scoring low in trait reappraisal demonstrated a vascular response during the negative task. High trait reappraisers exhibited less CD during the negative task. A median split was applied to reappraisal scores for demonstration purposes.
Figure 3.5. Individuals with greater difficulties in emotion regulation demonstrated a vascular response, while individuals scoring low in difficulties demonstrated a myocardial response during the negative task. There were no differences in CD. A median split was applied to difficulties in emotion regulation for demonstration purposes.
Discussion

The present study confirmed that greater habitual use of suppression was associated with greater blood pressure reactivity to acute stress, while habitual use of reappraisal was not associated with blood pressure reactivity. However, trait reappraisal was associated with a more adaptive cardiovascular response pattern to the negative-emotion stressor, indexed by greater CO and lower TPR reactivity; a myocardial response. Individual differences in emotion regulation style did not influence the psychological experience of the stressor, with all participants reporting similar levels of stress, anxiety, and affect after the tasks. Overall, the present study provides evidence that the propensity to engage in reappraisal and suppression influences CVR to active stress.

Consistent with prior research instructing suppression use, this study confirmed that greater habitual use of suppression was associated with increased SBP and DBP responding to the tasks (for a review see; Gross, 2015; Mauss & Gross, 2004). Interestingly, trait suppression was associated with exaggerated blood pressure in response to both the neutral-emotion and negative-emotion task. In light of research demonstrating exaggerated blood pressure reactivity to laboratory stressors is predictive of future CHD, it 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 (analyses not shown) nor due to differences in emotional experience of the task (assessed by post-task ratings of affect, anxiety, and stress), thus strengthening the finding that differences in blood pressure reactivity were a result of habitual suppression.

Habitual use of reappraisal, however, did not influence blood pressure reactivity, despite previous research indicating it predicts lower SBP and DBP responses to an
anger-provocation task (Memedovic et al., 2010). It may be the case that differences in the tasks employed led to these conflicting results. The present study used a well-controlled speech task, designed to remove the interpersonal aspect of anger-provocation and self-relevant speech tasks (such as the TSST and the like) used previously. This differs from previous tasks used in the literature. However, it is difficult to draw conclusions, given the paucity of research examining emotion regulation and blood pressure reactivity; a surprising omission given the utility of blood pressure reactivity in predicting the development of CHD (e.g., Carroll et al., 2001; Light et al., 1992).

Nevertheless, our findings suggest that reappraisal does not offer any benefits in terms of buffering blood pressure reactivity. However, when we examined the underlying hemodynamic profile, we see that greater habitual use of reappraisal was associated with greater CO and lower TPR responding to the negative task; a myocardial/challenge-oriented response.

As outlined in Chapter 1, a myocardial response pattern to an active stress task is hypothesised to signal a more adaptive response (Mendes, Reis, Seery, & Blascovich, 2003; Tomaka et al., 1997). The observed results mirror findings from studies employing trait reappraisal (Mauss, Cook, Cheng, et al., 2007) and instructed reappraisal (Jamieson et al., 2012, 2013). However, the results of this study suggest the effects of trait reappraisal may be limited to negative-emotion contexts. In support of this hypothesis, previous studies reporting a relationship between reappraisal and more adaptive cardiovascular responding have employed stress tasks that could be considered negative; throughout the tasks participants received negative feedback (e.g., Jamieson et al., 2012, 2013; Mauss, Cook, Cheng, et al., 2007). This suggests that when faced with a stressful situation (with negative-emotion content) habitual use of reappraisal offers a protective
function in terms of physiological arousal. Further research is needed to examine the boundary conditions under which reappraisal influences CVR.

The finding that difficulties in emotion regulation only influenced CVR during the negative-emotion task also suggests a role for context. During this task, greater difficulties in emotion regulation were associated with a vascular response profile; indexed by greater TPR and lower CO reactivity. 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); a homeostatic response (Obrist, 1981). This homeostatic relationship was not observed for individuals with greater difficulties regulating their emotions. The association between emotion regulation difficulties and vascular responding warrants further attention given vascular responsiveness has been implicated in the development of cardiovascular disease (e.g., 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. This conflicts with research using instructed reappraisal; reappraisal use typically leads to less NA and more PA. However, in such studies, participants are explicitly given instructions such as “think about what you are seeing in such a way that you feel less negative emotion”, then asked to report affect 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 may offer a more valid method of assessing the impact of emotion regulation strategy use on the psychological experience of stress.
A secondary aim of this study was to compare how the HP-CD model and the challenge-threat index classify patterns of CO and TPR responding. This study provides evidence that these two computations share considerable conceptual overlap. Participants classified as demonstrating a vascular response, or myocardial response, by the HP-CD model were also found to demonstrate a challenge, or threat response (respectively), by the challenge-threat index, and vice-versa. Although each model stems from a distinct literature, they appear to reflect the same construct.

The reported results are strengthened by the employment of a standardized laboratory stress paradigm. The inclusion of an acclimatization period, a Vanilla resting baseline (Jennings et al., 1992), and more sophisticated measures of cardiovascular responding add weight to our results. In addition, the use of an active task (a speech task) is a notable strength of the study. 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 past emotion regulation research where the focus tends to be on passive stressors, such as emotion-eliciting film clips or negative imagery.

The observed associations between individual differences in emotion regulation and CVR are limited by some methodological issues. First, although the results were in the expected directions and the sample size was sufficient to detect effects, there is the possibility of type I error due to the number of analyses conducted and the relatively small sample size employed. Similarly, the sample employed was homogenous in terms of age and ethnicity. 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.

The aim of this study was to examine the relationship between trait emotion
regulation and CVR in a context that facilitates active coping; however, the observed
results may not replicate in a more uncontrollable (passive) context. Within the emotion
regulation literature there is scant research using the individual difference approach to
examine the physiological consequences of emotion regulation – in both active and
passive coping contexts. Future research needs to examine the influence of the type of
stressor (active or passive) on the relationship between emotion regulation and
cardiovascular responding.

It appears individual differences in emotion regulation have distinct influences on
CVR. We found clear patterns of exaggerated blood pressure responses for high trait
suppressors to both tasks, and a more adaptive cardiovascular response pattern for high
trait reappraisers (albeit only in response to the negative task). This strengthens the
findings of past research reporting similar results; research that often relies on an atypical
laboratory stress paradigm and usually instructs the use of a strategy. We provide support
for the hypothesis that individual differences in the tendency to engage in these strategies
has implications for CVR.
Chapter 4, Study 3: Validity of a Novel Passive Stressor

CHAPTER 4

Establishing the Validity of a Novel Passive Stress Task

Introduction

Active and passive coping contexts are characterized by the opportunity afforded to individuals to influence performance or the outcomes of the situation (e.g., Obrist, 1981; Schneiderman & McCabe, 1989). Active stress tasks, such as speech or mental arithmetic tasks, require active engagement and participants can alter their performance on the task. In contrast, passive tasks require that participants simply endure the task; they cannot change the results of the task (Sherwood et al., 1990). Passive tasks are further defined as either physical or psychological stressors. For example, within the cardiovascular reactivity literature, a common physical passive stressor is the cold pressor task (e.g., Brindle, Whittaker, Bibbey, Carroll, & Ginty, 2017; Tuomisto et al., 2005; Vella & Friedman, 2007). Psychological passive stress tasks usually involve viewing distressing film clips or images (e.g., Kim & Hamann, 2012; Tuomisto et al., 2005; Zakowski, Cohen, Hall, Wollman, & Baum, 1994).

It is posited that active and passive tasks elicit different patterns of cardiovascular responding due to preferential activation of beta- and alpha-adrenergic receptors, respectively (see; Schneiderman & McCabe, 1989). Active tasks are characterized by greater beta-adrenergic activation, indexed by increased blood pressure, HR, and CO. In contrast, passive tasks appear to elicit less beta-adrenergic activation compared to active tasks, and more alpha-adrenergic activity, resulting in greater vascular tone (indexed by
increases in TPR). As a result, blood pressure increases to passive tasks are accompanied by lower CO, and elevated TPR (Bolli et al., 1981; Hurwitz et al., 1993; Obrist, 1981; Obrist et al., 1979; Saab et al., 1993; Sherwood et al., 1986). These patterns of responding are indicative of stimulus-response specificity and have been termed as a challenge-oriented response (active coping) or a threat-oriented response (passive coping), respectively (for a review see; Schneiderman & McCabe, 1989). Indeed, a myriad of research has demonstrated that active tasks have greater effects on blood pressure, CO, and HR, while passive tasks have a greater influence on TPR (e.g., Winzer et al., 1999). Speech tasks have been found to elicit greater SBP and DBP reactivity relative to the cold pressor task and viewing film-clips depicting stressful life events (Nyklicek, Bosch, & Amerongen, 2005). Similarly, mental arithmetic tasks elicit greater SBP, DBP, and HR reactivity compared to watching surgical film-clips (Patterson et al., 1994) and relative to the cold pressor task (Isowa, Ohira, & Murashima, 2004). In particular, the cold pressor task has highlighted the prominent role of vascular responding in driving blood pressure responses during passive tasks; characterized by little-to-no change in CO and heightened TPR (Saab et al., 1992; Saab et al., 1993).

More recently, research has extended these findings and examined the underlying hemodynamic profile elicited by active and passive stressors through the application of the HP-CD model (Gregg et al., 2002). Only a handful of studies have applied this model, so it is difficult to draw clear conclusions. Active tasks (mental arithmetic tasks) have been found to elicit both a myocardial response (Gregg et al., 2002) and a mixed hemodynamic response, where blood pressure is driven by synergistic increases in TPR and CO (Howard et al., 2011; Ottaviani et al., 2006). In contrast, physical passive tasks such as the handgrip stressor (Ottaviani et al., 2006) and the cold pressor task (Gregg et
al., 2002) elicited vascular response patterns. Although during the handgrip stressor only women demonstrated a vascular response; men showed a mixed response profile. To our knowledge no study has applied the HP-CD model to a purely psychological passive stress task. However, in response to a more cognitive passive task, an experimental sleep restriction manipulation, participants exhibited a vascular hemodynamic profile (James & Gregg, 2004).

Limitations of Current Passive Stressors

A number of weakness exist with the types of passive stress tasks employed by previous studies. For example, due to the nature of some physical passive stressors, such as the cold pressor task, it is likely that the vascular stress response observed is due to vasoconstriction associated with immersing a body part in cold water; rather than a psychological stress response. Exposure of skin to cold temperatures has been shown to trigger a thermoregulatory reflex leading to increased TPR (Epstein, Stampfer, Beiser, Goldstein, & Braunwald, 1969). Outside of the cardiovascular reactivity literature the cold pressor is often used as a pain tolerance test (e.g., Arendt-Nielsen & Lautenbacher, 2004), and individual differences in TPR and CO responses to the task are postulated to be a result of differences in perceived pain (Peckerman et al., 1994). Arguably, the observed changes in TPR may be a result of physical, rather than stress-related, mechanisms. The use of psychological passive stress tasks therefore offers an advantage over physical stress tasks.

Although passive viewing of film-clips and imagery has been associated with changes in affect (Gross, 1998b; Lang et al., 2008; Shiota & Levenson, 2009), such tasks may not create a coping context representative of real-life stressful situations. Film-clips lack dimensions inherent in day-to-day emotion-eliciting situations, such as motivational,
social, and evaluative aspects (Aldao, 2013), thus limiting our ability to draw accurate conclusions about cardiovascular response patterns elicited during passive stress. Furthermore, reported changes in affect may be a result of response bias. For example, if participants viewed a sad film-clip and subsequently were asked to rate the degree they feel “sad” participants may report the emotions they expect the experimenter wants them to feel, rather than reporting the actual intensity of emotions felt.

**More Novel Attempts to Create a Passive Stress Task**

To overcome such limitations, a handful of studies have designed and implemented more novel psychological passive tasks. Typically, the level of control participants have over the task is manipulated by employing an active version of the stress task followed by a passive version of the task. For example, Sherwood et al. (1990) asked participants to complete a reaction time task in pairs; quicker reaction times resulted in a greater monetary bonus. Each participant took part in both the active phase (making the reaction time response) and the passive phase (watching a team-mate make the response). Similar to previous research, the active phase of this task elicited greater SBP, DBP, HR, and CO reactivity in comparison to the passive phase. As expected, the active task was associated with mean decreases in TPR, while the passive task was associated with a mean increase in TPR.

Another methodology involves video-recording participants during an active task, such as singing song lyrics or completing a speech task, and later asking participants to view this video-recording (Harris, 2001; Hartley, Ginsburg, & Heffner, 1999; Schwerdtfeger & Rosenkaimer, 2011; Soye & O’Súilleabháin, 2019). A notable limitation with studies employing this paradigm is that CVR to the active and passive tasks are not directly compared (e.g., Harris, 2001; Hartley et al., 1999; Schwerdtfeger &
Rosenkaimer, 2011). For example, Schwerdtfeger and Rosenkaimer (2011) reported that both the act of completing, and viewing, the speech task elicited increases in SBP, DBP, and HR; however, this study did not compare the magnitude of reactivity elicited by each task. While Soye and O'Súilleabháin (2019), compared SBP, DBP, and HR responses to the speech and video elements of the paradigm (finding the active phase elicited greater SBP, DBP, and HR reactivity), this study did not report CO or TPR responses nor did it apply the HP-CD computational model to examine the underlying hemodynamic profile of responding.

A different approach was taken by Hartley et al. (1999); participants completed a pre-scripted speech task and a week later watched the video. However, in this study cardiovascular responses to the speech and video task were not compared. Rather, the level of control participants had over the video-viewing phase was manipulated; creating an “active viewing” task and a “passive viewing” task. Both groups were informed that they would watch the video-clip and afterwards it would be evaluated by external judges. One group were given the opportunity to mark segments of the clip they wished to reshoot before it was evaluated (active group), the other group was not afforded this opportunity (passive group). A third group were told to simply watch the clip, and the clip would be erased afterwards (passive control). All groups demonstrated elevations in SBP and DBP from baseline. The active viewing group showed an increase in CO, and no change in TPR. In contrast, the passive groups demonstrated decreases in CO and increases in TPR. Interestingly, there were also differences between the two passive conditions; those led to believe the clip would be evaluated afterwards had higher DBP, TPR, and CO responses than the control group; highlighting the influence of (anticipated) evaluation on cardiovascular responding.
Chapter 4, Study 3: Validity of a Novel Passive Stressor

The potential confound of evaluation is also inherent in the studies employing the more typical active speech task/passive video-viewing task paradigm. Within these studies participants watched the video-clip with the experimenter observing (Schwerdtfeger & Rosenkaimer, 2011; Soye & O’Súilleabháin, 2019), or with the experimenter and confederates present (Harris, 2001). Therefore, it is unclear if the reported findings are a result of social-evaluation apprehension, or due to viewing the clip of the speech task.

Although this novel psychological passive stressor attempts to address issues inherent in other passive tasks, it also has limitations. First, most research employing this paradigm does not compare the profile of physiological responding elicited by the active and passive versions of these tasks. Second, the focus of these studies is on blood pressure responses (SBP and DBP), rather than the parameters which underlie blood pressure responding (CO and TPR). Third, the cardiovascular response to the video-viewing task may be confounded by the presence of the experimenter.

The Current Study

The current study therefore sought to address limitations with previous passive stressors and test the validity of the passive stress task reported upon by Soye and O’Súilleabháin (2019). This research examines the effects of two laboratory stressors on cardiovascular responding; completing a speech task and viewing the video-recording of the speech task, but *without* the experimenter also watching it. Participants first completed a speech task, then a year later were invited back into laboratory to complete the passive stress task; employing a within-subjects design. It is hypothesized that each task will elicit a distinctive cardiovascular response. Both tasks are expected to elicit increases in blood pressure; however, it is hypothesized that this increase will be greater
in response to the active task. The active task is expected to elicit greater HR and CO reactivity, while the passive task will elicit greater TPR reactivity, indicative of a vascular response. The HP-CD model will be applied to examine the underlying hemodynamic response to each task. It is anticipated that the active task will elicit a myocardial or mixed response profile, while the passive task will elicit a vascular response profile. This report describes the design of this passive stress task and seeks to test the construct validity of the task in terms of (a) the task eliciting a physiological stress response, and (b) the task eliciting a pattern of autonomic activity distinct from the active stressor.

Methodology

Design

The current study employed a within-subject design. The within-subjects variables were phase (baseline, task) and task type (active, passive). The dependent variables were self-reported affect and cardiovascular parameters; SBP, DBP, HR, CO, and TPR.

Participants

Fifty-two participants completed a speech task (Study 2) and were later contacted to take part in the passive phase of the experiment; that is, to watch the video of themselves completing the active task. A total of 26 participants completed both tasks. One participant had resting blood pressure that could be classified as hypertensive, (SBP/DBP > 140/90 mmHg), so was excluded from all analyses. The present results report on a final sample of 25 participants (17 females, 8 males), aged 18-25 (\( M = 20.40, SD = 1.66 \)). Participants indicated they were non-smokers, reported good health, no
history of cardiovascular disease and were not taking medication known to affect blood pressure. 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.

A priori power analyses were conducted using G*Power (Faul et al., 2007) to establish sample size requirements to detect large and medium effects. Using an alpha of .05, and power of .80, in order to detect a large effect ($d = .80$) a sample size of 12 was needed, and to detect a medium effect ($d = .50$) a sample of 27 was required. As noted, 25 participants completed both visits, meaning the sample had sufficient power to detect medium to large effects. A post-hoc power analysis found that the recruited sample has 98.74% power to detect large effects and 78.34% power to detect medium effects. Effect sizes are reported throughout.

Materials and Apparatus

Cardiovascular Assessment.

As reported in Study 2, cardiovascular parameters were measured non-invasively using the Finometer PRO (Finapres Medical Systems BV, BT Arnhem, The Netherlands).

Measures of affect.

Positive Negative Affect Schedule (PANAS). The PANAS (Watson et al., 1988), described in Chapter 3, assessed positive affect and negative affect. The PANAS demonstrated good internal reliability at baseline and post-task for both visits, Cronbach’s $\alpha$ was $\geq .68$ for each scale.
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**Self-report questionnaires.** Participants were asked, pre- and post-task to rate on a ten-point Likert scale from 1 (*not at all*) to 10 (*extremely*) the extent to which they felt stressed or anxious.

**Stressor tasks.**

**Active stress task.** The socio-evaluative speech task described in Study 2 was employed. For the purpose of this study, CVR to the first task encountered (whether neutral or negative words) was used\(^2\), to avoid any effects due to habituation.

**Passive stress task.** The video of the participant completing the speech task was used as a passive stress task. The video-clip from the neutral emotion block of words was selected. This builds on paradigms previously developed, where participants were filmed completing an active task and later shown this film-clip to elicit a passive coping context (Harris, 2001; Hartley et al., 1999; Soye & O'Súilleabháin, 2019).

**Procedure**

On both visits, participants visited the laboratory at an assigned timeslot between 8:30am and 2pm to minimize the impact of diurnal changes to blood pressure. The laboratory paradigm employed is identical to that reported in Study 2. On both visits, participants were seated at a desk with a personal computer screen, separated from the experimenter by a partition. Demographic information was recorded, and participants completed a 20-minute acclimatization period. During this time, participants completed a series of psychometric scales and were given neutral reading material. Participants were then connected to the Finometer PRO. The Finometer cuff was attached to the middle

\(^2\) There was a significant difference in SBP reactivity between the negative and neutral words, \(t(47) = -2.26, p = .028, [-4.77, -0.28]\). However, there was no differences between DBP, HR, CO, or TPR reactivity, all \(ps > .052\).
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finger of the non-dominant hand. Resting cardiovascular measures were taken during an official 10-minute baseline period while participants completed the affective scales. Again, reading material was provided to lower the risk of potential boredom and/or rumination arousal, employing the Vanilla resting baseline as recommended by Jennings et al. (1992). Following this period, on visit 1, participants were verbally informed that words would appear on the computer screen in front of them and to talk about each word for as long as possible. The experimenter evaluated when to change the word presented; after three seconds of silence (as measured by a stopwatch), or repetition. Participants were instructed to look at the camcorder as much as possible during the task as the recorded video-clips would later be analysed for body language; this instruction was to heighten the socio-evaluative aspects of task. The task lasted for five minutes. On visit 2, participants were instead verbally informed that the video-clip would be played on the screen in front of them and to simply watch it. They then viewed themselves completing the task from visit 1 on-screen for five minutes. Following completion of the laboratory session at both visits, participants completed some affective rating scales, were immediately 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 preceding each task (active and passive), and during each task (active and passive) were calculated and are presented in Table 4.2. Internal consistency for each cardiovascular variable was excellent with Cronbach’s $\alpha > .89$. TPR values did not meet the assumption of normality, even after treatment of outliers (neither the
removal of outliers nor transformation of outliers to $2SD$ above/below the mean achieved normality). Therefore, non-parametric tests were employed when conducting analyses involving TPR values.

A series of $2 \times 2$ within-subjects ANOVAs were conducted to compare reactivity between the active and passive tasks. The first within-subjects factor was task type; active and passive. The second within-subjects factor was phase; baseline and task. To examine if each task successfully elicited a cardiovascular response paired samples $t$-tests were conducted.

HP and CD values for each task were calculated using the computational method proposed by Gregg et al. (2002). One-sample $t$-tests were conducted to examine HP and CD changes in response to the active and passive task. HP values were not normally distributed, therefore HP scores $2SD$ above and below the mean were transformed to the $2SD$ above/below the mean to improve normality. This resulted in two adjusted HP scores for both tasks. Likewise, CD scores in response to the passive task were not normally distributed, one CD score was transformed to $2SD$ above the mean to improve normality.

Physiological indices of challenge-threat were computed consistent with the approach used in the biopsychosocial model literature (e.g., Seery et al., 2010). Similar to Chapter 3, challenge-threat scores were transformed and compared with indices of challenge-threat categorised from HP scores, to confirm these measures overlap conceptually.

Effect sizes for correlations are presented as $r$, with values of .10, .30, and .50 taken to be indicative of small, medium, and large effect sizes respectively (J. Cohen, 1988). Effect sizes for repeated measures and one-sample $t$-tests were calculated using
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G*Power, and are presented as $d$, with values of .20, .50, and .80 indicative of small, medium and large effect sizes.

**Psychological Experience of the Stress Task**

Paired samples $t$-tests found no difference in ratings of stress, NA, and PA between baseline and post-active task, all $p$s > .077. However, ratings of anxiety significantly increased from baseline ($M = 3.16, SD = 1.72$) to post-task ($M = 4.24, SD = 2.20$), $p = .023$, $d = 0.49$. Paired samples $t$-tests found no difference in ratings of stress, anxiety, NA, and PA between the baseline and post-passive task period, all $p$s > .618. Full results are presented in Table 4.3.

Change scores were computed by subtracting reported affect during the task from the preceding baseline. Paired samples $t$-tests, using the computed change scores, found no differences between the active and passive task in terms of reported stress, anxiety, NA, or PA: stress, $t(24) = -1.51, p = .145$; anxiety, $t(24) = -1.78, p = .088$; NA, $t(23) = 0.76, p = .456$; PA, $t(23) = 1.46, p = .159$. The current findings suggest that there was no difference in the psychological experience of each task.

**Resting cardiovascular assessment**

Repeated measures $t$-tests found significant differences in resting SBP and DBP between the active and passive baselines: SBP, $t(24) = 3.35, p = .003$, $d = 0.67$, 95% CI [2.07, 8.69]; DBP, $t(24) = 4.17, p < .001$, $d = 0.83$, [2.94, 8.72]. SBP and DBP were significantly lower during the baseline preceding the passive task (the second laboratory visit); potentially due to habituation to the laboratory environment.

**Cardiovascular reactivity**

A series of $2 \times 2$ within-subjects ANOVAs were conducted to compare reactivity between the active and passive tasks. The first within-subjects factor was task type;
active and passive. The second within-subjects factor was phase; baseline and task. The ANOVAs confirmed main effects for phase on SBP, $F(1, 24) = 42.64, p < .001$, partial $\eta^2 = .64$, DBP, $F(1, 24) = 60.97, p < .001$, partial $\eta^2 = .72$, HR, $F(1, 24) = 17.76, p < .001$, partial $\eta^2 = .71$, CO, $F(1, 24) = 16.55, p < .001$, partial $\eta^2 = .41$, and TPR, $F(1, 24) = 4.89, p < .001$, partial $\eta^2 = .17$. This confirmed that overall, levels were higher during the tasks compared to baseline, as can be seen in Table 4.2.

There were main effects for task type on SBP, $F(1, 24) = 29.27, p < .001$, partial $\eta^2 = .55$, and DBP, $F(1, 24) = 32.91, p < .001$, partial $\eta^2 = .58$. As can be seen in Table 4.2, across the two phases of the tasks, SBP and DBP levels were higher during the active task. There were no main effects for task type on HR, CO, or TPR (all $p$s > .068).

There were Phase × Task type interaction effects on SBP, $F(1, 24) = 29.27, p < .001$, partial $\eta^2 = .55$, DBP, $F(1, 24) = 35.95, p < .001$, partial $\eta^2 = .60$, HR, $F(1, 24) = 57.55, p < .001$, partial $\eta^2 = .71$, CO, $F(1, 24) = 9.57, p = .005$, partial $\eta^2 = .298$, but not TPR, $F(1, 24) = .03, p = .858$.

**Comparison of Active and Passive Task**

As the factorial ANOVAs did not allow direct comparison of patterns of reactivity associated with passive and active tasks, it was decided to examine each task separately, using paired samples $t$-tests.

**Active task.** The active task resulted in an increase in SBP, $t(24) = -7.10, p < .001$, $d = 1.42, [-19.19, -10.55]$, DBP, $t(24) = -8.34, p < .001$, $d = 1.67, [-11.75, -7.09]$, HR, $t(24) = -6.89, p < .001$, $d = 1.08, [-7.41, -3.99]$, and CO, $t(24) = -4.36, p < .001$, $d = 0.79, [-0.97, -0.35]$ from baseline to task. Means and standard deviations are presented in Table 4.2. There was no change in TPR from baseline ($Md = 0.86$) to task ($Md = 0.88$). z
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SBP, DBP, HR, and CO all increased from baseline to task in response to the active task; however, the active task had no effect on TPR.

**Passive task.** The passive task resulted in a significant increase in SBP, \( t(24) = -3.69, p = .001, d = 0.74, [-7.95, -2.25] \) and DBP, \( t(24) = -3.91, p = .001, d = 0.79, [-4.47, -1.38] \) from baseline to task. TPR also significantly increased from baseline (\( Md = 0.84 \)) to task (\( Md = 0.87 \)), \( z = -2.19, p = .028, r = .31 \). There were no significant changes in HR, \( t(24) = 1.64, p = .114, d = 0.33, [-0.25, 2.15] \) or CO, \( t(24) = -1.05, p = .306, d = 0.21, [-0.32, 0.11] \). Means and standard deviations are presented in Table 4.2. The passive task elicited increases in SBP, DBP, and TPR, but had no effect on HR or CO.

**Magnitude of reactivity.** Reactivity scores were calculated separately for each task by subtracting baseline cardiovascular values from task values and are presented in Table 4.1. A series of repeated measures \( t \)-tests, using the computed reactivity scores, indicated that SBP, DBP, HR, and CO reactivity to the *active* task was significantly greater than reactivity to the *passive* task: SBP, \( t(24) = 5.43, p < .001, d = 1.09, [6.05, 13.49] \); DBP, \( t(24) = 5.99, p < .001, d = 1.20, [4.26, 8.73] \); HR, \( t(24) = 7.59, p < .001, d = 1.08, [4.84, 8.46] \); CO, \( t(24) = 3.21, p = .004, d = 0.79, [0.21, 0.96] \). There was no difference in TPR reactivity elicited by the tasks, \( z = -.87, p = .375 \).

**Hemodynamic profile**

The passive task had a mean HP of .01 (\( SD = .04 \)) and a mean CD of .01 (\( SD = .02 \)). The active task had a mean HP of -.02 (\( SD = .05 \)) and mean CD of .04 (\( SD = .02 \)). One-sample \( t \)-tests indicated that both tasks elicited significant CD changes: passive task, \( t(24) = 2.92, p = .007, d = 0.58, [0.00, 0.02] \); active task; \( t(24) = 9.19, p < .001, d = 1.84, [0.03, 0.04] \). In terms of HP, the passive task did not elicit significant HP change, which is indicative of a mixed response profile, \( t(24) = 1.07, p = .297, d = 0.21, [-0.01, 0.03] \). In
contrast, the active task appears to have elicited a myocardial response profile, $t(24) = -2.00, p = .029$ (one-tailed), $d = 0.40, [-0.04, 0.00]$, indicated by the negative $t$-value. A one-tailed $t$-test confirmed that HP values were significantly higher than during the active task, $t(24) = 1.78, p = .044$, confirming that the active task was associated with a myocardial profile.

**Challenge-threat index**

**Comparison of HP values and challenge-threat values.** As outlined in Chapter 3, indices of challenge and threat as determined by the HP-CD model and the challenge-threat index were transformed to create categorical indices of challenge and threat. A chi-squared test of association confirmed that the categories created by both computations were significantly associated: passive task, $\chi^2 = 10.86, p = .001, w = .659$; active task, $\chi^2 = 7.64, p = .006, w = .553$. There was no significant difference in indices of challenge-threat elicited by the tasks, $t(25) = 0.01, p = 1.00, [-0.39, .0.39]$.

Table 4.1.

*Mean (with SDs) reactivity scores to the passive and active task*

<table>
<thead>
<tr>
<th></th>
<th>Passive Task</th>
<th></th>
<th>Active Task</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
<td>$M$</td>
<td>$SD$</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>5.10</td>
<td>6.91</td>
<td>14.87*</td>
<td>10.47</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>2.92</td>
<td>3.74</td>
<td>9.42*</td>
<td>5.65</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>-0.95</td>
<td>2.90</td>
<td>5.70*</td>
<td>4.13</td>
</tr>
<tr>
<td>CO (lpm)</td>
<td>0.03</td>
<td>0.36</td>
<td>0.62*</td>
<td>0.74</td>
</tr>
<tr>
<td>TPR (pru)</td>
<td>0.30</td>
<td>0.09</td>
<td>0.01</td>
<td>0.07</td>
</tr>
</tbody>
</table>

*Note. The magnitude of SBP, DBP, HR, and CO reactivity to the active task was greater than reactivity to the passive task. There was no difference in TPR reactivity.*
### Table 4.2.

*Mean (with SDs) cardiovascular parameters at each phase*

<table>
<thead>
<tr>
<th></th>
<th>Passive Task</th>
<th></th>
<th>Active Task</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Phase</td>
<td></td>
<td>Phase</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Baseline</td>
<td>Task</td>
<td>Baseline</td>
<td>Task</td>
</tr>
<tr>
<td></td>
<td><em>M</em></td>
<td><em>SD</em></td>
<td><em>M</em></td>
<td><em>SD</em></td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>115.35</td>
<td>9.42</td>
<td>120.44*</td>
<td>13.99</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>66.96</td>
<td>5.78</td>
<td>69.88*</td>
<td>8.32</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>78.98</td>
<td>13.78</td>
<td>78.03</td>
<td>13.74</td>
</tr>
<tr>
<td>CO (lpm)</td>
<td>6.53</td>
<td>1.62</td>
<td>6.64</td>
<td>1.83</td>
</tr>
<tr>
<td>TPR (pru)</td>
<td>0.85</td>
<td>0.16</td>
<td>0.88*</td>
<td>0.16</td>
</tr>
</tbody>
</table>

*Note.* The passive task elicited increased SBP, DBP, and TPR responses. The active task elicited increased SBP, DBP, HR, and CO responses. Significant increases from baseline are denoted by *. 
Table 4.3.

*Differences between baseline and post-task ratings of affect*

<table>
<thead>
<tr>
<th></th>
<th>Passive Task</th>
<th></th>
<th></th>
<th>Active Task</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>t</td>
<td>df</td>
<td>p</td>
<td>Lower CI</td>
<td>Upper CI</td>
<td>t</td>
</tr>
<tr>
<td>Stress</td>
<td>-0.09</td>
<td>24</td>
<td>.928</td>
<td>-0.94</td>
<td>0.86</td>
<td>-1.85</td>
</tr>
<tr>
<td>Anxiety</td>
<td>-0.21</td>
<td>24</td>
<td>.836</td>
<td>-0.87</td>
<td>0.71</td>
<td>-2.44</td>
</tr>
<tr>
<td>NA</td>
<td>-0.51</td>
<td>24</td>
<td>.618</td>
<td>-2.12</td>
<td>1.29</td>
<td>-0.80</td>
</tr>
<tr>
<td>PA</td>
<td>0.41</td>
<td>24</td>
<td>.685</td>
<td>-1.85</td>
<td>2.76</td>
<td>1.85</td>
</tr>
</tbody>
</table>

*Note.* * denotes a significant $p$ value ($< .05$)
Chapter 4, Study 3: Validity of a Novel Passive Stressor

Discussion

The present study confirmed that the passive stress task employed was successful in eliciting a physiological stress response. Watching the video-recording resulted in observable increases in blood pressure; changes driven by an increase in TPR responding (with little or no change in CO). This hemodynamic response pattern is suggestive of a more vascular response. While this pattern of responding is consistent with reported cardiovascular responses to other, more traditional, passive stressors such as the cold pressor task and film-clip stimuli (e.g., Hurwitz et al., 1993; Saab et al., 1993; Sherwood et al., 1990; Winzer et al., 1999), the current task controls for potential confounds associated with earlier operationalisations of passive stress. Furthermore, this response pattern aligns with the hypothesis that blood pressure responses during passive tasks are driven primarily by alpha-adrenergic sympathetic activation and less beta-adrenergic activation (Obrist, 1981). In contrast, the active task elicited a cardiovascular response marked by cardiac activation with observable increases in blood pressure, HR, and CO, with little or no change in TPR. As in Study 2, HP values and challenge-threat index values appear to be measuring the same construct.

A comparison of the magnitude of cardiovascular reactivity elicited by each task confirmed that the active task elicited greater SBP, DBP, HR, and CO reactivity relative to the passive stress task. Research comparing active and passive stressors consistently report active tasks elicit greater cardiovascular reactivity than passive tasks; strengthening the construct validity of the current passive stress task (e.g., Nyklicek et al., 2005; Patterson et al., 1994; Soye & O'Súilleabháin, 2019). Notably, the active and passive tasks did not differ in terms of task appraisals. There was no difference in the
We extended past research by applying the HP-CD model proposed by Gregg et al. (2002) to examine the underlying hemodynamic response profile. As expected, the active task elicited a more myocardial hemodynamic profile, thought to indicate a challenge-oriented response (Mendes et al., 2003; Tomaka et al., 1997). We expected the passive task to elicit a vascular response profile; however, a mixed hemodynamic profile was observed. It is worth noting that not much research has examined cardiovascular responding during passive stress using the HP-CD model. Although a vascular response was noted in three studies (Gregg et al., 2002; James & Gregg, 2004; Ottaviani et al., 2006), in the latter only women demonstrated a vascular response; men had a mixed hemodynamic profile. Furthermore, two of these studies used passive tasks that were physical in nature, not psychological. It is possible that task differences led to the observed responses. Future research should employ psychological passive tasks, such as the current stressor, in order to elucidate this relationship.

It must be acknowledged that the sample size of the current study is small (\(N = 25\)) and should be taken into consideration when interpreting the reported results. However, due to the implementation of a repeated measures design the current study had sufficient power to detect large effects and, to a degree, detect medium effects. Furthermore, the observed cardiovascular responses were in the hypothesised directions. It is also worth noting that a similar sample size has been employed by past research comparing active and passive stressors (20-30 participants; Bosch et al., 2001; Harrison et al., 2000; Hubert & de Jong-Meyer, 1991; Hurwitz et al., 1993; Isowa et al., 2004; Lovallo et al., 1985; Saab et al., 1992; Winzer et al., 1999). Future research is needed to
replicate the validity of this novel stress task in a larger sample and include a more diverse sample in terms of ethnicity and age.

It must also be noted that the order of tasks could not be counterbalanced. Viewing the video-clip of the speech task was dependent on the speech being recording beforehand. Therefore, we cannot claim the results were not influenced by order effects or familiarity with the experimental procedure/laboratory. To minimise this, the two visits were conducted a year apart rather than within a single session. This strengthens our findings, in that the results are less likely to be a result of habituation.

The observed pattern of results demonstrates the utility of using this paradigm to elicit a passive coping context. First, the current passive stress task was successful in eliciting a notable cardiovascular stress response. Second, participants’ psychological experience of the task (in terms of NA, PA, perceived stress, and anxiety) was no different to their experience during a well-established active stress task (Hughes & Callinan, 2007; O’Súilleabháin et al., 2018). Third, the task elicited a pattern of cardiovascular responding consistent with other more traditional passive stress tasks. Blood pressure increased, and this increase was driven by vascular, rather than cardiac responding. This extends research previously employing this paradigm (Schwerdtfeger & Rosenkaimer, 2011; Soye & O’Súilleabháin, 2019). Fourth, in the present study participants watched the task alone so any demonstratable changes in cardiovascular responding were a result of watching the video-clip, not a result of evaluation apprehension; a confound with previous studies employing this method. Fifth, examination of the underlying hemodynamic profile of the stressors was somewhat consistent with past literature; the active task elicited a myocardial profile and the passive task elicited a mixed response.
Most importantly, the current passive task offers several advantages over previous tasks considered to elicit passive coping. The psychological nature of the stressor ensures that observed differences in physiological responses were due to changes in cognitive and motivational states, rather than external physical stimuli (such as during the cold pressor task). Likewise, the task presented participants with a more socially relevant experience compared to watching an excerpt from a film or viewing negative imagery. The self-relevant aspect of the task strengthens the ecological validity of the coping context, beyond that provided by previous passive stressors. The presented passive stressor is relatively easy to employ and has been shown to effectively perturb the cardiovascular system even when used in the same laboratory session as the speech task (e.g., Soye & O'Súilleabháin, 2019). Future studies should consider employing this paradigm; it overcomes limitations with previous passive stressors and is associated with a cardiovascular response pattern consistent with other passive stress tasks.
CHAPTER 5

Individual Differences in Emotion Regulation and Cardiovascular Reactivity to a Passive Stressor

Introduction

Research examining the influence of instructed reappraisal and suppression on physiological responding to stress typically employs passive stress tasks, such as negative imagery or emotion-eliciting film-clips. However, results to date have been inconsistent. While some research reports instructed suppression and instructed reappraisal leads to lower, and greater, SNS activation, respectively (e.g., Gruber et al., 2014; N. A. Roberts et al., 2008), other research fails to find this relationship (e.g., Shiota & Levenson, 2009). Such inconsistencies may be due to the use of tasks that are not socially relevant. Therefore, in Study 3 we tested, and provided evidence for, the construct validity of a more socially relevant passive stress task. Furthermore, no research has yet examined how individual differences in habitual reappraisal use and difficulties in emotion regulation may influence CVR during passive stress. The present study aimed to employ this more novel passive stress task to examine the relationship between individual differences in emotion regulation and stress responsivity within a passive coping context.

Instructed Emotion Regulation and Affect during Passive Tasks

Instructions to reappraise negative images from the IAPS and emotion-eliciting film-clips typically results in lower self-reported unpleasant emotions (Dillon et al.,
Chapter 5, Study 4: Trait ER and CVR to Passive Stress

2007; Gross, 1998b; Gruber et al., 2014; Richards & Gross, 2000; Shiota & Levenson, 2009; Troy et al., 2018; Wolgast et al., 2011). In contrast, instructed suppression results in little-to-no change in reported affect compared to control conditions (Dillon et al., 2007; Dunn et al., 2009; Richards & Gross, 1999, 2000; Shiota & Levenson, 2009). Across a range of emotion-eliciting paradigms it appears reappraisal reduces unpleasant emotions while suppression does little to alter the emotional experience of the task. While the picture regarding instructed emotion regulation use and the psychological experience of passive stress is clear, the physiological consequences of using these strategies is not.

Instructed Emotion Regulation and Physiological Responding during Passive Tasks

In some studies, the hypothesis that reappraisal leads to lower physiological arousal, while suppression leads to greater physiological arousal, is supported. For example, instructions to reappraise emotion-eliciting film clips by adopting an objective perspective led to lower skin conductance responding compared to “just watch” conditions (Gruber et al., 2014; Wolgast et al., 2011). In contrast, instructed suppression has led to greater skin conductance, and greater SBP and DBP reactivity, during emotion-eliciting film-clips (Gross, 1998b; N. A. Roberts et al., 2008) and a negative-emotion slide viewing paradigm (Richards & Gross, 1999). Likewise, during a more novel passive task, where participants were asked to view a video of themselves singing, instructed use of suppression was associated with greater SBP reactivity compared to the “just watch” condition (Harris, 2001).

Other studies have failed to support this hypothesis. No differences in skin conductance were observed between reappraisal and control groups in response to emotion-eliciting film-clips and the IAPS (Gross, 1998b; Kim & Hamann, 2012).
Similarly, suppression instructions prior to a distressing video did not alter HR or skin conductance responses compared to a control group (Dunn et al., 2009). Furthermore, Shiota and Levenson (2009) found no differences in blood pressure or skin conductance responding between groups instructed to reappraise or suppress emotions during sad and disgusting film-clips. Overall, research to date has reported inconsistent findings regarding *instructed* use of emotion regulation and physiological responding to passive stress. This may be due to methodological differences between studies in terms of how physiological responding was assessed, the types of tasks used, and/or the way in which suppression and reappraisal were manipulated.

It appears, in the context of a passive stressor, instructed reappraisal results in regulated emotions, while instructed suppression does not offer relief from the negative emotions elicited. The literature, however, does not reliably suggest that reappraisal predicts lower physiological arousal, and suppression greater physiological arousal. Nor is the relationship between habitual use of these strategies and CVR during passive stress known.

**The current study**

The present study, therefore, will examine associations between emotion regulation style and CVR during passive stress, and address a number of methodological limitations inherent in past research. Cardiovascular responses to the validated passive task reported upon in Chapter 4 will be assessed using a standardised laboratory stress paradigm. This overcomes limitations of previous research that rely on passive tasks that arguably lack construct validity. The inclusion of an acclimatization and baseline period will allow for a more reliable assessment of CVR (described in Chapter 3). The Finometer PRO will be employed; this offers a more sophisticated measurement of
physiological responding than that afforded by skin conductance measurements. Importantly, this study will assess *individual differences* in emotion regulation; a lack of research in this area makes it difficult to conclude how the individual propensity to engage in such strategies may influence health.

It was expected that greater habitual use of suppression would be associated with greater CVR, while habitual use of reappraisal would be associated with lower CVR. Based on previous research (employing active tasks), and the results of Study 2, habitual use of reappraisal may be associated with a more challenge-oriented cardiovascular response. However, the principles of the biopsychosocial model posit challenge/threat responses occur during motivated performance situations, and so, will be absent during a passive stress task. It is therefore unclear if reappraisal will influence physiological patterns of challenge/threat responding. No published research has examined the role of emotion regulation difficulties in CVR to acute stress, but it is expected greater difficulties in emotion regulation will be associated with a more maladaptive cardiovascular response.

**Methodology**

**Design**

The current study employed a $2 \times 1$ within-subjects design. The within-subjects factor was phase with two levels; baseline and task. Emotion regulation scores (reappraisal, suppression, and emotion regulation difficulties) were entered as covariates. The dependent variables were self-reported affect (negative, positive), ratings of stress, and anxiety, and mean cardiovascular responding at each stage of the protocol.
Participants

The current study presents findings from a final sample of 25 participants (17 females, 8 males), aged 18-25 ($M = 20.40$, $SD = 1.66$) reported upon in Study 3. These participants were recruited from the sample reported upon in Study 2 ($N = 52$). All participants were contacted to participate in the second part of the study, but only 26 successfully completed both visits; one participant was excluded due to high resting blood pressure. Again, participants were non-smokers, reported good health, no history of cardiovascular disease, and were not taking medication known to affect blood pressure. There were no differences between participants who completed both visits, and participants who failed to complete the second visit, in terms of emotion regulation, task performance, and CVR to the task, all $p_s > .150$. 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. Participants received no monetary benefit to participate in the follow-up study.

A priori power analyses were conducted using G*Power (Faul et al., 2007) to establish sample size requirements to detect large and medium effects, for the interaction between emotion regulation style and CVR. Using an alpha of .05, and power of .80, in order to detect a large effect ($f = .40$) 16 participants were needed and to detect a medium effect ($f = .25$) 32 participants were needed. As noted, 25 participants completed both visits, meaning the sample had sufficient power to detect large effects.

Materials and Apparatus

The ERQ and the DERS assessed individual differences in emotion regulation at time point one (during the first laboratory visit, prior to the active task). The reappraisal,
suppression, overall DERS, and DERS subscales all demonstrated good-to-excellent reliability, with Cronbach’s αs of .92, .79, .95, and αs > .82, respectively.

All participants completed the PANAS and rated the degree to which they felt stressed or anxious prior to the task and post-task. The PANAS demonstrated good internal reliability at baseline and task, Cronbach’s α was ≥ .72 for each scale.

Participants reported the degree to which they engaged in reappraisal and suppression during the task, as described in Study 2. Spearman-Brown co-efficient was .88 and .76 for the self-reported use of suppression subscale during the passive and active tasks respectively, indicating good reliability. Likewise, the self-reported use of reappraisal subscale demonstrated good reliability in response to the active task, α = .76, but less-than-ideal reliability post-passive task, α = .48.

Beat-to-beat blood pressure and hemodynamic parameters were measured non-invasively using a Finometer Pro, as described in Study 2.

**Laboratory stressor.** The current study reports on data from Study 3. Participants watched a video-clip of themselves completing a speech task from a previous laboratory visit. The stress-task for the current study required participants to simply sit and watch this video-clip. The validity of this novel stressor is described in Study 3.

**Procedure**

The procedure described in Study 3 was employed. In summary, participants completed two laboratory visits, a year apart. During each visit, participants were greeted by the experimenter and provided informed consent. Participants were seated in front of a computer screen, separated from the experimenter by a partition. Participants were connected to the Finometer PRO and completed a 20-minute acclimatization period, an
official 10-minute resting baseline, and a 5-minute stress task. At the first visit participants filled out the emotion regulation scales (ERQ and DERS), and then completed the speech task while being video recorded. Participants were told this recording would later be analysed for body language. In fact, participants were later contacted and asked to view this video-clip during a second laboratory visit. Participants filled out psychometric questionnaires during the acclimatization periods and reported indices of affect pre- and post-task. Cardiovascular measures were recorded continuously using the Finometer PRO. Figure 5.1 presents each phase of the study.

Results

Overview of Analyses

The mean values for each cardiovascular parameter (SBP, DBP, HR, CO, and TPR) during the baseline and passive task were calculated and are presented in Chapter 4.

A series of custom 2 × 1 repeated measures ANCOVAs were conducted to examine if emotion regulation style influenced stress responsivity to the passive stress task. The within-subjects factor was phase (baseline, task). Continuous scores on emotion regulation (reappraisal, suppression, and emotion regulation difficulties) were entered as covariates in separate models.

Effect sizes are presented as partial $\eta^2$ for ANOVA analyses with values of .04, .25, and .40 taken to demonstrate small, medium, and large effects, respectively (J. Cohen, 1992). Partial $\eta^2$ has been recommended for ANOVA designs (Tabachnick & Fidell, 1989). Effect sizes for correlations are presented as $r$, with values of .10, .30, and .50 taken to be indicative of small, medium, and large effect sizes respectively (J.
Cohen, 1988, 1992). Effect sizes for repeated measures and one-sample \( t \)-tests were calculated using G*Power, and are presented as \( d \), with values of .20, .50, and .80 indicative of small, medium, and large effect sizes.

**Confirmation of Reactivity**

Table 5.1 displays means, standard deviations, and the range of scores on each emotion regulation subscale. As outlined in Chapter 4, the passive task was successful in eliciting SBP, DBP, and TPR reactivity, but did not elicit CO or HR reactivity. It also appeared that the task did not elicit a negative emotional experience, with stress, anxiety, NA, and PA similar between baseline and post-task periods. Full analyses and results are outlined in Chapter 4.

**Table 5.1.**

Means, standard deviations, and range of scores for each emotion regulation scale

<table>
<thead>
<tr>
<th></th>
<th>( M )</th>
<th>( SD )</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reappraisal</td>
<td>30.08</td>
<td>6.84</td>
<td>14-42</td>
</tr>
<tr>
<td>Suppression</td>
<td>12.08</td>
<td>4.33</td>
<td>5-20</td>
</tr>
<tr>
<td>Total DERS</td>
<td>86.44</td>
<td>24.49</td>
<td>42-142</td>
</tr>
<tr>
<td>Impulse Control</td>
<td>12.12</td>
<td>4.88</td>
<td>6-21</td>
</tr>
<tr>
<td>Nonacceptance</td>
<td>15.12</td>
<td>6.00</td>
<td>6-25</td>
</tr>
<tr>
<td>Goal-directed behaviour</td>
<td>16.08</td>
<td>4.89</td>
<td>5-23</td>
</tr>
<tr>
<td>Clarity</td>
<td>11.12</td>
<td>3.75</td>
<td>7-23</td>
</tr>
<tr>
<td>Awareness</td>
<td>15.04</td>
<td>4.94</td>
<td>7-29</td>
</tr>
<tr>
<td>Strategies</td>
<td>17.40</td>
<td>6.69</td>
<td>8-32</td>
</tr>
</tbody>
</table>
Figure 5.1. Visual schema of the laboratory procedure employed.
Chapter 5, Study 4: Trait ER and CVR to Passive Stress

**Trait Emotion Regulation**

**Physiological outcomes.** There was no significant Phase × Reappraisal interaction effect on CVR: SBP, $F(1, 23) = 0.13, p = .719$, partial $\eta^2 = .006$; DBP, $F(1, 23) = 0.27, p = .611$, partial $\eta^2 = .011$; HR, $F(1, 23) = 1.72, p = .202$, partial $\eta^2 = .070$; CO, $F(1, 23) = 0.91, p = .350$, partial $\eta^2 = .038$; TPR, $F(1, 23) = 0.11, p = .741$, partial $\eta^2 = .005$. Similarly, there were no significant Phase × Suppression interaction effects: SBP, $F(1, 23) = 0.22, p = .647$, partial $\eta^2 = .009$; DBP, $F(1, 23) = 0.16, p = .693$, partial $\eta^2 = .007$; HR, $F(1, 23) = 0.02, p = .881$, partial $\eta^2 = .001$; CO, $F(1, 23) = 0.46, p = .506$, partial $\eta^2 = .020$; TPR, $F(1, 23) = 0.31, p = .586$, partial $\eta^2 = .013$.

Overall, it appeared that individual differences in trait emotion regulation did not influence CVR to the passive task.

**Psychological outcomes.** There were no significant associations between habitual emotion regulation (reappraisal and suppression) and self-reported PA, NA, stress, and anxiety at baseline, all $p$s > .120. Trait emotion regulation did not influence self-reported affect in the absence of acute stress.

A series of custom-built ANCOVAs were conducted to examine if trait emotion regulation influenced affect over the phases of the laboratory procedure. There were no significant Phase × Reappraisal interaction effects on self-reported affect: stress, $F(1, 23) = 2.94, p = .100$, partial $\eta^2 = .113$; anxiety, $F(1, 23) = 0.34, p = .566$, partial $\eta^2 = .015$; NA, $F(1, 22) = 0.79, p = .382$, partial $\eta^2 = .035$; PA, $F(1, 22) = 0.21, p = .652$, partial $\eta^2 = .009$. Nor were there any significant Phase × Suppression interaction effects on: stress, $F(1, 23) = 0.18, p = .677$, partial $\eta^2 = .008$; anxiety, $F(1, 23) = 1.07, p = .313$, partial $\eta^2 = .044$; NA, $F(1, 22) = 3.70, p = .068$, partial $\eta^2 = .144$; PA, $F(1, 22) = 1.43, p = .245$. 

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partial $\eta^2 = .061$. The results suggest that habitual use of reappraisal and suppression had no influence on the psychological experience of the task.

### Difficulties in Emotion Regulation

**Physiological outcomes.** There was no significant Phase × Difficulties interaction effect on any of the cardiovascular parameters: SBP, $F(1, 23) = 0.49, p = .490$, partial $\eta^2 = .021$; DBP, $F(1, 23) = 0.29, p = .595$, partial $\eta^2 = .012$; HR, $F(1, 23) = 2.39, p = .136$, partial $\eta^2 = .094$; CO, $F(1, 23) = 0.02, p = .897$, partial $\eta^2 = .001$; TPR, $F(1, 23) = 0.44, p = .515$, partial $\eta^2 = .019$.

Repeating these analyses, using the DERS subscales as covariates, there was a Phase × Strategies interaction effect for HR, $F(1, 23) = 5.08, p = .034$, partial $\eta^2 = .181$. Greater perceived difficulties in accessing effective emotion regulation strategies were associated with greater HR reactivity in response to the passive task, $r = +.425, p = .034$. No other Phase × DERS subscale interaction effects on CVR were observed, all $ps > .133$.

**Psychological outcomes.** At baseline, greater emotion regulation difficulties were associated with greater self-reported NA ($r = +.616, p = .001$), stress ($r = +.602, p = .001$), and anxiety ($r = +.427, p = .033$), but unrelated to self-reported PA ($r = -.088, p = .682$). Individuals with greater emotion regulation difficulties perceived more NA, stress, and anxiety in the absence of acute stress.

ANCOVA analyses found a significant Phase × Difficulties interaction effect on ratings of stress, $F(1, 23) = 10.99, p = .003$, partial $\eta^2 = .323$. Individuals with greater emotion regulation difficulties reported more stress at baseline, but in the presence of stress, there was no difference in perceived stress between those high and low in difficulties. There were no other significant Phase × Difficulties interaction effects on
Chapter 5, Study 4: Trait ER and CVR to Passive Stress

self-reported affect: anxiety, $F(1, 23) = 2.21, p = .151$, partial $\eta^2 = .088$; PA, $F(1, 22) = 0.23, p = .637$, partial $\eta^2 = .010$; NA, $F(1, 22) = 3.16, p = .089$, partial $\eta^2 = .125$.

**Hemodynamic Profile**

A series of independent samples $t$-tests were conducted to examine between-group differences in HP and CD (median split applied to emotion regulation scores). There was no difference in HP or CD between individuals scoring high and low in; emotion regulation difficulties (HP; $p = .346$, CD; $p = .609$), reappraisal (HP; $p = .858$, CD; $p = .519$), or suppression (HP; $p = .595$, CD; $p = .856$).

**Challenge-threat Index**

Challenge-threat index and emotion regulation style. There were no differences in cardiovascular indices of challenge/threat responding between individuals scoring high and low in reappraisal ($p = .773$), suppression ($p = .597$), and emotion regulation difficulties ($p = .454$).

**Spontaneous Reappraisal and Suppression**

A series of independent sample $t$-tests found that trait emotion regulation did not influence spontaneous use of reappraisal and suppression during the task. Similarly, there were no Phase × Spontaneous emotion regulation interaction effects on CVR, all $p$s > .112, except for a significant Phase × Spontaneous suppression interaction effect on HR, $F(1, 23) = 5.59, p = .027$, partial $\eta^2 = .196$, where greater self-reported use of suppression during the passive task was associated with lower HR reactivity, $r = -.442, p = .027$.

Repeated samples $t$-tests found that participants reported engaging in reappraisal ($M = 7.96, SD = 2.30$) and suppression ($M = 6.68, SD = 2.30$) to a greater extent during the active task than during the passive task (reappraisal, $M = 6.88, SD = 2.32$;
suppression, $M = 5.28, SD = 2.42$): spontaneous reappraisal, $t(24) = 2.70, p = .012, d = 0.47, 95\% CI [0.26, 1.90]$; spontaneous suppression; $t(24) = 2.49, p = .020, d = 0.59, [0.24, 2.56]$.

**Discussion**

It appears that individual differences in emotion regulation style do not influence cardiovascular responding during passive stress. Likewise, emotion regulation style had no effect on the emotional experience of the task. The findings are contrary to the assumptions of the process model of emotion regulation (Gross, 1998a, 1998b) with neither trait reappraisal nor trait suppression influencing CVR during the task. Results from research instructing the use of reappraisal and suppression, that have employed typical passive stress tasks, have been somewhat inconsistent. While the observed results support research reporting no relationship between instructed emotion regulation and physiological responding (e.g., Kim & Hamann, 2012; Shiota & Levenson, 2009), the results conflict with a large literature supporting the assumptions of the process model (e.g., Gruber et al., 2014; Harris, 2001; Hofmann et al., 2009; Wöl gast et al., 2011). We extend this research and provide evidence that individual differences in the habitual use of these strategies has little impact on CVR during passive stress.

Similarly, all participants reported similar levels of stress, anxiety, PA, and NA during the task, regardless of individual differences in emotion regulation. The observed results provide support for the hypothesis that suppression has little effect on the emotional experience of the stress task, corroborating results from research instructing the use of suppression (Dillon et al., 2007; Dunn et al., 2009; Richards & Gross, 1999). However, instructed reappraisal is typically associated with lower negative affect (e.g.,
Richards & Gross, 2000; Shiota & Levenson, 2009; Wolgast et al., 2011); it appears habitual use of reappraisal has little influence on emotional responding during stress. It must be noted that research manipulating reappraisal explicitly instructs participants to decrease negative affect. This may have resulted in self-report bias; examining individual differences in strategy use overcomes this limitation.

While difficulties in emotion regulation did not influence self-reported affect during the task in the absence of acute stress (during the baseline) individuals with greater difficulties in emotion regulation perceived more stress, consistent with the results of Study 1 and Study 2. It may be the case that individuals with greater difficulties in emotion regulation cannot accurately report their emotions when under distress. Perhaps these individuals lack an awareness of their emotional responses when upset (lack of emotional awareness or clarity), or perhaps report less negative emotion despite actually experiencing negative emotions (lack of emotional acceptance).

While it appears that individual differences in emotion regulation style have little influence on CVR during passive stress, the emotional nature of the task must be considered. Previous research suggests that emotion regulation (specifically use of suppression) affects physiological responding only when the stressor is negative in nature (Gross & Levenson, 1997). Similarly, Shiota and Levenson (2009) found no differences in physiological arousal between suppression, reappraisal, and control conditions, but noted that the film-clip used in the suppression condition was perceived as less emotionally arousing. In the present study, a neutral video-clip was employed, which may have influenced our results. Likewise, in Study 2, trait reappraisal and difficulties in emotion regulation only influenced patterns of cardiovascular responding during the negative-emotion task. Further research is needed to elucidate the relationship between
emotion regulation style and cardiovascular responding, and to test the boundary conditions for emotion regulation to have an influence on CVR.

Furthermore, the passive nature of the task must be taken into consideration. As outlined in Chapter 3, passive and active tasks elicit distinct patterns of cardiovascular responding and elicit different coping contexts in terms of participant control, motivation, and the socio-evaluative aspects of the task. Perhaps passive coping contexts do not elicit as strong a need to regulate emotional responses. Indeed, participants reported engaging in reappraisal and suppression to a greater extent during the active task relative to the passive task. In particular, due to the passive nature of the task, patterns of cardiovascular challenge and threat should not be observable, that is, changes in CO and TPR responding. The biopsychosocial model posits that cardiovascular challenge-threat responses are elicited by motivated performance situations; therefore, it is not surprising that emotion regulation style had little influence on hemodynamic response patterns. Further research is needed using a variety of active and passive tasks to establish the conditions necessary for emotion regulation to be (a) elicited, and (b) influence physiological responding.

The current results offer insights in the role of emotion regulation in stress responsivity during passive coping; however, considering the small sample size ($n = 25$), the results must be interpreted with caution. Attrition is a common problem for research employing a follow-up phase; although all participants were contacted, less than 50% of the original sample completed the both laboratory visits. Due to the smaller sample the study may have lacked sufficient power to detect significant effects. The current study needs to be replicated using a larger, and more diverse, sample size in order to improve the reliability and generalisability of the observed results.
Despite these limitations, the current study is strengthened by its use of an
established laboratory stress paradigm. The inclusion of an official acclimatization period
and Vanilla baseline ensured accurate measurement of blood pressure at rest; a clear
limitation of past research in this area. Furthermore, the current study employed a more
natural coping context than previous passive tasks. This paradigm may be useful in
examining the outcomes of trait emotion, but with a larger sample and potentially a task
with negative-emotion content.

The current study builds on previous research focusing on instructed emotion
regulation and was the first to examine the role of trait reappraisal, and emotion
regulation difficulties, on CVR during a passive stress task; suggesting individual
differences in emotion regulation style do not influence CVR during passive stress. The
observed results somewhat conflict with the hypothesis that the use of reappraisal buffers
the negative consequences of stress, while suppression exacerbates cardiovascular
responses to stress (Gross, 1998a; Mauss & Gross, 2004). However, both the non-
emotional and passive nature of the stress task employed may have influenced our
results. Future research needs to address methodological limitations in this area and
examine the conditions under which trait emotion regulation influences stress
responsivity.
CHAPTER 6

Experimentally Manipulating Emotion Regulation Style:
Reappraisal and Cardiovascular Habituation to Active Stress

Introduction

The previous chapters focused on establishing if trait indices of emotion regulation are related to cardiovascular responses during stress and found support for this in the context of an active stressor. If habitual use of a hypothesized healthful strategy, such as reappraisal, can be instructed or taught, this may be beneficial for health. Instructions to reappraise stress arousal as a resource (something that is adaptive and aids performance) is hypothesized to influence CVR as it switches appraisals of the upcoming task from a threat to a challenge response (Jamieson, Hangen, Lee, & Yeager, 2018). In contrast, perceiving physiological arousal (e.g., increased heart rate, sweaty palms) as a sign of insufficient personal resources to cope with the stressor, may result in a threat response. Indeed, research has shown that instructed reappraisal results in greater perceived resources to cope with the subsequent task (Beltzer, Nock, Peters, & Jamieson, 2014; Jamieson, Mendes, Blackstock, & Schmader, 2010; Jamieson et al., 2012, 2013; Jamieson, Peters, Greenwood, & Altose, 2016). In line with the biopsychosocial model, greater resource appraisals should result in a challenge-oriented cardiovascular response (e.g., Tomaka et al., 1997), and results from studies instructing reappraisal support this hypothesis.
Individually instructed to use reappraisal, when completing a speech task (including the TSST), exhibited a challenge-orientated cardiovascular response (indexed by greater CO and lower TPR reactivity) compared to “ignore” stress groups and/or a control group (Jamieson et al., 2012, 2013). Likewise, reappraisal instructions led to a greater challenge response (indexed by the challenge-threat index) during a mental arithmetic task compared to individuals instructed to view stress as debilitating (Hangen et al., 2019); however, compared to an uninstructed group, reappraisal did not lead to observable differences in cardiovascular responding. Use of reappraisal also has physiological benefits for individuals interacting with the reappraiser. During a teamwork task, instructions to reappraise led to a challenge-oriented physiological response (greater CO/lower TPR) for both the team-mate receiving the instructions, and their partner (unaware of the manipulation), compared to suppression or no instructions (Oveis, Gu, Ocampo, Hangen, & Jamieson, 2018). The reported findings demonstrate instructed reappraisal results in an adaptive cardiovascular response profile, at least in the short-term.

**Instructed Reappraisal and Habituation to Recurrent Stress**

However, the focus of this research is on CVR reactivity to one stress exposure – a novel stress task. In recent years, the CVR paradigm has been extended to include a second exposure to the same task. This allows for CVR to recurrent stress to be assessed. Habituation, a hypothesized healthful cardiovascular response, is indexed by a notable cardiovascular response to the first stress task, followed by a lower cardiovascular response to the second stressor (Hughes et al., 2018). A failure to habituate, or indeed exhibit heightened CVR to the second stress task (sensitization), is thought to reflect an underlying lack of ability to adapt to, and cope, with stress (Howard & Hughes, 2013;
Hughes et al., 2018), which overtime may have consequences for physical health (e.g., Kelsey, 1993; McEwen & Stellar, 1993). As described in Chapter 1, evidence has accumulated to demonstrate how individual differences in certain personality traits are associated with patterns of habituation (e.g., Lu & Wang, 2017; Lu, Wang, & Hughes, 2016; Lu, Wang, & You, 2016). Similarly, manipulating task feedback prior to the second stress exposure has been reported to alter habituation (Brown & Creaven, 2017); positive feedback on task performance resulted in habituation to the second task, whereas negative feedback resulted in sensitization. In a similar vein of reasoning, instructed reappraisal should alter habituation to recurrent stress; however, this has not yet been examined.

Further Limitations of Past Research

A further criticism of past research is that although blood pressure is assessed, the effects of instructed reappraisal on blood pressure responding are not reported (e.g., Hangen et al., 2019; Jamieson et al., 2012, 2013). While acknowledging the role of CO and TPR in elucidating whether a challenge or threat response is elicited, there is scant research on the long-term impact of challenge and threat responses on physical health. Conversely, exaggerated SBP and DBP responding to laboratory stressors has demonstrable predictive ability in determining future cardiovascular disease risk (e.g., Carroll et al., 1995; Carroll et al., 2001; Treiber et al., 2003). It is therefore pertinent to determine if instructed reappraisal alters blood pressure responses to stress.

Similarly, the way in which emotion regulation research calculates “cardiovascular reactivity” contrasts with the approach employed by research in the stress literature. Typically, studies examining emotion regulation calculate reactivity by subtracting the final minute of baseline, “the most relaxed portion”, from the first minute
of the task, “the most reactive portion” (e.g., Hangen et al., 2019; Jamieson et al., 2012, 2013; Oveis et al., 2018). Conversely, research with cardiovascular reactivity as the focus recommends cardiovascular measurements taken throughout each task period (e.g., baseline, task) are averaged, and reactivity is calculated using the average of each task period. The latter computation has been demonstrated to be a more reliable measurement of reactivity (e.g., Kamarck & Lovallo, 2003), and is used consistently in CVR research (e.g., Carroll, Phillips, Der, Hunt, & Benzeval, 2011; Gallagher, Howard, & Heffernan, 2015; Ginty, Brindle, & Carroll, 2015; Stewart, Janicki, & Kamarck, 2006).

Furthermore, past research does not consider how individual differences in the habitual tendency to engage in reappraisal may influence the effects of the manipulation (e.g., Hofmann et al., 2009). If the ability to engage in reappraisal is universal, there should be no differences in physiological responding between individuals who habitually engage in reappraisal, and those who do not, when instructed to reappraise stress. However, if the ability to use reappraisal varies between individuals, people who habitually use reappraisal may find it easier to implement in the laboratory (Wolgast et al., 2011). Few studies have considered this relationship, and results have been mixed. Some research has shown that individuals scoring high in reappraisal, instructed to reappraise, demonstrated lower cortisol responses during stress (Mauersberger, Hoppe, Brockmann, & Hess, 2018), while others have found no interaction between trait and instructed reappraisal on skin conductance (Wolgast et al., 2011). This relationship has not been examined with blood pressure parameters as the outcome.

**Aims of the Present Study**

The current study aims to address limitations with past research by employing a standardized laboratory protocol (including an acclimatization and baseline period) and
calculating reactivity using recommended methods. It will examine the influence of instructed reappraisal on adaption to recurrent stress; reappraisal instructions will be manipulated prior to the second stress exposure, consistent with the positive/negative feedback manipulation used in previous research (Brown & Creaven, 2017). This will substantiate research suggesting reappraisal buffers physiological stress responses. This study will extend past previous research by reporting blood pressure responding, and will examine patterns of CO and TPR responding using the HP-CD computational model (Gregg et al., 2002). Furthermore, the potential interaction between the habitual tendency to engage in reappraisal and the ability to implement reappraisal instructions will be explored. It is hypothesized that instructed reappraisal will result in habituation to the second stress exposure and greater self-reported PA, less NA, less threat/demand, and more resource appraisals. We have no a priori hypothesis regarding the interaction between trait and instructed reappraisal.

Methodology

Design

The current study employed $2 \times 2 \times 1$ mixed design. The within-subjects factor was task (reactivity to Task 1, reactivity to Task 2). The between-subjects factor was experimental group (control, reappraisal). Individual differences in emotion regulation were entered as a covariate (trait reappraisal, trait suppression, emotion regulation difficulties). The dependent variables were SBP, DBP, HR, CO, TPR, and self-reported affect.
Participants

A total of 139 non-smokers participated in the study. Participants reported good health, no history of cardiovascular disease, and were not taking medication known to affect blood pressure. Thirty-three participants were excluded from this sample; cardiovascular assessment was not available for 17 participants, eight participants reported English as their second language, one participant was over the threshold of 25-years of age, and five participants had resting blood pressure classified as potentially hypertensive (SBP/DBP > 140/90 mmHg). A final sample of 106 healthy young adults, testing as normotensive, were included in the analyses (77 women and 29 men) aged 18-25 \( (M = 19.63, \text{SD} = 1.74) \). Participants were randomly assigned to either the control \((n = 54)\) or reappraisal experimental group \((n = 52)\).

Previous research has reported medium to large effect sizes for the influence of instructed reappraisal on cardiovascular parameters (e.g., Jamieson et al., 2010; Jamieson et al., 2012, 2013) and performance (e.g., Jamieson et al., 2010; Jamieson et al., 2016). A priori power analyses were conducted using G*Power (Faul et al., 2007), using an alpha of .05, and power of .80. In order to acquire sufficient power a minimum of 16 participants per condition were needed to detect large effects \((f = .40)\) and a minimum of 34 participants per condition were needed to detect medium effects \((f = .25)\); resulting in a minimum total sample size of 32 and 68 to detect a large and medium effect, respectively.

Ethical approval was obtained from the institutional research ethics committee. Participation was voluntary, participants signed an informed consent form and could withdraw from the study at any time. Participants received course credit for their participation.
Chapter 6, Study 5: Instructed Reappraisal and Habituation

Materials and Apparatus

**Individual differences in emotion regulation.** The ERQ (Gross & John, 2003) and the DERS (Gratz & Roemer, 2004) assessed individual differences in emotion regulation style, as described in Chapter 2. In the present study all scales demonstrated excellent reliability: reappraisal, Cronbach’s $\alpha = .80$; suppression, Cronbach’s $\alpha = .75$; DERS, Cronbach’s $\alpha = .93$.

**Self-reported use of reappraisal.** A one-item measure of spontaneous reappraisal use was employed, adapted from Egloff et al. (2006) and Ehring et al. (2010) to reflect reappraisal use specific to feelings of stress; “I thought about the task in a way that helped me experience less stress”.

**Affect.** Similar to previous chapters the PANAS (Watson et al., 1988) was included to assess state PA and NA, during pre- and post-task periods, and demonstrated good internal reliability, Cronbach’s $\alpha$ was $\geq .80$ for each scale. Furthermore, participants were asked to indicate on ten-point Likert scale from 1 (not at all) to 10 (extremely), pre- and post-task, to rate the extent to which they felt stressed or anxious at that moment (state stress/state anxiety).

**Pre-task stress appraisals.** Appraisals of each task, in terms of expected demands and perceived personal resources to cope with the task, were assessed on a six-point Likert scale from 1 (not at all/strongly disagree) to 6 (very/agree strongly) prior to each task. Three items assessed perceived demands (“how stressful do expect to find the task”, “how difficult do you expect to find the task”, and “I feel threatened by the task”). Four items assessed perceived resources (“I am excited about this task”, “I have control over the outcomes of the task”, “I have the ability to do well on the task”, and “I think this task will go well”). Items were adapted from resource/demand appraisal.
questionnaires (e.g., Mendes, Blascovich, Major, & Seery, 2001; Mendes, Gray, Mendoza-Denton, Major, & Epel, 2007; Tomaka et al., 1993). Both questionnaires demonstrated good to excellent reliability at pre-task 1 (demand; α = .73, resource; α = .65) and at pre-task 2 (demand; α = .83, resource; α = .77).

**Post-task appraisals.** Task appraisals were also assessed immediately post-task. Participants rated on a six-point Likert scale from 1 (*not at all*) to 6 (*very*) the degree to which stress and anxiety were experienced *during* the task, and task difficulty (e.g., “how stressful did you find the task”). Likewise, a single item was employed to assess challenge appraisals of the task (“I viewed the task as a challenge”). Participants rated their agreement with this item on a six-point Likert scale from 1 (*strongly disagree*) to 6 (*strongly agree*).

**Stressor task.** The socio-evaluative speech task described in Study 2 was employed. Task performance data was missing for 23 participants as the computer storing this information malfunctioned. Results on performance are based on the data available for 83 participants (40 in the control condition, 43 in the reappraisal condition).

**Cardiovascular Assessment.** Cardiovascular parameters were measured non-invasively using the Finometer PRO (Finapres Medical Systems BV, BT Arnhem, The Netherlands), as described in previous chapters.

**Procedure**

All participants visited the laboratory at an assigned morning time slot between 8:30am and 2:00pm to minimize the impact of diurnal changes to blood pressure. In addition, participants were asked to refrain from drinking caffeinated products for six hours and alcohol for 12 hours prior to participation. Participants were randomly
assigned to either the reappraisal or control condition prior to data collection commencement using an online randomiser tool (https://stattrek.com/).

The laboratory procedure employed in Study 2 was used; however, prior to the second stress exposure participants received either reappraisal or control instructions. Briefly, participants completed a 20-minute acclimatization period. Following this, resting cardiovascular measures were taken during an official 10-minute baseline period. Reading material was provided to lower the risk of potential boredom and/or rumination arousal, employing the Vanilla resting baseline as recommended by Jennings et al. (1992). Participants then completed the five-minute speech task employed in Study 2. After a ten-minute inter-task rest period, participants received a block of instructions depending on their assigned experimental condition. Participants in the reappraisal condition heard instructions describing signs of arousal (e.g., sweaty palms, increased heart rate) as normal - that stress is not harmful and helps people perform better at tasks. To control for time and experimental instructions, individuals in the control condition also heard a set of instructions. These participants also heard that that any signs of arousal are normal, and that blood pressure was going to be analysed during the task. Full instructions are reported in Appendix D. Reappraisal instructions were adapted from past research (Jamieson et al., 2010; Jamieson et al., 2012, 2013; Jamieson et al., 2016). Control instructions were adapted from instructions employed by Jamieson et al. (2010).

Participants completed the speech task for a second time. Prior to each task, and after each task, participants filled out questionnaires assessing task appraisals and affect. Following completion of the laboratory session participants were debriefed and thanked for their participation. Figure 6.1 displays the questionnaires completed during each phase of the laboratory experiment.
Figure 6.1. Description of self-report measures completed at each time point. State affect = self-reported stress and anxiety felt at that moment; Task appraisals = task difficulty/stressfulness/anxiety experienced during the task, and challenge appraisals; PANAS-NA = the negative affect subscale.
Chapter 6, Study 5: Instructed Reappraisal and Habituation

Results

Overview of Analyses

The mean values for each cardiovascular parameter during each experimental phase were calculated and are presented in Table 6.1. Internal consistency for each cardiovascular variable was excellent with Cronbach’s αs > .95. Change scores were computed for SBP, DBP, HR, CO, and TPR reactivity to each task by subtracting mean responses during the task period from baseline; and are presented in Table 6.2. Change scores were also computed for self-reported stress, anxiety, PA, and NA, by subtracting self-reported affect immediately preceding the task from post-task ratings of affect. Table 6.3 displays means, standard deviations, and the range of scores on each emotion regulation scale.

A series of 2 × 2 ANOVAs were conducted to examine the influence of reappraisal instructions on CVR and self-report measures. The between-subjects factor was group (reappraisal, control). The within-subjects factor was task (task 1, task 2). The dependent variables were cardiovascular reactivity, self-reported affect, and task appraisals. Using the same variables, a series of 2 × 2 × 1 custom-built ANCOVAs were conducted to examine the potential influence of emotion regulation style. Emotion regulation scores (trait reappraisal, trait suppression, emotion regulation difficulties) were entered as covariates.

Effect sizes are presented as partial η² for ANOVA analyses with values of .04, .25, and .64 taken to demonstrate small, medium, and large effects, respectively.

PA was not assessed immediately prior to Task 2, therefore for changes in PA self-reported PA at baseline was subtracted from PA post-task 2.
Confirmation of Random Assignment

A series of independent samples $t$-tests were conducted to examine random assignment to experimental group. There were no differences in cardiovascular parameters at rest or CVR to Task 1 (uninstructed task) between the reappraisal and control group. Likewise, there were no significant differences between the two groups in terms of; individual differences in emotion regulation, age, Task 1 performance, spontaneous use of reappraisal during Task 1, affect, or Task 1 appraisals (pre-task and post-task), all $ps > .070$. The two groups did not significantly differ on gender composition, $\chi^2 = .114, p = .736$. The results suggest that random assignment to group was successful.

Confirmation of CVR

There was a main effect of phase on each cardiovascular parameter: SBP, $F(1.61, 165.08) = 243.78, p < .001$, partial $\eta^2 = .703$; DBP, $F(1.70, 175.07) = 314.35, p < .001$, partial $\eta^2 = .753$; HR, $F(1.83, 187.94) = 61.12, p < .001$, partial $\eta^2 = .372$; CO, $F(1.72, 176.96) = 42.80, p < .001$, partial $\eta^2 = .294$; TPR, $F(1.41, 144.83) = 27.94, p < .001$, partial $\eta^2 = .213$. Pairwise comparisons confirmed that SBP ($ps < .001$), DBP ($ps < .001$), HR ($ps < .001$), CO (task 1; $p < .001$, task 2; $p = .005$), and TPR ($ps < .001$) increased
from baseline to task for both stress exposures; both tasks successfully elicited a
cardiovascular stress response.

SBP \( (p = .005) \), HR \( (p = .024) \), and CO \( (p < .001) \) responding were significantly
lower during Task 2 compared to Task 1, indicative of cardiovascular habituation. There
was no difference in DBP responding between Task 1 and Task 2 \( (p = 1.00) \). Task 2 elicited greater TPR responding than Task 1 \( (p = .001) \). Table 6.1 displays means and
standard deviations for each phase.

**Effects of Emotion Regulation on CVR**

There was no significant Phase \( \times \) Group interaction effect on any cardiovascular
parameter: SBP, \( F(1, 102) = 1.23, p = .271 \), partial \( \eta^2 = .012 \); DBP, \( F(1, 102) = 0.84, p \)
= .363, partial \( \eta^2 = .008 \); HR, \( F(1, 102) = 0.60, p = .441 \), partial \( \eta^2 = .006 \); CO, \( F(1, 102) \)
= 0.33, \( p = .569 \), partial \( \eta^2 = .003 \); TPR, \( F(1, 102) = 1.09, p = .300 \), partial \( \eta^2 = .011 \).
Reappraisal instructions did not influence cardiovascular habituation.

There were no significant Phase \( \times \) Group \( \times \) Trait reappraisal interaction effects
on: SBP, \( F(1, 100) = 0.92, p = .339 \), partial \( \eta^2 = .009 \); DBP, \( F(1, 100) = 0.45 p = .502, \)
partial \( \eta^2 = .005 \); HR, \( F(1, 100) = 0.71, p = .401 \), partial \( \eta^2 = .007 \); CO, \( F(1, 100) = 0.01, \)
\( p = .988 \), partial \( \eta^2 = .001 \); TPR, \( F(1, 100) = 0.10, p = .751 \), partial \( \eta^2 = .001 \).

Likewise, there were no significant Phase \( \times \) Group \( \times \) Trait suppression interaction
effects on: SBP, \( F(1, 100) = 3.32, p = .072 \), partial \( \eta^2 = .032 \); DBP, \( F(1, 100) = 0.91, p \)
= .342, partial \( \eta^2 = .009 \); HR, \( F(1, 100) = 3.41, p = .068 \), partial \( \eta^2 = .030 \); CO, \( F(1, 100) \)
= 0.10, \( p = .757 \), partial \( \eta^2 = .001 \); TPR, \( F(1, 100) = 0.11, p = .774 \), partial \( \eta^2 = .008 \).

Finally, there were no significant Phase \( \times \) Group \( \times \) DERS interaction effects: SBP, 
\( F(1, 100) = 0.02, p = .889 \), partial \( \eta^2 = .001 \); DBP, \( F(1, 100) = 0.02, p = .889 \), partial \( \eta^2 \)
= .001; HR, \( F(1, 100) = 0.49, p = .486 \), partial \( \eta^2 = .005 \); CO, \( F(1, 100) = 1.41, p = .238, \)
Chapter 6, Study 5: Instructed Reappraisal and Habituation

Partial $\eta^2 = .014$; TPR, $F(1, 100) = 0.37, p = .545$, partial $\eta^2 = .004$. These results indicate that there was no significant interaction between experimental group and habitual indices of emotion regulation on cardiovascular habituation.

Hemodynamic Profile

Although reappraisal instructions did not influence SBP, DBP, HR, CO, or TPR responding, we examined if the manipulation influenced the underlying hemodynamic profile of responding.

Examination of the underlying hemodynamic profile found that overall, participants in both groups, demonstrated a mixed HP in response to Task 1: control group, $t(53) = 1.03, p = .306, d = 0.14, 95\% \text{ CI} [-0.01, 0.03]$; reappraisal group, $t(51) = 1.57, p = .122, d = 0.22, [-0.01, 0.05]$, and a vascular response profile during the second stress exposure: control group, $t(51) = 4.86, p < .001, d = 0.67, [0.04, 0.11]$; reappraisal group, $t(51) = 6.47, p < .001, d = 0.90, [0.06, 0.11]$. Both tasks elicited significant CD changes for participants in both groups (all $p$s < .001). While blood pressure increased in response to both tasks, the second task was marked by blood pressure increases driven by more vascular mechanisms. Figure 6.2 displays CO and TPR responding across the three phases for individuals in each experimental group.

Effects of emotion regulation on hemodynamic profile. There was no significant Phase × Group interaction effect on HP scores, $F(1, 102) = 0.01, p = .998$, partial $\eta^2 = .001$, or on CD scores, $F(1, 102) = 0.76, p = .387$, partial $\eta^2 = .007$. There was also no significant Phase × Group × Emotion regulation style interaction effect on HP scores: reappraisal, $F(1, 100) = 0.01, p = .978$, partial $\eta^2 = .001$; suppression, $F(1, 100) = 0.05, p = .817$, partial $\eta^2 = .001$; DERS; $F(1, 100) = 0.11, p = .740$, partial $\eta^2 = .001$. 

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There was no significant Phase × Group × Trait Reappraisal interaction effect on CD scores, $F(1, 100) = 1.96, p = .164$, partial $\eta^2 = .019$, or Phase × Group × DERS interaction effect, $F(1, 100) = 0.12, p = .734$, partial $\eta^2 = .001$.

While there was a significant Phase × Group × Trait Suppression interaction effect on CD values, $F(1, 100) = 4.37, p = .039$, partial $\eta^2 = .042$, post-hoc tests found no significant associations between trait suppression and CD values, all $ps > .167$. There was no effect of the manipulation on hemodynamic responding to the tasks, nor did habitual emotion regulation style influence hemodynamic responding.

**Comparison of HP Scores and Challenge-threat Index.**

As in previous chapters, we examined if indices of challenge/threat as computed using the HP-CD computational model were associated with indices of challenge/threat as computed by the challenge-threat index. Chi-squared tests of association confirmed that there was significant overlap in classifications for both tasks: Task 1, $\chi^2 = 74.47, p < .001, \omega = .838$; Task 2, $\chi^2 = 15.48, p < .001, \omega = .386$.

**Challenge-threat Index.**

There was no significant Phase × Group interaction effect on challenge-threat scores, $F(1, 102) = 0.50, p = .481$, partial $\eta^2 = .005$. Furthermore, there were no significant Phase × Group × Emotion Regulation style interaction effects on challenge-threat scores: trait reappraisal, $F(1, 100) = 0.14, p = .713$, partial $\eta^2 = .001$; trait suppression, $F(1, 100) = 0.67, p = .531$, partial $\eta^2 = .068$; DERS, $F(1, 100) = 0.33, p = .569$, partial $\eta^2 = .003$. The manipulation did not influence physiological indices of challenge/threat, nor did habitual emotion regulation style influence responses.
Table 6.1.

*Mean (with SDs) cardiovascular parameters at each phase*

<table>
<thead>
<tr>
<th></th>
<th>Control Group (n = 54)</th>
<th>Reappraisal Group (n = 52)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Phase</td>
<td>Baseline</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>Baseline</td>
<td>124.25</td>
</tr>
<tr>
<td></td>
<td>Task 1</td>
<td>125.43</td>
</tr>
<tr>
<td></td>
<td>Task 2</td>
<td>125.43</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>Baseline</td>
<td>76.38</td>
</tr>
<tr>
<td></td>
<td>Task 1</td>
<td>75.74</td>
</tr>
<tr>
<td></td>
<td>Task 2</td>
<td>77.98</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>Baseline</td>
<td>80.48</td>
</tr>
<tr>
<td></td>
<td>Task 1</td>
<td>77.98</td>
</tr>
<tr>
<td></td>
<td>Task 2</td>
<td>77.98</td>
</tr>
<tr>
<td>CO (lpm)</td>
<td>Baseline</td>
<td>5.39</td>
</tr>
<tr>
<td></td>
<td>Task 1</td>
<td>5.32</td>
</tr>
<tr>
<td></td>
<td>Task 2</td>
<td>5.32</td>
</tr>
<tr>
<td>TPR (pru)</td>
<td>Baseline</td>
<td>1.24</td>
</tr>
<tr>
<td></td>
<td>Task 1</td>
<td>1.20</td>
</tr>
<tr>
<td></td>
<td>Task 2</td>
<td>1.20</td>
</tr>
</tbody>
</table>
Table 6.2.

*Mean (with SDs) cardiovascular reactivity for each task*

<table>
<thead>
<tr>
<th></th>
<th>Control Group</th>
<th></th>
<th>Reappraisal Group</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Stress Exposure</td>
<td></td>
<td>Stress Exposure</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Task 1</td>
<td>Task 2</td>
<td>Task 1</td>
<td>Task 2</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>18.03</td>
<td>8.36</td>
<td></td>
<td>16.43</td>
<td>10.63</td>
</tr>
<tr>
<td>12.87</td>
<td>4.95</td>
<td></td>
<td>13.53</td>
<td>8.02</td>
</tr>
<tr>
<td>5.53</td>
<td>6.46</td>
<td></td>
<td>1.10</td>
<td>5.94</td>
</tr>
<tr>
<td>0.37</td>
<td>0.68</td>
<td></td>
<td>-0.18</td>
<td>0.68</td>
</tr>
<tr>
<td>TPR (pru)</td>
<td>0.13</td>
<td>0.22</td>
<td>0.37</td>
<td>0.70</td>
</tr>
</tbody>
</table>

*Note.* Task 1 was uninstructed for both groups. Task 2 reflects CVR to the task after the experimental manipulation.
Table 6.3.

*Means, standard deviations, and range of scores for each emotion regulation scale*

<table>
<thead>
<tr>
<th></th>
<th>Control Group</th>
<th>Reappraisal Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( M )</td>
<td>( SD )</td>
</tr>
<tr>
<td>Reappraisal</td>
<td>29.37</td>
<td>5.40</td>
</tr>
<tr>
<td>Suppression</td>
<td>12.81</td>
<td>4.77</td>
</tr>
<tr>
<td>DERS</td>
<td>87.93</td>
<td>22.86</td>
</tr>
<tr>
<td>Impulse</td>
<td>12.81</td>
<td>5.12</td>
</tr>
<tr>
<td>Nonaccept</td>
<td>13.24</td>
<td>5.82</td>
</tr>
<tr>
<td>Goal</td>
<td>16.89</td>
<td>4.67</td>
</tr>
<tr>
<td>Clarity</td>
<td>12.00</td>
<td>3.29</td>
</tr>
<tr>
<td>Awareness</td>
<td>14.26</td>
<td>4.07</td>
</tr>
<tr>
<td>Strategies</td>
<td>18.88</td>
<td>7.09</td>
</tr>
</tbody>
</table>

**Affect and Task Appraisals**

At baseline, greater emotion regulation difficulties were associated with greater self-reported NA (\( r = +.339, p < .001 \)), greater self-reported stress (\( r = +.285, p = .003 \)), and lower self-reported PA (\( r = -.305, p = .002 \)). Emotion regulation difficulties were unrelated to self-reported anxiety (\( r = +.133, p = .175 \)). Habitual use of reappraisal and suppression were unrelated to affect at baseline, all \( ps > .123 \).

There was a significant Phase \( \times \) Group interaction effect on pre-task resource appraisals (\( p = .026 \)). Individuals in the reappraisal group reported similar resource appraisals in anticipation of Task 1 and Task 2, but individuals in the control group reported significantly lower resources to cope with Task 2 compared to Task 1. Means
and standard deviations are reported in Table 6.4. There were no other Phase × Group interaction effects on PA, NA, state stress, state anxiety, demand/resource appraisals, or post-task appraisals (stressfulness, anxiety, difficulty, challenge-appraisals), all $p$s > .095. Full results are reported in Table 6.5. The manipulation did little to alter the psychological experience of the task.

There was a significant Phase × Group × Trait Reappraisal interaction effect on demand appraisals. Post-hoc tests found that individuals scoring higher in trait reappraisal, in the reappraisal group, reported more demands in anticipation of the first task, $r = +.270, p = .053$; however, in anticipation of the second task, after reappraisal instructions, this was no longer the case, $r = -.083, p = .561$. For individuals in the control group there was no relationship between trait reappraisal and reported demands, (task 1; $r = -.208, p = .140$, task 2; $r = +.223, p = .105$). There were no other Phase × Group × Emotion Regulation Style interaction effects. Full results are reported in Table 6.5. Overall, the manipulation had little effect on the psychological experience of the task. Individuals in control group appeared to report less perceived resources to cope with Task 2. While, individuals scoring high in trait reappraisal, instructed to reappraise, reported less perceived demands prior to Task 2.

**Manipulation checks**

There was no significant difference in self-reported reappraisal between the two groups, $t(101.57) = 0.06, p = .950, [-0.45, 0.45]$. While performance on the task increased from Task 1 ($M = 40.59$, $SD = 17.98$) to Task 2 ($M = 48.11$, $SD = 29.02$), $F(1, 81) = 13.16, p < .001$, partial $\eta^2 = .140$, neither the reappraisal manipulation, nor trait emotion regulation style, influenced performance. Full results reported in Table 6.5.
Figure 6.2. CO habituated to Task 2, while TPR increased during Task 2.
### Table 6.4.

**Mean (with SDs) self-report measures**

<table>
<thead>
<tr>
<th></th>
<th>Control Group</th>
<th>Reappraisal Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Task 1</td>
<td>Task 2</td>
</tr>
<tr>
<td>NA</td>
<td>1.83</td>
<td>4.36</td>
</tr>
<tr>
<td>PA</td>
<td>-1.25</td>
<td>5.34</td>
</tr>
<tr>
<td>State_S</td>
<td>0.92</td>
<td>2.25</td>
</tr>
<tr>
<td>State_A</td>
<td>0.68</td>
<td>2.40</td>
</tr>
<tr>
<td>Demands</td>
<td>8.31</td>
<td>3.22</td>
</tr>
<tr>
<td>Task Difficulty</td>
<td>4.21</td>
<td>1.47</td>
</tr>
<tr>
<td>Task Stress</td>
<td>3.70</td>
<td>1.42</td>
</tr>
<tr>
<td>Task Anxiety</td>
<td>4.04</td>
<td>1.41</td>
</tr>
<tr>
<td>Task Challenge</td>
<td>4.48</td>
<td>1.16</td>
</tr>
<tr>
<td>Spon_Reapp</td>
<td>3.22</td>
<td>1.41</td>
</tr>
</tbody>
</table>

*Note.* Change scores were computed for NA, PA, state stress, and state anxiety. State_S/A = state stress/anxiety; Spon_Reapp = self-reported use of reappraisal during the task.
Table 6.5.

*Phase × Group (Manipulation), and Phase × Group × (Trait) Emotion Regulation interaction effects on task appraisals*

<table>
<thead>
<tr>
<th></th>
<th>Manipulation</th>
<th>Trait Reappraisal</th>
<th>Trait Suppression</th>
<th>DERS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>F</td>
<td>η_p^2</td>
<td>p</td>
<td>F</td>
</tr>
<tr>
<td>NA</td>
<td>0.16</td>
<td>.002</td>
<td>.692</td>
<td>0.87</td>
</tr>
<tr>
<td>PA</td>
<td>0.59</td>
<td>.006</td>
<td>.445</td>
<td>1.25</td>
</tr>
<tr>
<td>State_S</td>
<td>0.15</td>
<td>.001</td>
<td>.702</td>
<td>0.21</td>
</tr>
<tr>
<td>State_A</td>
<td>0.01</td>
<td>.001</td>
<td>.972</td>
<td>3.57</td>
</tr>
<tr>
<td>Demands</td>
<td>0.31</td>
<td>.003</td>
<td>.581</td>
<td>13.16</td>
</tr>
<tr>
<td>Resources</td>
<td>5.07</td>
<td>.047</td>
<td>.026*</td>
<td>2.03</td>
</tr>
<tr>
<td>Task Difficulty</td>
<td>0.44</td>
<td>.004</td>
<td>.508</td>
<td>0.24</td>
</tr>
<tr>
<td>Task Stress</td>
<td>0.42</td>
<td>.004</td>
<td>.520</td>
<td>0.28</td>
</tr>
<tr>
<td>Task Anxiety</td>
<td>2.85</td>
<td>.027</td>
<td>.095</td>
<td>0.31</td>
</tr>
<tr>
<td>Challenge</td>
<td>0.26</td>
<td>.002</td>
<td>.612</td>
<td>0.05</td>
</tr>
<tr>
<td>Performance</td>
<td>3.12</td>
<td>.037</td>
<td>.081</td>
<td>2.57</td>
</tr>
</tbody>
</table>

*Note.* *p < .05. State_S/A = state stress/anxiety
Chapter 6, Study 5: Instructed Reappraisal and Habituation

Discussion

This study confirmed that participants exhibited significant cardiovascular habituation on exposure to the same stressor; however, reappraisal instructions had no influence on the degree of habituation. Furthermore, reappraisal instructions did not affect the psychological experience of the task and had little effect on resource and demand appraisals. However, individuals scoring high on trait reappraisal, instructed to reappraise, reported less demands prior to the second task. Habitual use of reappraisal did not interact with instructed reappraisal to influence cardiovascular responding to the task. It appears that instructed reappraisal has no influence on cardiovascular adaptation to recurrent stress.

This was the first study to examine if instructed reappraisal influences patterns of cardiovascular habituation to stress. While, previous research has provided consistent evidence that instructed reappraisal results in a cardiovascular challenge response to a single stress exposure (e.g., Hangen et al., 2019; Jamieson et al., 2012, 2013), the results of this study suggest that reappraisal has little influence on adaption to recurrent stress. This relationship, or lack thereof, was not influenced by habitual use of reappraisal. However, considering that in the present study the reappraisal manipulation had little effect on CVR, the potential moderating role of habitual reappraisal needs further examination.

Likewise, instructed reappraisal did not influence the psychological experience of the task, or influence resource/demand appraisals. In fact, across a range of measures (CVR, affect, task appraisals) instructed reappraisal had no effects. This contrasts with previous research, where instructions to reappraise stress were associated with greater perceived resources (Beltzer et al., 2014; Jamieson et al., 2012, 2013). Indeed, greater
Chapter 6, Study 5: Instructed Reappraisal and Habituation

perceived resources are argued to be the mechanism through which reappraisal influences physiological responding, consistent with the predictions of the transactional model of stress and coping and the biopsychosocial model (Lazarus & Folkman, 1987; Tomaka et al., 1997). If individuals perceive their stress response as adaptive and helpful, that is, see stress responses as a resource, this should be reflected by their cardiovascular responses. In the present study, instructed reappraisal did not lead to greater perceived resources which may explain the observed lack of cardiovascular adaption for those in the reappraisal group.

Methodological differences between the current study and previous research must be considered when interpreting the observed results. The instruction period used in the present study was significantly shorter than that employed by other research. Participants heard instructions over a three-minute period, while other research has used an arguably more comprehensive manipulation lasting 10-15 minutes; participants heard the reappraisal instructions (as in this study) but also read summaries of three scientific journal articles (some real, some imaginary) endorsing this information (Hangen et al., 2019; Jamieson et al., 2012, 2013; Jamieson et al., 2016). It is possible that our method was too brief. Further research is warranted to examine if the length, and/or format, of the instruction period influences the relationship between instructed reappraisal and physiological responding.

Furthermore, the control manipulation employed differs from some other research, where participants were asked complete a non-demanding task, or ignore stress by focussing on an X displayed in front of them (e.g., Jamieson et al., 2012; Jamieson et al., 2013; Jamieson et al., 2016). Participants in the present study were simply informed that arousal during stressful situation is normal; instructions adapted from Jamieson et al.
Chapter 6, Study 5: Instructed Reappraisal and Habituation (2010). However, this may have led to acceptance of physiological arousal, rather than an “uninstructed” control condition. Indeed, studies which manipulate acceptance typically instruct participants to view arousal as normal, and have reported no differences between acceptance and reappraisal groups in terms of skin conductance and HR responding (Hofmann et al., 2009; Wolgast et al., 2011).

Furthermore, our study differs from previous research such that reappraisal was instructed after an initial stress exposure. While the purpose of this was to examine if instructed reappraisal influences adaption to recurrent stress, it is a methodological difference which must be noted. The process model of emotion regulation construes reappraisal as an antecedent strategy; it occurs before the emotion experience has been fully elicited (Gross, 1998a, 1998b). Considering reappraisal is an antecedent strategy yet was instructed after an initial stress exposure this may, or may not, have influenced participant’s ability to employ reappraisal. Further research is needed to clarify if the timing of reappraisal instructions influences the ability to engage in this strategy.

Despite these methodological considerations the present study addressed a number of limitations inherent in emotion regulation research, which adds significant weight to the observed results. Unlike previous studies an official acclimatization period followed by a Vanilla baseline period were employed. This strengthens the internal validity of the observed cardiovascular responses; participants were fully rested prior to engaging in the task, reflecting a more accurate assessment of cardiovascular parameters at rest. Furthermore, cardiovascular reactivity was computed using the recommended approach. The influence, or lack thereof, of reappraisal on blood pressure was also examined. Past research typically reports CO and TPR responding but fails to report blood pressure responses; the present study addresses this gap.
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The current study was the first to examine if instructed reappraisal influences habituation and demonstrated that reappraisal instructions had little impact on the psychological and physiological experience of the stressor. Much research suggests that cardiovascular reactivity to recurrent stress may help elucidate links between CVR and future disease risk; an aspect of the CVR hypothesis that has been ignored by emotion regulation research to date. While the methodological differences highlighted need to be addressed by future research, it is possible that instructed reappraisal does little to buffer the stress response, underscoring the importance of considering individual differences in this emotion regulation strategy.
Chapter 7: Discussion

CHAPTER 7

Discussion

Integrated Summary of Studies

The primary focus of this thesis was to examine the influence of emotion regulation style on stress responsivity. Typically, successful emotion regulation, such as the use of reappraisal, is associated with a more adaptive physiological response profile during acute stress. Habitual reappraisal is also associated with greater self-reported indices of well-being. In contrast, difficulties in emotion regulation and use of certain strategies, such as suppression, have been associated with poorer psychological health and exaggerated cardiovascular reactivity to acute stress; an established indicator of future cardiovascular disease risk (e.g., Chida & Steptoe, 2010). The present research therefore targeted methodological shortcomings with previous studies examining this relationship, in order to elucidate psychosomatic pathways through which individual differences in emotion regulation style may influence physical health.

First, a standardized laboratory stress paradigm was employed. Second, more sophisticated indices of cardiovascular responding than previously used were assessed, namely blood pressure, CO, and TPR. Third, CVR to both an active and passive stressor was examined. Outside of these methodological refinements, we choose to primarily examine individual differences in use of reappraisal and suppression, as well as emotion dysregulation. Previous research focuses on instructed use of these strategies rather than the propensity to engage in these strategies in day-to-day life. Examination of habitual
emotion regulation style may be more pertinent in understanding the long-term consequences of reappraisal and suppression use. This overcomes another potential issue; self-report bias. Instructions to “feel nothing” or “feel less negative emotion” may have influenced self-reported emotion in research that instructed the use of emotion regulation strategies. By not manipulating the strategy employed and instead measuring trait emotion regulation style, self-reported emotions may be more reflective of individual differences in the experience of the stressor.

Overall, five empirical studies were reported; one online cross-sectional study and four laboratory-based studies, one with a prospective design. The first study examined if individual differences in emotion regulation style were associated with perceptions of stress, and how this may translate to psychological health. Three laboratory studies then examined the relationship between emotion regulation and responses to acute stress. One laboratory study sought to establish the validity of a novel passive stress task.

**Overview of Study 1**

Study 1 examined the association between individual differences in emotion regulation and perceived stress. It is well-documented that habitual use of reappraisal is associated with greater indices of well-being (e.g., Balzarotti et al., 2017; Gross & John, 2003), while suppression is associated with greater anxiety, depression, and experience of NA (e.g., Appleton et al., 2013; Nolen-Hoeksema & Aldao, 2011). Likewise, greater emotion regulation difficulties are associated with poorer indices of psychological health (Ghorbani et al., 2017; Ritschel et al., 2015; Salters-Pedneault et al., 2006). This study aimed to examine if emotion regulation style was associated with perceptions of stress in
daily life, and if this could elucidate the links between emotion regulation style and psychological well-being.

This study confirmed that greater habitual use of reappraisal was associated with greater satisfaction with life but was unrelated to anxiety and depressive symptoms (distress) or perceived stress. In contrast, both greater habitual use of suppression and greater difficulties in emotion regulation were associated with greater anxiety and depression symptoms (distress), and greater perceived stress, but unrelated to satisfaction with life. Mediation analyses confirmed that perceived stress mediated the relationship between suppression and distress, and between emotion regulation difficulties and distress; such that individuals scoring higher in habitual use of suppression, and in difficulties in emotion regulation, perceived more stress and this was associated with greater distress.

The results of this study suggest that even in the absence of an acute stressor individuals who report greater habitual suppression use, and greater emotion regulation difficulties, perceive more stress in daily life. This may be a pathway through which suppression and emotion regulation difficulties are associated with poorer psychological health, and by extension perhaps physical health. Therefore, the next chapter aimed to examine the influence of individual differences in emotion regulation on responses to acute stress in the laboratory.

**Overview of Study 2**

Study 2 examined the influence of individual differences in emotion regulation on CVR to an active stress task. Previous research has demonstrated that a hypothesized “healthful” strategy, reappraisal, is generally associated with lower SNS responding and lower self-reported NA during acute stress (e.g., Gruber et al., 2014; McRae et al., 2012;
Urry, 2001). Use of suppression is generally associated with greater SNS responding with little or no change in self-reported affect (e.g., Dillon et al., 2007; Gross, 1998b; Richards & Gross, 1999; N. A. Roberts et al., 2008). However, these results have typically been observed when participants are instructed to use either suppression or reappraisal during a passive stress task, such as viewing film-clips, and SNS responding is typically assessed via skin conductance. Thus, Study 2 examined this relationship by measuring trait indices of reappraisal and suppression use and included blood pressure responding as an outcome; with a particular focus on the underlying hemodynamic determinants of blood pressure. This study also explored the relationship between emotion regulation difficulties and cardiovascular responding to stress. Furthermore, it addressed a number of key limitations with previous studies; it employed a standardized laboratory stress paradigm and an active coping context. The emotional content of the speech task was manipulated; one task involved speaking about neutral-emotion words, the other included negative-emotion words.

Similar to research instructing the use of suppression, Study 2 found greater reported use of habitual suppression was associated with greater SBP and DBP responding to both the negative and neutral emotion task. Trait reappraisal had no influence on blood pressure responses, which mirrors previous research (Memedovic et al., 2010). However, consistent with other research, a relationship was observed between habitual reappraisal use and the underlying determinants of blood pressure (Mauss, Cook, Cheng, et al., 2007). Individuals reporting a greater propensity to use reappraisal demonstrated a more myocardial, or challenge-oriented, cardiovascular response during the negative emotion task; indexed by greater CO and lower TPR responding. This challenge-oriented response was confirmed by examination of HP values and the
challenge-threat index. This study was to first to examine the influence of emotion regulation difficulties on CVR to acute stress; individuals with greater emotion regulation difficulties exhibited a vascular, or a threat-oriented, cardiovascular response during the negative-emotion speech task; indexed by greater TPR and lower CO responding. This vascular response profile was confirmed by the application of the HP-CD model and the challenge-threat index. Interestingly, neither trait reappraisal nor emotion regulation ability influenced CVR to the neutral task, suggesting the emotional content of the stressor is important.

This study was the first to connect two separate approaches of classifying patterns of CO and TPR responding; the HP-CD computation model (Gregg et al., 2002) and the challenge-threat index (Blascovich et al., 2004; Seery et al., 2010). The HP-CD model claims to categorize responders as myocardial, vascular, or mixed. While, the challenge-threat index claims to classify responders as demonstrating a challenge response (higher values) or a threat response (lower values). This study confirmed these computations have construct validity; there was considerable overlap between individuals classified as exhibiting a myocardial response and a challenge-response. Likewise, individuals exhibiting a vascular response (as indexed by the HP-CD model) were identified as showing a threat response (as indexed by the challenge-threat index).

In terms of self-reported affect, none of the emotion regulation variables influenced the psychological experience of either task; participants reported similar levels of stress, anxiety, PA, and NA regardless of trait emotion regulation style. However, at baseline, individuals with greater difficulties in emotion regulation reported greater perceived stress, anxiety, and NA; this mirrors the results from Study 1.
Furthermore, this study also assessed self-reported, or spontaneous use of, reappraisal and suppression during the task. Interestingly, habitual use of each strategy was not related to implementation of this strategy during the task. There appears to be a disconnect between habitual use of a strategy and use of it during acute stress, or perhaps, in reporting the use of it during stress. Furthermore, neither self-reported use of reappraisal nor suppression influenced cardiovascular responding to the task.

These findings build on results from the handful of studies examining trait emotion regulation use and CVR. Furthermore, it collaborates results from experimental research that manipulates the emotion regulation strategy employed. The results suggest the propensity to engage in these strategies in daily life translates to CVR when responding naturally to a stressor in the laboratory. In particular, the observed effects on CO and TPR responding points towards the inclusion of an active coping context to examine the effects of emotion regulation style; a hypothesis consistent with the biopsychosocial model of challenge and threat (Blascovich & Tomaka, 1996). Perhaps, as the biopsychosocial model posits, only when a task sufficiently elicits a motivated performance situation can we observe physiological indices of challenge and threat. The next chapters aimed to address this by examining if individual differences in emotion regulation influence cardiovascular responding during a passive stress task; particularly as much of the literature to date reports on instructed emotion regulation and physiological responding within a passive coping context.

**Overview of Study 3**

While Study 2 examined the relationship between trait emotion regulation and cardiovascular responding during *active* stress, much of the previous research in this area has focused on responses during *passive* stress. We therefore aimed to replicate findings
from the instructed emotion regulation literature that reports a relationship between reappraisal and lower SNS activity, and between suppression and greater SNS activity, during passive stress.

However, previous passive tasks are limited in how ecologically representative they are of actual coping situations in day-to-day life (Aldao, 2013). Furthermore, tasks involving physical stimuli, such as the cold pressor, may result in greater vascular responding due to temperature changes (Epstein et al., 1969) – not due to the actual psychological experience of the task. To overcome such limitations, this chapter examined the construct validity of a novel psychological passive stress task in order to establish if emotional regulation influenced stress reactivity to a passive stressor. During this task, participants viewed a video-clip of themselves completing an active stress task, the speech task used in Study 2 (similar to; Harris, 2001; Hartley et al., 1999; Schwerdtfeger & Rosenkaimer, 2011; Soye & O'Súilleabháin, 2019). While previous research has established that this task effectively elicits a cardiovascular stress response, the type of response elicited has not been examined (e.g., Schwerdtfeger & Rosenkaimer, 2011; Soye & O'Súilleabháin, 2019). Passive stressors are hypothesised, and have been shown, to elicit a more vascular response profile in comparison to active stressors, where increases in blood pressure are driven by increases in TPR (e.g., Hurwitz et al., 1993; Obrist et al., 1978; Saab et al., 1993). Furthermore, past research employing this task required participants to watch the video-recording with the experimenter, and/or confederates, alongside them; therefore, the observed results may be confounded by evaluation apprehension. This study therefore sought to test if this passive task had construct validity, such that it elicited a pattern of cardiovascular responding distinct from the active task and typical of passive coping.
Study 3 confirmed that the activity of watching oneself complete the task elicited a stress response; SBP and DBP increased from baseline to task. Examination of the underlying hemodynamic profile found that the active task elicited a myocardial response, while the passive task elicited a mixed hemodynamic response. Importantly, the passive stress task elicited lower blood pressure responding than the active task, which is typical of other passive tasks in the literature (e.g., Nyklicek et al., 2005; Patterson et al., 1994). Furthermore, this pattern of responding was driven by increases in TPR, with little change in CO. In contrast, blood pressure responses to the active task were driven by increased cardiac activity (HR and CO). The observed results are consistent with previous studies comparing passive and active stressors (e.g., Nyklicek et al., 2005; Sherwood et al., 1990; Winzer et al., 1999). Likewise, the psychological experience of the passive task and active task were comparable; both tasks elicited similar levels of NA, PA, stress, and anxiety. As the passive task elicited cardiovascular responding consistent with that elicited by passive coping contexts, the relationship between trait emotion regulation and CVR to this task was examined in the next chapter.

Overview of Study 4

Study 4 examined the influence of individual differences in emotion regulation on stress responsivity during the validated passive stress task established in Study 3. This study found individual differences in habitual use of suppression and reappraisal did not influence blood pressure responses to the passive task. Likewise, emotion regulation difficulties did not influence blood pressure reactivity.

Consistent with the results of Study 2, individual differences in emotion regulation did not influence self-reported affect during the task. However, at baseline, individuals with greater difficulties in emotion regulation reported more stress, anxiety,
and NA than individuals with less difficulties; consistent with the results of Study 1 and Study 2. In the absence of stress individuals with greater emotion regulation difficulties perceive more stress and negative emotion, but this does not appear to be the case during an acute stress exposure.

In this study, habitual use of reappraisal and suppression were not related to self-reported use of these strategies (spontaneous reappraisal/suppression) in response to acute stress. This is consistent with the results of Study 2. In both an active and passive coping context, habitual use of these strategies did not influence self-reported use of these strategies when faced with an acute stressor. Furthermore, self-reported use of reappraisal and suppression did not translate to patterns of CVR.

Few studies have examined the relationship between individual differences in emotion regulation and CVR to passive (and active) stress, therefore it is difficult to draw conclusions. It appears that in response to a passive stress task emotion regulation style does not influence CVR. However, participants reported greater spontaneous use of reappraisal and suppression during the active task compared to the passive task. It may be the case that the effects of individual differences in emotion regulation on stress responding are stronger, or more observable, within active coping contexts.

The emotional content of the task must also be considered. In this study participants watched the neutral version of the speech task. Perhaps individual differences in emotion regulation have a greater influence on CVR when the stress task is negative in nature; a hypothesis consistent with research suggesting suppression only influences SNS responding when the stressor is negative in nature (Gross & Levenson, 1997). The results of Study 2 somewhat support this proposition; habitual reappraisal and emotion regulation difficulties only influenced CVR during the negative task. However,
it does not explain why suppression led to exaggerated SBP and DBP responding to both the negative and neutral tasks.

Of course, the small sample size needs to be acknowledged, and it may be the case that we did not have power to detect significant effects. Although the sample employed was sufficiently powered to detect large effects, considering the lower range of reactivity scores in response to passive task, perhaps we needed a larger sample to detect small to medium size effects.

Overview of Study 5

Study 2 provided evidence that habitual reappraisal use was associated with a more challenge-oriented cardiovascular response. This is consistent with a large literature reporting that trait reappraisal (Mauss, Cook, & Gross, 2007) and instructed reappraisal (e.g., Hangen et al., 2019; Jamieson et al., 2012, 2013; Oveis et al., 2018) are associated with a challenge-oriented cardiovascular response. A limitation of this research is that the influence of instructed reappraisal on CVR is only examined during a single stress exposure, no research has examined instructed reappraisal and habitation to recurrent stress. Previous research has successfully manipulated performance feedback prior to the second stress exposure and observed differences in habituation depending if negative or positive feedback was provided (Brown & Creaven, 2017). Therefore, Study 5 aimed to examine if instructed reappraisal would influence patterns of cardiovascular habituation to recurrent stress, and if trait reappraisal would moderate this relationship.

Participants completed the speech task employed in Study 2, and prior to completing the task for a second time received either reappraisal or control instructions. It was hypothesized that instructed reappraisal would result in more pronounced habituation to the second task and greater resource appraisals to cope with the stressor.
Contrary to our expectations, instructed reappraisal did not influence these outcomes, nor did trait reappraisal interact with reappraisal instructions to predict CVR. While all participants demonstrated cardiovascular habituation to the second task, instructions to reappraise the task did not moderate the degree of habituation. This study was also the first to examine the influence of arousal reappraisal on self-reported affect; similar to Study 2 and Study 4 which assessed habitual emotion regulation style, instructed reappraisal had no influence on self-reported NA, PA, stress, or anxiety.

The current study provides evidence that instructed reappraisal does not buffer the physiological stress response, at least in terms of habituation to recurrent stress. This is the first study to examine instructed reappraisal and habituation and suggests avenues for future research. In particular, the present study had a number of methodological strengths which add weight to the results, such as the inclusion of a standardized laboratory stress paradigm, computation of CVR using the recommended approach, and the use of a standardized speech task. However, there are a number of methodological differences between our study and research reporting a significant relationship between instructed reappraisal and CVR. Future research needs to examine the influence of the timing and/or format of reappraisal instructions, and the manipulation of the control group, on patterns of habituation. The finding that instructed reappraisal did not influence cardiovascular responding suggests that individual differences in the habitual propensity to engage in reappraisal may be more important in determining responses to stress.

Overall Implications of the Findings

The cardiovascular reactivity hypothesis has amassed substantial research over the last forty years (Chida & Steptoe, 2010; Lovallo, 2010; Obrist, 1981), demonstrating
that exaggerated cardiovascular responding to acute stress is predictive of the development of cardiovascular disease. A separate, yet overlapping area of research, has provided evidence that instructed use of certain emotion regulation strategies alters physiological responses to acute stress tasks such as viewing film-clips, viewing images from the IAPS, and completing speech tasks (e.g., Butler et al., 2003; Gruber et al., 2014; Harris, 2001; Hofmann et al., 2009; Quartana & Burns, 2010). More recently, research has shown instructed reappraisal is associated with a challenge-oriented cardiovascular response profile (e.g., Jamieson et al., 2010; Jamieson et al., 2012, 2013). Therefore, this thesis examined the relationship between emotion regulation and stress responsivity, addressing some of the methodological shortcomings in this area. We provide some evidence that habitual use of these strategies has consequences for CVR; however, the coping context elicited, and emotional nature of the task need to be considered. The hypothesized effects of instructed reappraisal on cardiovascular habituation were not confirmed.

**Individual Differences in Emotion Regulation and CVR**

**Suppression use.** As outlined throughout the thesis, a large body of research reports instructed use of suppression leads to heightened SNS responding in the laboratory (e.g., Gross, 1998b; N. A. Roberts et al., 2008). While this may act as a proxy for how habitual use of this strategy, in day-to-day life, influences physiological responding, the correlates of habitual suppression use are not well-documented. To our knowledge, the only study which assessed both habitual use of suppression and blood pressure responding, found no relationship between these indices in response to a speech task (Memedovic et al., 2010). We tested this relationship using a well-established CVR paradigm and a standardized speech task and provided support for the hypothesis that
trait suppression leads to exaggerated CVR responding, in terms of SBP and DBP. Furthermore, this response was observed during the negative- and neutral-emotion versions of this task, extending past research that posited suppression may only alter responding when the stimulus is negative (Gross & Levenson, 1997; Wegner & Gold, 1995).

If the tendency to engage in suppression is indeed physiologically taxing this may have long-term implications for physical health. In particular, this warrants further attention considering that several studies have reported a relationship between inhibition of negative emotions and heightened CVD risk, including greater incidence of heart disease and all-cause mortality (e.g., Denollet et al., 1996; Gallacher et al., 1999; Grossarth-Maticek et al., 1985; Jorgensen et al., 1996; M. Julius et al., 1986). Exaggerated cardiovascular reactivity to stress may be the mechanism through which the inhibition of negative emotion is linked to poorer physical health outcomes.

Conversely, it appears that habitual use of suppression has little influence on cardiovascular responding during acute passive stress. However, the passive nature and/or neutral-emotion nature of the task employed may have influenced responding. Likewise, a relatively small sample was employed ($N = 25$), thus the study may have lacked sufficient power to detect small and medium effects. It is difficult to situate the present findings in the context of previous research considering the lack of research assessing trait suppression. This thesis presents evidence that the habitual use of suppression is related to greater perceived stress in daily life, and by association, lower psychological well-being. Furthermore, the results support past research reporting a relationship between instructed suppression and greater SNS responding; when faced with an acute active stressor, individuals who habitually engage in suppression exhibited...
exaggerated SBP and DBP responding. This research suggests individual differences in suppression influence cardiovascular responding to acute stress, which may have implications for physical health in the longer term.

**Reappraisal use.** The results suggest that habitual use of reappraisal is not associated with perceived stress in day-to-day life. However, in response to acute stress, individuals reporting a greater propensity to engage in reappraisal exhibited a more challenge-oriented cardiovascular response. This mirrors a wealth of research demonstrating instructed reappraisal (Hangen et al., 2019; Jamieson et al., 2012, 2013) and trait reappraisal (Mauss, Cook, Cheng, et al., 2007) are associated with challenge-oriented cardiovascular responding; suggesting that habitual use of reappraisal may buffer physiological responses to stress.

It is worth noting that habitual use of reappraisal did not influence cardiovascular responding during the neutral-emotion active task nor patterns of CO/TPR responding during a neutral-emotion passive task. This may be due to the emotional content of the task, and in the case of the latter, the passive context elicited by the task. Indeed, previous research reporting a relationship between habitual reappraisal and a myocardial response, employed a mental arithmetic task (active task) during which participants received negative feedback designed to elicit anger (Mauss, Cook, Cheng, et al., 2007). Similarly, studies employing instructed reappraisal found this response pattern during active tasks that involved negative feedback (Jamieson et al., 2012, 2013). The emotional content or nature of the stress task appears important. Although both the neutral and negative tasks were appraised as being equally stressful, clear patterns of cardiovascular responding emerge, dependent on trait reappraisal, for the negative-emotion task. This research suggests for reappraisal processes to be initiated the stressor must be negative in
nature for it to influence CVR. Furthermore, as previously highlighted, trait reappraisal may not have influenced CO/TPR responding to the passive task as this task did not require active engagement. The biopsychosocial model posits that indices of challenge and threat states are only elicited during motivated performance situations, such as active tasks. Indeed, published studies typically report significant effects of instructed reappraisal and trait reappraisal on CO and TPR responding during active stress tasks.

Although previous research has reported a relationship between reappraisal and a myocardial response, these studies do not report analyses including SBP or DBP (e.g., Hangen et al., 2019; Jamieson et al., 2012, 2013; Mauss, 2007). As a result, there is limited evidence available to suggest that trait reappraisal influences blood pressure responding. This thesis addressed this limitation and reports that trait reappraisal was not associated with SBP or DBP responding during an active and a passive stressor. The lack of a relationship between reappraisal and blood pressure reactivity is notable considering support for the CVR hypothesis mainly stems from exaggerated SBP and DBP reactivity to stress (for a review see; Chida & Steptoe, 2010). If an individual’s habitually tendency to engage in reappraisal is unrelated to blood pressure reactivity this questions the validity of the hypothesized protective benefits of reappraisal, at least in terms of physical health. Certainly more research is needed to elucidate the influence of a vascular versus a myocardial response during active stress on physical health outcomes in the long-term.

**Difficulties in emotion regulation.** This thesis is the first to explore how individual differences in emotion regulation difficulties, also termed emotion regulation competencies, may affect cardiovascular responding to stress. Although greater emotion regulation difficulties have been associated with lower resting HRV (Visted et al., 2017;
Williams et al., 2015), an indicator of a less flexible parasympathetic nervous system, no previous research has examined how the ability to regulate emotions may translate to CVR to acute stress. This thesis provided evidence that individuals with greater difficulties in emotion regulation typically perceive more stress in their environment, and this in turn may translate to poorer psychological health (in terms of life satisfaction, anxiety, and depression). In the absence of stress (i.e., during the baseline period) these individuals reported more negative emotions. Although, when faced with acute stress, difficulties in emotion regulation did not influence self-reported affect. Despite no change in affect, individuals reporting greater difficulties in emotion regulation demonstrated a more vascular, or threat-oriented, profile of cardiovascular responding.

This thesis presents preliminary evidence that greater difficulties in emotion regulation are associated with a threat-oriented, or a vascular cardiovascular response, during active stress. Vascular responses to active tasks appear to be the most damaging in terms of cardiovascular health, and have been associated with increased vascular resistance, leading to vascular hypertrophy (Folkow, 1982; Obrist, 1982, 1985; Palatini & Julius, 2009). Indeed, research has shown that individuals with greater emotion regulation difficulties have higher levels of CRP (Powers et al., 2016). Similarly, in a sample of individuals reporting chronic stress, only those who reported greater emotion regulation difficulties had greater CVD risk (Roy et al., 2018). The results from this thesis suggest a psychosomatic pathway through which difficulties in emotion regulation may lead to increased disease risk. Further research is needed to replicate the observed patterns of cardiovascular responding and ascertain the conditions under which difficulties in emotion regulation alter physiological responses to stress.
Emotion Regulation and the Affective Experience of Stress

Typically, habitual reappraisal use, when assessed outside of a laboratory stress paradigm (e.g., as part of a psychometric questionnaire pack, or via daily diary entry reports), is associated with lower NA and greater PA, while suppression is associated with greater NA. Few studies have examined how habitual emotion regulation style influences the emotional experience of acute stress exposure (self-reported NA, PA, stress, and anxiety).

The present results suggest that individual differences in emotion regulation have little influence on the affective experience of the stressor. In response to both the active task and passive task participants reported similar levels of PA, stress, and anxiety regardless of their tendency to engage in reappraisal and suppression.

Previously only a handful of studies have assessed trait emotion regulation in the context of acute stress. Habitual use of suppression has been reported to have no effect on anger experience (Memedovic et al., 2010), while greater habitual use of reappraisal has led to lower self-reported anger (Mauss, Cook, Cheng, et al., 2007; Memedovic et al., 2010), lower NA, and greater PA (Mauss, Cook, Cheng, et al., 2007) post-stressor compared to individuals scoring lower in trait reappraisal. Our results differ; trait reappraisal did not alter self-reported affect. Consistent with results found by Memedovic et al. (2010), suppression use had no influence on affect across both studies. Interestingly, we also found no differences in baseline affect. In contrast, Mauss, Cook, Cheng, et al. (2007) found greater habitual use of reappraisal was associated with lower NA, lower anger, and greater PA pre-task.

Certainly, more research is needed to elucidate how trait indices of emotion regulation style influence the affective experience of stress. If one was to consider...
research from manipulation studies alone it would appear that reappraisal results in “better” affective outcomes, while suppression has little or no influence on emotion experience. As highlighted, it is important to note that in such studies participants are explicitly told to feel less negative affect, or feel nothing, hence the effects of reappraisal and suppression may be a consequence of self-report bias. Assessing trait indices of emotion regulation use, and not instructing the strategy use, overcomes this limitation. However, we also instructed the use of reappraisal and despite being informed signs of arousal are adaptive and aid performance, this did not alter self-reported affect.

This was the first research to examine if, and how, emotion regulation difficulties influence the affective experience of acute stress. Previous research has found that, in the absence of acute stress, greater emotion regulation difficulties are associated with greater NA (Pollock et al., 2016; Salsman & Linehan, 2012), and higher scores on measures of depression, chronic worry, anxiety, and perceived stress (e.g., Allan et al., 2015; Bardeen et al., 2012; Ritschel et al., 2015; Roemer et al., 2009; Ruganci & Gencoz, 2010; Salters-Pedneault et al., 2006). Indeed, the results from this thesis confirmed individuals with greater difficulties in emotion regulation perceive more stress and typically report greater negative emotions. However, when presented with an acute stress-task these individuals report similar levels of affect as those reporting less difficulties in regulating their emotional responses.

There are a number of possibilities as to why individual differences in emotion regulation are associated with distinct patterns of self-reported affect when assessed in the absence of a laboratory stressor – yet have no influence on self-reported affect in the context of a stress exposure. Perhaps, emotion regulation style does not influence the psychological experience of stress, and instead influences daily affect through some other
mechanism. Alternatively, individual differences in reappraisal/suppression and emotion regulation difficulties do indeed influence the affective experience of stress; however, people are not able to accurately report their stress experience. Indeed, the latter is supportive of a large literature highlighting discordance between subjective stress ratings and physiological responding (e.g., Ginty et al., 2012; Lee & Hughes, 2014; O'Súilleabháin et al., 2018); suggesting during times of acute stress people are not always consciously aware of their emotions. A number of studies have documented this disunity, and typically concordance between physiological responding and self-reported affect is poor (for reviews see; Campbell & Ehlert, 2012; Dickerson & Kemeny, 2004).

The results presented in this thesis suggest that self-reported affect indices were not related to emotion regulation style, suggesting that irrespective of dispositional emotion regulation style, similar levels of stress, NA, PA, and anxiety are experienced by all participants during active and passive stress exposures. Further research is needed to replicate the present findings and elucidate why emotion regulation style influences physiological responses to stress, but not the self-reported psychological experience of acute stress.

**Instructed Reappraisal and Habituation**

It appears that instructed reappraisal has little effect on cardiovascular habituation to recurrent stress. The ability to habituate to a second stress exposure is hypothesized to indicate an adaptive physiological response to stress; the body prepares to cope with the initial stressor and this response dissipates during the second encounter, meaning the cardiovascular system is not under prolonged SNS activation (e.g., Hughes et al., 2018; McEwen, 1998). In daily life a range of psychological stressors are experienced, such as traffic jams, work-deadlines, and interpersonal stress. Typically, the type of stress
experienced does not occur just once, but often. Therefore, examination of cardiovascular responding to recurrent stress may be more revealing in terms of the longer-term consequences of CVR on physical health. In the emotion regulation literature, numerous studies have presented evidence that instructed reappraisal results in a challenge-oriented cardiovascular response profile in response to a single stress exposure (Hangen et al., 2019; Jamieson et al., 2012, 2013; Oveis et al., 2018). The finding that instructed reappraisal does not alter patterns of habituation questions the validity of the hypothesis that reappraisal leads to adaptive CVR. If reappraisal does not aid the ability to adapt, or habituate, to recurrent stress, the influence of reappraisal on long-term health outcomes is uncertain.

The present study was the first to examine instructed reappraisal within a habituation paradigm. It was also the first to adopt a standardized laboratory stress paradigm, including an acclimatization phase and a Vanilla resting baseline. Furthermore, this study employed more reliable indices of CVR; the recommended approach for computing CVR was employed, and the inclusion of an acclimation and baseline phase ensured blood pressure parameters were at rest prior to the baseline phase. The methodological approach used strengthens the presented findings. However, a number of considerations, such as the timing and manipulation of reappraisal instructions, need to be examined by future research in order to ascertain the influence of reappraisal on habituation to stress.

**Spontaneous Use of Emotion Regulation Strategies**

Few studies have assessed the influence of spontaneous use of reappraisal and suppression on CVR during acute stress. It seems plausible that if individuals report using a particular strategy this would be reflected in their CVR to the task. Indeed, self-
reported use of suppression during a speech task has been associated with greater skin conductance responding, while reappraisal was not (Egloff et al., 2006). However, we found self-reported use of emotion regulation strategies had no effect on CVR or self-reported affect. Furthermore, there was a lack of concordance between habitual use of reappraisal and suppression and reported use of these strategies during the tasks. These results, along with the finding that instructed reappraisal did not alter patterns of habituation, suggest the importance of examining personality differences in the propensity to use these strategies in day-to-day life. Perhaps, in times of stress, people are not overly conscious of what strategy they are using, that is, they may be unconsciously reverting to their habitual tendency to regulate their emotions.

**Limitations of the Present Research**

The present thesis makes a substantial contribution to research examining the role of emotion regulation on stress responsivity. However, a number of limitations must be considered in the interpretation of the results. The sample sizes incorporating the passive stressor are modest; due to participant attrition. However, power analyses found the studies were significantly powered to detect large effects and, to an extent, detect medium effects. In particular, the results of Study 4 (reporting on the relationship between individual differences in emotion regulation and stress responsivity during the passive task) needs to be replicated, as due to the modest sample size, we may have lacked sufficient power to detect significant effects.

The reappraisal manipulation employed is limited by the brevity of the manipulation period and our manipulation of the control condition, making it difficult to ascertain if the reappraisal instructions were effective. Future research needs to
incorporate the highlighted methodological strengths of this study, and experimentally test the effects of instruction reappraisal on habituation to recurrent stress, considering the type of instruction, the conceptualization of the control group, and the timing of reappraisal instructions in their study design.

The composition of the laboratory study samples must also be taken into consideration. All participants who attended the laboratory sessions were recruited from a university sample, aged 18-25 years and not taking medication known to influence blood pressure responding. These exclusion criteria were set to ensure a “clean” cardiovascular baseline and assessment of reactivity, not confounded by medication or age. However, it may limit the generalizability of the results to older populations or individuals taking medication. Furthermore, this limits our results to “healthy” populations. Nonetheless, the association between CVR to stress tasks and future CVD risk has been established in healthy individuals.

Similarly, the observed results are limited to Western cultures. Participants in Study 1 were American and Canadian, while participants in the laboratory studies were all White Europeans (predominately Irish). There is some evidence to suggest that people from cultures which emphasize social order and harmony, such as Asian participants, tend to report greater habitual use of suppression (e.g., Gross & John, 2003; Matsumoto, Yoo, & Nakagawa, 2008), and that the costs and effectiveness of suppression vary across cultures. For example, in Western cultures, suppression use is typically associated with poorer wellbeing (e.g., Gross & John, 2003; Richards & Gross, 2000); however, in Eastern samples, positive or no associations between suppression and well-being have been observed (Cheung & Park, 2010; Soto, Perez, Kim, Lee, & Minnick, 2011; Yeung, Wong, & Lok, 2011). In contrast, habitual use of reappraisal is generally associated with
better well-being across cultures (Balzarotti et al., 2010; Gross & John, 2003; Spaapen, Waters, Brummer, Stopa, & Bucks, 2014; Yeung et al., 2011). Future research needs to replicate the results of this thesis across different cultures and explore if differences exist in trait emotion regulation style and stress responsivity in other ethnic groups and cultural contexts.

**Strengths of the Present Research**

The current research is strengthened by a number of methodological advancements. In particular, the use of a standardized laboratory stress protocol adds weight to the reported results. The inclusion of both an acclimatization period and a Vanilla baseline ensured more accurate measurement of cardiovascular parameters at rest, compared to that afforded by a one- or three-minute baseline (with no acclimatization period), and instructions to simply rest quietly. As a result, indices of cardiovascular reactivity during the stress exposure could be compared to a more reliable baseline assessment. Furthermore, physiological responding to the tasks was assessed via the Finometer PRO, which provides continuous measurement of blood pressure. This allowed for more sophisticated indices of physiological responding to be assessed compared to previous studies of emotion regulation, which typically focused on skin conductance. In addition to this, the reported research is the first (in context of emotion regulation literature) to compute reactivity using the recommended approach (e.g., Kamarck, Jennings, & Manuck, 1992; Kamarck & Lovallo, 2003; Linden et al., 2003), strengthening the observed results.

The laboratory studies reported upon employed standardized stress tasks to elicit cardiovascular stress reactions. Similar speech tasks have been used in previous research
and have been found to effectively perturb the cardiovascular system (e.g., Hughes & Callinan, 2007; O'Súilleabháin et al., 2018). Likewise, the examination of emotion regulation ability within an active coping context is arguably more realistic, or more comparable, to everyday stress experiences. In particular, a public speech task combines motivational and socio-evaluative aspects that are often lacking in paradigms involving film-clips or negative imagery (Aldao, 2013). A wealth of research within the emotion regulation literature employs such passive tasks, therefore this thesis builds on the few studies reporting on active tasks. The inclusion of an active coping context is particularly pertinent considering; (a) the biopsychosocial model postulates that active stress tasks elicit challenge/threat states, and (b) much of the literature reporting a positive relationship between reappraisal and CVR relates to patterns of challenge and threat responding.

The present research contributes to the field of stress reactivity. It adds to research employing a habituation paradigm and tested the validity of a more novel psychological passive stress task. In doing so, it provided evidence that this novel task was successful in eliciting a cardiovascular response profile characteristic of passive coping contexts; blood pressure increased, and this increase was a result of greater TPR reactivity. Likewise, the task was experienced as just as stressful as the speech task. This offers future researchers a new way to examine CVR to passive stress; an approach that overcomes limitations inherent in other passive tasks.

The present research extends literature examining the challenge-threat index. Across three experimental studies we demonstrate that patterns of challenge/threat cardiovascular responding identified using this index statistically overlapped with patterns of responding identified by the HP-CD computational model, indicative of
conceptual overlap. However, similar to earlier approaches classifying participants as challenge or threat responders (e.g., Eliot et al., 1982; Girdler et al., 1990; Sherwood et al., 1990), the challenge-threat index fails to directly consider the compensatory relationship between CO and TPR responses; an advantage offered by the HP-CD computational model. Arguably, the HP-CD model offers a more reliable assessment of challenge-threat responding, considering it accounts for this reciprocal relationship. The validity of this model has been confirmed by a number of studies (e.g., Howard et al., 2011; Hughes et al., 2011; James & Gregg, 2004; James et al., 2012; O'Leary et al., 2013; Ottaviani et al., 2006; Ottaviani et al., 2007), and could prove useful in elucidating patterns of cardiovascular responding within the emotion regulation literature.

The current research examined *trait* emotion regulation in isolation, that is, the effects of scoring higher versus lower on a continuum, in the habitual use of each strategy were compared. This approach addresses a key limitation with past research; traditionally the hypothesized effects of instructed reappraisal and suppression have emerged when comparing these strategies against one another. Therefore, it is not clear if use of each strategy itself is adaptive/harmful, or just when compared to other strategies. The current research allowed for the influence of individual differences in strategy use on CVR to be assessed. Likewise, in Study 5 we aimed to address this issue by examining the effects of instructed reappraisal against a control condition, instead of a rumination or suppression condition. However, as acknowledged we cannot be confident this condition was sufficiently uninstructed, as instructions may have encouraged awareness or acceptance of emotional responding. In particular, the focus on trait indices of emotion regulation is a strength of this research; habitual emotion regulation style may be more indicative of long-term patterns of emotion regulation compared to instructed use of
strategies in the laboratory. Indeed, the lack of observed effects of instructed reappraisal and spontaneous use of reappraisal and suppression on CVR suggests the importance of examining personality. It must be acknowledged although we refer to habitual use of reappraisal and habitual use of suppression, this does not mean that individuals scoring high in one construct score low on the other.

**Future Directions**

Although the results from this research implicate trait emotion regulation style in influencing cardiovascular responses to acute stress, this needs to be replicated by future research. While we provide support for a relationship between emotion regulation style (use of reappraisal, use of suppression, and emotion regulation difficulties) and CVR to active stress, in response to a passive stressor (with neutral-emotion content) emotion regulation style had no effect on CVR. The current research suggests a gap in the current literature, whereby the exact conditions under which trait emotion regulation influences stress responsivity needs to be established. If promoting more successful emotion regulation, such as greater use of reappraisal or targeting facets of emotion competence (e.g., awareness, acceptance), can influence cardiovascular responses to stress this may have long-term consequences for physical health.

Another consideration, not examined by this thesis, is how emotion regulation style may interact with chronic stress. Research has suggested emotion regulation ability buffers the link between chronic stress and increased risk of CVD (Roy et al., 2018). While, the goal of the current research was to provide evidence of a direct psychosomatic link between emotion regulation difficulties and CVR, it would be beneficial to examine this relationship longitudinally and attain indices of chronic stress.
This thesis did not find evidence to suggest instructed use of reappraisal alters cardiovascular habituation to stress. As previously highlighted the reappraisal manipulation and the control manipulation may have influenced the observed results. Further research is needed to explore if the instruction manipulation used was too brief; perhaps by manipulating the length and format of the instructions and examining the subsequent influence on CVR. This would establish if the results observed were due to the brevity of the instruction period, or if instructed reappraisal does not influence habituation to CVR. Likewise, it is pertinent for future research to test the effects of instructed reappraisal on habituation against a more accurate control condition. Notably, our reappraisal instruction was given after the first stress exposure; consistent with research manipulating feedback during a habituation paradigm (Brown & Creaven, 2017). More research is needed to determine if the timing of reappraisal instructions matter.

The present study adds to the scant research examining the interaction between habitual reappraisal use and instructed use of reappraisal. To date, the potential moderating role of trait emotion regulation on the ability to implement emotion regulation strategies when instructed has received limited attention. While the present research found no relationship, it must be noted that the manipulation did not alter CVR. We cannot be certain that individuals in the reappraisal group effectively engaged in this strategy, therefore we cannot draw conclusions regarding the relationship between trait and instructed reappraisal. This relationship warrants attention; if use of reappraisal can be implemented easily and quickly within a laboratory session across all participants, regardless of their tendency to engage in reappraisal in day-to-day life, this provides support for the development of reappraisal interventions (assuming reappraisal does in
fact buffer responses to stress). If, however, individuals who do not habitually engage in reappraisal, have difficulties implementing this strategy in a short laboratory paradigm it points towards the need for more longer-term interventions.

**Overall Conclusions**

The present thesis presents an enhanced methodological approach to studying the psychophysiological effects of emotion regulation. The results of this research advocate the assessment of the underlying determinants of blood pressure, CO and TPR. The reported studies provide support for the hypothesis that greater habitual use of reappraisal results in a more adaptive profile of cardiovascular responding. Likewise, we provide evidence that greater habitual use of suppression results in an exaggerated stress response during active stress. The reported results mirror research instructing the use of reappraisal and suppression but demonstrates how trait indices of emotion regulation can translate to differences in CVR during acute stress. However, conflicting evidence for this was found, where trait reappraisal and suppression did not influence cardiovascular responses during a passive stress task. Furthermore, although trait reappraisal influenced patterns of CO and TPR responding to the active task, trait reappraisal did not influence SBP and DBP reactivity; more research is needed to elucidate the relationship between trait emotion regulation and blood pressure responding. This research tested an established instructed reappraisal manipulation and applied a number of methodological refinements to this paradigm. The results suggest that instructed reappraisal does not aid cardiovascular habituation to recurrent stress. This questions the reliability of instructed reappraisal in buffering the stress response.
While the focus of the current thesis was on individual differences in reappraisal and suppression, we also explored the effects emotion dysregulation may have on stress responses. This is the first study to investigate a possible direct psychosomatic pathway through which emotion regulation difficulties may affect health. We found that individuals with greater emotion regulation difficulties demonstrated altered patterns of cardiovascular responding to a negative-emotion active stressor. Likewise, in the absence of stress these individuals appear to perceive more stress in their environment and report more NA and anxiety. Considering the links between heightened experience of negative emotion and CVD (for a review see; Suls & Bunde, 2005), this suggests another pathway through which greater emotion regulation difficulties may translate to greater CVD risk over time.

Overall this thesis provides support for the effects of individual differences in emotion regulation and cardiovascular responses to active stress. Neither instructed reappraisal, nor spontaneous use of reappraisal and suppression during the tasks, influenced CVR. The results of this research highlight the importance of examining how personality differences in emotion regulation style may translate to patterns of cardiovascular responding.
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cardiovascular disease: a review and methodologic critique. *Psychol Bull, 96*(3),
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marker of cumulative biological risk: MacArthur studies of successful aging.
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measures independently predict performance in a university course.

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8986.1986.tb00602.x


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Appendices

Appendix A. The Emotion Regulation Scale (ERQ)

INSTRUCTIONS: We would like to ask you some questions about your emotional life, in particular how you manage (control or regulate) your emotions. The questions relate to two different areas of your emotional life (a) your emotional experience, i.e. what you feel on the inside, and (b) your emotional expression, i.e. how you show your emotions in the way you behave, talk, gesture, facial expression, etc. Although some of the following questions seem similar to one another they are different in important ways so please answer carefully. Please indicate the extent to which you agree or disagree with that statement.

<table>
<thead>
<tr>
<th></th>
<th>Disagree strongly</th>
<th>Neither</th>
<th>Agree strongly</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 2 3 4 5 6 7</td>
<td>&gt; &gt; &gt; &gt; &gt; &gt;</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>When I want to feel more positive emotions (such as happiness, amusement) I change what I’m thinking about</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>A1</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>I keep my emotions to myself.</td>
<td></td>
</tr>
<tr>
<td>A2</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>When I want to feel less negative emotions (such as stress, sadness, anger), I change what I’m thinking about</td>
<td></td>
</tr>
<tr>
<td>A3</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>When I am feeling positive emotions, I am careful not to express them.</td>
<td></td>
</tr>
<tr>
<td>A4</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>When I am faced with a stressful situation, I make myself think about it in a way that helps me stay calm.</td>
<td></td>
</tr>
<tr>
<td>A5</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>I control my emotions by not expressing them.</td>
<td></td>
</tr>
<tr>
<td>A6</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>When I want to feel more positive emotion, I change the way I’m thinking about the situation.</td>
<td></td>
</tr>
<tr>
<td>A7</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>I control my emotions by changing the way I think about the situation I’m in.</td>
<td></td>
</tr>
<tr>
<td>A8</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>When I am feeling negative emotions, I make sure not to express them.</td>
<td></td>
</tr>
<tr>
<td>A9</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>When I want to feel less negative emotion, I change the way I’m thinking about the situation</td>
<td></td>
</tr>
<tr>
<td>A10</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
**Appendix B.** Difficulties in Emotion Regulation Scale (DERS)

INSTRUCTIONS: Please indicate how often the following statements apply to you by marking the correct box beside each item.

<table>
<thead>
<tr>
<th></th>
<th>Almost never</th>
<th>Sometimes</th>
<th>About half the time</th>
<th>Most of the time</th>
<th>Almost always</th>
</tr>
</thead>
<tbody>
<tr>
<td>B1</td>
<td>I am clear about my feelings</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B2</td>
<td>I pay attention to how I feel</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B3</td>
<td>I experience my emotions as overwhelming and out of control</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B4</td>
<td>I have no idea how I am feeling</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B5</td>
<td>I have difficulty making sense out of my feelings</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B6</td>
<td>I am attentive to my feelings</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B7</td>
<td>I know exactly how I am feeling</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B8</td>
<td>I care about what I am feeling</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B9</td>
<td>I am confused about how I feel</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B10</td>
<td>When I’m upset, I acknowledge my emotions</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B11</td>
<td>When I’m upset, I become angry with myself for feeling that way</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B12</td>
<td>When I’m upset, I become embarrassed for feeling that way</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B13</td>
<td>When I’m upset, I have difficulty getting work done</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B14</td>
<td>When I’m upset, I become out of control</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B15</td>
<td>When I’m upset, I believe that I will remain that way for a long time</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B16</td>
<td>When I’m upset, I believe that I’ll end up feeling very depressed</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Appendix B</td>
<td></td>
<td></td>
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<tr>
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<td>-----------------</td>
<td>-----------------</td>
<td>-----------------</td>
<td>-----------------</td>
<td>-----------------</td>
</tr>
<tr>
<td>B17 When I’m upset, I believe that my feelings are valid and important</td>
<td>Almost never</td>
<td>Sometimes</td>
<td>About half the time</td>
<td>Most of the time</td>
<td>Almost always</td>
</tr>
<tr>
<td>B18 When I’m upset, I have difficulty focusing on other things</td>
<td>Almost never</td>
<td>Sometimes</td>
<td>About half the time</td>
<td>Most of the time</td>
<td>Almost always</td>
</tr>
<tr>
<td>B19 When I’m upset, I feel out of control</td>
<td>Almost never</td>
<td>Sometimes</td>
<td>About half the time</td>
<td>Most of the time</td>
<td>Almost always</td>
</tr>
<tr>
<td>B20 When I’m upset, I can still get things done</td>
<td>Almost never</td>
<td>Sometimes</td>
<td>About half the time</td>
<td>Most of the time</td>
<td>Almost always</td>
</tr>
<tr>
<td>B21 When I’m upset, I feel ashamed with myself for feeling that way</td>
<td>Almost never</td>
<td>Sometimes</td>
<td>About half the time</td>
<td>Most of the time</td>
<td>Almost always</td>
</tr>
<tr>
<td>B22 When I’m upset, I know that I can find a way to eventually feel better</td>
<td>Almost never</td>
<td>Sometimes</td>
<td>About half the time</td>
<td>Most of the time</td>
<td>Almost always</td>
</tr>
<tr>
<td>B23 When I’m upset, I feel like I am weak</td>
<td>Almost never</td>
<td>Sometimes</td>
<td>About half the time</td>
<td>Most of the time</td>
<td>Almost always</td>
</tr>
<tr>
<td>B24 When I’m upset, I feel like I can remain in control of my behaviors</td>
<td>Almost never</td>
<td>Sometimes</td>
<td>About half the time</td>
<td>Most of the time</td>
<td>Almost always</td>
</tr>
<tr>
<td>B25 When I’m upset, I feel guilty for feeling that way</td>
<td>Almost never</td>
<td>Sometimes</td>
<td>About half the time</td>
<td>Most of the time</td>
<td>Almost always</td>
</tr>
<tr>
<td>B26 When I’m upset, I have difficulty concentrating</td>
<td>Almost never</td>
<td>Sometimes</td>
<td>About half the time</td>
<td>Most of the time</td>
<td>Almost always</td>
</tr>
<tr>
<td>B27 When I’m upset, I have difficulty controlling my behaviors</td>
<td>Almost never</td>
<td>Sometimes</td>
<td>About half the time</td>
<td>Most of the time</td>
<td>Almost always</td>
</tr>
<tr>
<td>B28 When I’m upset, I believe that there is nothing I can do to make myself feel better</td>
<td>Almost never</td>
<td>Sometimes</td>
<td>About half the time</td>
<td>Most of the time</td>
<td>Almost always</td>
</tr>
<tr>
<td>B29 When I’m upset, I become irritated with myself for feeling that way</td>
<td>Almost never</td>
<td>Sometimes</td>
<td>About half the time</td>
<td>Most of the time</td>
<td>Almost always</td>
</tr>
<tr>
<td>B30 When I’m upset, I start to feel very bad about myself</td>
<td>Almost never</td>
<td>Sometimes</td>
<td>About half the time</td>
<td>Most of the time</td>
<td>Almost always</td>
</tr>
<tr>
<td>B31 When I’m upset, I believe that wallowing in it is all I can do</td>
<td>Almost never</td>
<td>Sometimes</td>
<td>About half the time</td>
<td>Most of the time</td>
<td>Almost always</td>
</tr>
<tr>
<td>B32 When I’m upset, I lose control over my behaviours</td>
<td>Almost never</td>
<td>Sometimes</td>
<td>About half the time</td>
<td>Most of the time</td>
<td>Almost always</td>
</tr>
<tr>
<td>B33 When I’m upset, I have difficulty thinking about anything else</td>
<td>Almost never</td>
<td>Sometimes</td>
<td>About half the time</td>
<td>Most of the time</td>
<td>Almost always</td>
</tr>
<tr>
<td></td>
<td>Almost never</td>
<td>Sometimes</td>
<td>About half the time</td>
<td>Most of the time</td>
<td>Almost always</td>
</tr>
<tr>
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<td>--------------</td>
</tr>
<tr>
<td>B34</td>
<td>When I’m upset, I take time to figure out what I’m really feeling</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>B35</td>
<td>When I’m upset, it takes me a long time to feel better</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>B36</td>
<td>When I’m upset, my emotions feel overwhelming</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
</tbody>
</table>
Appendix C. Word List: ANEW

A block of forty negative and forty neutral words were chosen from the Affective Norms for English Words (ANEW; Bradley & Lang, 1999), a set of words rated in terms of valence (negative/positive emotions elicited) and arousal (high, low), with the aim to provide a verbal version of the existing IAPS (Lang, Bradley, & Cuthbert, 2008). Words for the current stress task were selected based on the cut-offs identified by Scott, O'Donnell, Leuthold, & Sereno (2009). Words were deemed negative if they had an arousal value greater than 6.00 and a valence value less than 4.00. For Study 2, the block of negative and the block of neutral words were presented, counterbalanced. For Study 5, the block of negative words were presented.

<table>
<thead>
<tr>
<th>Negative Emotion Words</th>
<th>Neutral Words</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abuse</td>
<td>Ankle</td>
</tr>
<tr>
<td>Accident</td>
<td>Paint</td>
</tr>
<tr>
<td>Afraid</td>
<td>Basket</td>
</tr>
<tr>
<td>Agony</td>
<td>Jelly</td>
</tr>
<tr>
<td>Annoy</td>
<td>Windmill</td>
</tr>
<tr>
<td>Murderer</td>
<td>Book</td>
</tr>
<tr>
<td>Stress</td>
<td>Bowl</td>
</tr>
<tr>
<td>Bankrupt</td>
<td>Building</td>
</tr>
<tr>
<td>Ambulance</td>
<td>Butter</td>
</tr>
<tr>
<td>Angry</td>
<td>Cabinet</td>
</tr>
<tr>
<td>Nightmare</td>
<td>Cat</td>
</tr>
<tr>
<td>Blackmail</td>
<td>Chair</td>
</tr>
<tr>
<td>Bloody</td>
<td>Clock</td>
</tr>
<tr>
<td>Bomb</td>
<td>Cow</td>
</tr>
<tr>
<td>Crash</td>
<td>Curtains</td>
</tr>
<tr>
<td>Tragedy</td>
<td>Detail</td>
</tr>
<tr>
<td>Victim</td>
<td>Door</td>
</tr>
<tr>
<td>Danger</td>
<td>Egg</td>
</tr>
<tr>
<td>Despise</td>
<td>Elbow</td>
</tr>
<tr>
<td>Destroy</td>
<td>Elevator</td>
</tr>
<tr>
<td>Rejected</td>
<td>Engine</td>
</tr>
<tr>
<td>Disaster</td>
<td>Lawn</td>
</tr>
<tr>
<td>Unfaithful</td>
<td>Month</td>
</tr>
<tr>
<td>---------------</td>
<td>------------</td>
</tr>
<tr>
<td>Divorce</td>
<td>Farm</td>
</tr>
<tr>
<td>Drown</td>
<td>Foot</td>
</tr>
<tr>
<td>Pain</td>
<td>Fork</td>
</tr>
<tr>
<td>Evil</td>
<td>Frog</td>
</tr>
<tr>
<td>Fear</td>
<td>Fur</td>
</tr>
<tr>
<td>Fight</td>
<td>Glass</td>
</tr>
<tr>
<td>Fire</td>
<td>Golfer</td>
</tr>
<tr>
<td>Flood</td>
<td>Kettle</td>
</tr>
<tr>
<td>Poison</td>
<td>Hairdryer</td>
</tr>
<tr>
<td>Guilty</td>
<td>Window</td>
</tr>
<tr>
<td>Gun</td>
<td>Hand</td>
</tr>
<tr>
<td>Hate</td>
<td>Hat</td>
</tr>
<tr>
<td>Horror</td>
<td>Hay</td>
</tr>
<tr>
<td>Hostage</td>
<td>Headlight</td>
</tr>
<tr>
<td>Panic</td>
<td>History</td>
</tr>
<tr>
<td>Rude</td>
<td>Horse</td>
</tr>
<tr>
<td>Jealousy</td>
<td>Ink</td>
</tr>
</tbody>
</table>
Appendix D

Appendix D. Experimental Manipulation Instructions

Instructions presented to the reappraisal condition:

“During the task you may feel anxious or stressed. For example, you might notice that your palms are sweaty, your heart may be beating faster. This is a normal stress response to the task. Increased arousal during stressful situations is not harmful. In fact, research has shown that increased arousal actually helps us perform better at tasks. Therefore, while doing this task remind yourself that arousal is beneficial and adaptive. It is helping you adapt to the stressor and perform better”.

Instructions presented to the control condition:

“The goal of this research is to examine how physiological arousal during a speech task relates to performance. During the task you may feel anxious or stressed. For example, you might notice that your palms are sweaty, your heart may be beating faster. This is a normal stress response to the task. We will analyse your blood pressure during the task as this indicates your arousal level”.

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Appendix E

Appendix E. Sample Participant Information Sheet

Title of Project: Examining how engaging in tasks affects blood pressure and mood

You are invited to take part in a research study. Before you decide, it is important that you understand why the research is being done and what it will involve. This Participant Information Sheet tells you about the purpose, risks, and benefits of this research study. If you agree to take part, we will ask you to sign a Consent Form. If there is anything that you are not clear about, we will be happy to explain it to you. Please read this information sheet. You should only consent to participate in this research study when you feel you understand what is being asked of you, and you have had enough time to think about your decision. Thank you for reading this.

Purpose of the Study: This study is concerned with the effect that completing a speech task has on mood and blood pressure. You have been asked to take part because significant insights can be gained by examining healthy people. We will ask you to report your mood using standard forms for that purpose, and we wish to measure your blood pressure at rest and while you perform some tasks in the laboratory.

TAKING PART

Do I have to take part? It is up to you to decide whether or not to take part. If you do decide to take part you will be given this information sheet to keep and be asked to sign a consent form. If you decide to take part you are still free to withdraw at any time and without giving a reason. A decision to withdraw at any time, or a decision not to take part, will not affect your rights in any way.

What will happen to me if I take part? We will ask you to answer some standard questionnaires that ask you about your general health and lifestyle (e.g., whether you are a smoker, drink alcohol) and some personality scales. We also wish to measure your blood pressure at rest and while you perform some tasks in the laboratory. This laboratory session will last approximately 53 minutes. You will be asked to complete the same task twice, separated by a 10-minute rest and instruction period. This task will be video-recorded.

What are the possible benefits in taking part? You will learn something about your general level of cardiovascular fitness. The findings from this study will help us understand how stress affects our blood pressure, and what personality factors moderate this. At the end of the study, if you wish, we will email you the results of the study.

What are the possible disadvantages and risks of taking part? This study includes questionnaires that measure your feelings and well-being now and in the recent past. You might find while you are answering them that you would like to talk to someone about some of the issues raised. We will be happy to recommend someone to you.

What happens at the end of the study? When all participants have been tested (this
should be within 12 months of your participation), you will receive a summary (no more than 2 pages) of our main findings. While it could be up to 2 years before final results are published, we would be pleased to include you on an address list to receive publications arising from the study.

Confidentiality: All information that is collected about you during the course of the research will be kept strictly confidential and will not be shared with anyone else. You will be assigned a unique identification number, which will not be linked to your personal details. Results from the study will be reported as group data and will not identify you in any way.

What happens if I change my mind during the study? Your participation is voluntary, and you are free to withdraw at any time without giving any reason and without your rights being affected in any way. Once you have taken part in any part of the study, the data we have already collected may still be used. This is because we ensure that there is no identifiable information linked to data collected from you. So, if we have already collected some data, we are not able to remove your data after you leave the laboratory.

What if I have more questions or do not understand something? If you have any questions about the study you may contact the researchers. It is important that you feel that all your questions have been answered. Thank you for taking the time to read this. I would be grateful if you would consider participating in this study.

Contact names and numbers of the principle investigators:

Principal Investigator:
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This research study has received Ethics approval from the Education and Health Sciences Research Ethics Committee (quote approval number). If you have any concerns about this study and wish to contact someone independent you may contact:
Chairman Education and Health Sciences Research Ethics Committee
EHS Faculty Office
University of Limerick
Tel (061) 234101
Appendix F. Sample Participant Consent Form

PARTICIPANT CONSENT FORM

Watching a video of oneself completing a speech task: Effects on blood pressure and heart rate.

Researcher: Ms. Siobhán Griffin
Supervisor: Dr. Siobhán Howard

1. I confirm that I have read the information sheet for the above study and have had the opportunity to ask questions

2. I am satisfied that I understand the information provided

3. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason

4. I understand that I will be watching a video-clip of myself completing the speech-task in a previous study

5. I agree to take part in the above study

Name of Participant __________________ Signature __________________ Date __________

Name of Researcher __________________ Signature __________________ Date __________