DYNAMIC CHANGES IN HEART RATE VARIABILITY UNDER THREAT: EXPLORING THE EFFECTS OF EMOTION REGULATION ON THE PARASYMPATHETIC NERVOUS SYSTEM

BY

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Abstract

How an organism responds to the fluctuating metabolic demands imposed by the environment – that is, self-regulates – is crucial to its success. Several theorists argue that this self-regulation depends on the connection between brain and heart via *cardiac vagal control*. The efficiency/integrity of this brain–heart link is reflected in certain measures of heart-rate variability (HRV). Although trait-like HRV measured under resting conditions is often linked to the ability to flexibly regulate emotions, we are yet to fully understand the dynamic changes in cardiac vagal control that occur *during* emotional challenge (i.e. reactivity and recovery), or the factors that modulate this response. One potential modulating factor is the use of antecedent- and response-focused emotion regulation strategies, i.e. expressive suppression and cognitive reappraisal, respectively. Another is a history of non-suicidal self-injurious behaviour. I conducted three studies analysing continuous HRV recorded before, during and after exposure to a social stressor (Study 1) or a VR plank-walking simulation (Studies 2 and 3). In Study 1, we recorded history of self-injurious behaviour, and in Study 3, we manipulated the use of emotion regulation strategies via explicit instructions. Across studies, findings consistently support a view of vagal withdrawal being a component of the response to emotional challenge. When using individual differences in resting vagal control to predict vagal reactivity, a link was found in Study 1 which points to higher resting levels as a marker for greater vagal withdrawal during social stress (but was not replicated in studies 2 and 3). Self-reported use of emotion regulation strategies during the emotional challenge bore no relationship to vagal reactivity, and neither did a history of self-injurious behaviour. However, relative to the instructed suppression condition, participants in the instructed reappraisal group showed greater vagal withdrawal during the plank-walk. While inconclusive, this thesis points to a role for flexible withdrawal of vagal control in adaptive functioning and provides an important contribution to the scarce literature on dynamic HRV in the context of emotion regulation. The thesis also includes a discussion of theory and the need for more work to explain the complexities of this relationship, as well as crucial methodological factors which future researchers might consider.

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Abbreviations

HRV:	Heart Rate Variability — used here in reference to measures of vagal activity
RMSSD:	Root mean square of successive R-R differences (a time-domain measure)
HF:	Power, in milliseconds^2, of the High-Frequency band of the HRV power spectrum (a frequency-domain measure)
RR:	The length of interval between R-peaks of the ECG trace (aka "heart period")
HR:	Heart rate, expressed in beats per minute (roughly the inverse of RR)
RSA:	Respiratory Sinus Arrhythmia, i.e. HRV which maps onto the respiratory cycle
Suppression:	Expressive Suppression (emotion regulation strategy)
Reappraisal:	Cognitive Reappraisal (emotion regulation strategy)
PFC:	Prefrontal cortex, a collection of anterior cortical forebrain structures
Resting:	Tonic (trait-like) HRV measured under resting conditions
Reactivity:	Phasic change in HRV in response to a stressor (Stressor minus Baseline values)
Recovery:	Phasic change in HRV in recovery from a stressor (recovery minus Stressor values)
TSST:	Trier Social Stress Task (laboratory paradigm)
VR:	Virtual Reality
NSSI:	Non-suicidal self-injury
ERQ:	Emotion Regulation Questionnaire, a 10-item self-report measure with a 1-7 Likert- type scale. Includes suppression and reappraisal subscales.

Dynamic Changes in Heart Rate Variability Under Threat: Exploring the Effects of Emotion Regulation on the Parasympathetic Nervous System

Intuition tells us that the heart is at the centre of our emotions. This sentiment is present in common language: the advice to "follow your heart" for those making important decisions, and exclamations such as "my heart was pounding" or "my heart skipped a beat" when afraid. Heart-centred metaphors are used to describe emotional states both across languages and cultures (e.g. Bas, 2017), and across history. Homer's *Odyssey* contains many such instances (Mumford, 1996), as does the Hebrew Bible and Shakespeare's plays (Goodhart, 2014). The influence of the latter still echoes in modern English – in *Othello* one can find the phrase *"wear your heart on your sleeve".* Finally, more than just a metaphor, Aristotle firmly believed that all sensation, including emotions were fundamentally cardio-centric processes.

And indeed, the heart truly is central in our experience of emotions. Yet, it may be equally central in cognition. Often, the brain is associated with reasoning and thinking capacities, while the heart is held responsible for passions and emotion. In truth, they are more like two sides of the same coin; the heart and brain are dependent on one another in a fascinating and complex manner, and their dynamic interplay gives rise to both thought and emotion. On one hand, the brain *controls* the heartbeat, in order to help the brain and body deal with challenges. On the other hand, the brain *receives* data from the heartbeat, interpreting the data in ways that create emotions. Crucially, mental processes that we use in everyday life to control our thoughts and emotions are thought to be reflected in the way the heart functions.

The brain-heart connection as described above is subserved by a vast network of nerves known as the *autonomic nervous system*. So-called in reference to its (mostly) automatic functioning, the autonomic nervous system liaises between the brain and many

organs in the body. Two opposing forces are exerted through two branches of this system: the sympathetic branch, responsible for preparing the body for action, and the parasympathetic branch, which allows the body to rest and recover. The branches exert their opposite influences over the heart, and their respective influences cause patterns of variability in the heart rate. This is the basis of *heart rate variability* (HRV): a phenomenon which tells us a lot about the link between the brain and heart, and how well it is functioning.

Perhaps counterintuitively, decades of research give an overall impression that the *higher* a person's resting heart rate variability, the better their physical and psychological health (e.g. Young & Benton, 2018). This makes sense, however, because we manage our emotions by calibrating metabolic resources to our needs, in part through flexibly shifting the balance between sympathetic arousal and parasympathetic relaxation (Barrett, 2017). The capacity for this kind of flexible shifting is reflected in HRV. Measuring patterns of heart rate variability under resting conditions (*Resting* HRV) tells us how well someone manages their emotions in daily life – or at least how well they report doing so (Williams et al., 2015; Visted et al., 2017). A similar correlation exists between resting HRV and higher-order cognitive functions, including *cognitive control*; the more heart rate variability a person has the better they are likely to perform on tasks requiring voluntary control and inhibition of mental and physical responses (Forte et al., 2019; Capuana et al., 2014; Colzato & Steenberg, 2017).

The well-established links between resting HRV and both cognitive control and emotion regulation highlight the centrality of a well-integrated brain-body system in cognitive and affective processes. These observations are consistent with emerging perspectives of cognition as a collection of fundamentally embodied phenomena (Thompson, 2010; Depraz & Desmidt, 2019), as opposed to traditional cognitivist views which treat cognitive mechanisms as abstract, representational phenomena.

Although much is known about Resting HRV as a marker of cognitive and emotional health, it is poorly understood how HRV *changes* in response to environmental demands (HRV *Reactivity*). A subset of environmental conditions known to be emotionally-challenging are those which are perceived as threatening, as they signal to an organism the need for action. It is unknown exactly what happens to the brain's control over the heart during conditions of threat. Measuring the dynamics of heart rate variability under conditions of threat will provide a more fine-grained assessment of how the two autonomic branches, and particularly the parasympathetic branch, facilitate adaptive responses.

The relationship between Resting HRV and dynamic changes during threat is also unknown. The general consensus among researchers is that high levels of Resting HRV represent an adaptive capacity in the nervous system which is important for dealing with changing environmental demands and adjusting flexibly. Building on this proposition, an interesting question is whether those with high Resting HRV also have high HRV during threat (and therefore less HRV *Reactivity*, or change), or whether they tend to have a larger drop in HRV during threat. Extant theories that purportedly explain the link between HRV and psychological phenomena are generally underspecified in this regard, and the limited empirical data reveals mixed findings.

It is also thought that HRV reactivity differ between people, depending on certain individual difference variables, such as one's capacity for emotion regulation. *Emotion regulation* often involves the implementation of cognitive and behavioural strategies to reduce the experience of negative emotions. Research has focused on two main types of strategies. It has been theorised that those who use *cognitive reappraisal*, a kind of mental reframing of an event or stimulus, will have more success in controlling their emotions, relative to those who use *expressive suppression* to hide their bodily expressions of the emotion (Gross, 2001). It is also thought that successful use of these strategies relies on

cognitive control mechanisms (Ochsner et al., 2012). In light of the known link between the brain and the heart it would make sense that these two strategies should have observable, and perhaps divergent, effects on the autonomic nervous system during emotional challenge (i.e exposure to threatening contexts). It is therefore important to investigate how the two common strategies modulate the functioning of the autonomic nervous system in the context of threat — analysing HRV Reactivity is a way in which we can do just that.

The aim of this thesis is to further our understanding of dynamic changes in HRV under conditions of emotional challenge. I will address three specific questions. In this thesis, I explore 1) the dynamic changes in HRV that occur under conditions of different types of threat. Importantly, I then use these patterns to test the hypothesis that reactivity of cardiac vagal control is modulated by individual differences in 2) tonic, trait-like levels of cardiac vagal control (Resting HRV) and 3) the use of different emotion regulation strategies (expressive suppression and cognitive reappraisal).

Allostasis

Living organisms are immersed in a constantly changing world. The changes that characterise existence in the world impose demands upon the organism. Metabolic resources, or energy, are invaluable and must be maintained by an organism if it is to survive, reproduce and flourish across time. To navigate environmental demands that are so often in flux, organisms need to respond with the appropriate level of resources. Too little and the demands will not be sufficiently met; too much and precious resources will be wasted. This is, perhaps, the reason central nervous systems evolved at all – to guide the behaviour of an organism through these challenges (Barrett, 2017). To the degree that this guidance is operating effectively, the organism can maintain a balance of energy and a relatively constant internal milieu, known as *homeostasis* (Cannon, 1963). Rather than the organism reacting to changes

to maintain this constant state, it is more efficient to proactively anticipate future demands, and meet them before they arise. The central nervous system maintains homeostasis by predicting upcoming challenges, anticipating the needs associated with them, and coordinating many sub-systems in the body to meet these needs. This active process of maintaining of homeostatic balance is known as *allostasis* (Sterling, 2012). By virtue of such mechanisms, organisms are afforded a wide repertoire of behavioural responses to environmental stimuli and can maintain enough energy to thrive and reproduce. It is argued by authors (e.g. Godfrey-Smith, 1996; Thompson, 2007) that it is in fact this very precariousness, and the associated capacities evolved to preserve it, that is fundamental to both mind and life in organisms. Allostasis is certainly central to the flexibility required to survive in a changing world.

The heart and cardiovascular system play a key role in allostasis. They provide a continuous stream of metabolic resources to the brain and body. With every beat, the blood propelled by the heart transports oxygen, glucose, water, electrolytes, hormones (among other things) to tissues in every part of the body to keep it alive. Its rhythms however are not constant and metronome-like. Rather, the beating of a healthy heart has a dynamic and ever-changing rate, and the interval between each beat differs, ever so slightly, from the preceding interval. Allostatic mechanisms that operate within an organism are reflected in this dynamic variability of the heart rate.

Variability in the heart rate is observed on different time scales. It varies quickly in response to external events: when we see something threatening, the heart rate instantaneously slows down over the course of milliseconds, and if we must somehow deal with the threat, it subsequently speeds up to facilitate action. It also varies as a function of endogenous processes. The heart rate varies substantially with the breath: as we inhale it speeds up, and as we exhale it slows down. On a roughly ten-second scale, it speeds and

slows due to the slower effects of stress hormones. Across a whole day, it ebbs and flows as a function of circadian rhythms, thereby facilitating sleep, rest, and the business of the day.

Modern neuroscience has revealed that the brain and the heart are inseparably coupled, engaged in a continuous interaction which gives rise to feeling states and emotions. Bidirectional connections allow for the brain to alter the speed of the heart rate (consciously and unconsciously) in the services of its own goals – i.e. to fuel the muscles to fight, or even to upregulate the resources the brain itself receives for times of cognitive demand. The heart rate influences the functioning of the brain, as the brain relies on the oxygen and glucose sent to it by the heart. Further still, conscious perceptions of the rate of the heart play a key role in how we feel, because of a phenomenon called *interoception* – our brain's interpretation of changes in the body. When we sense our heart rate increasing, this tells us perhaps we are feeling afraid, and when it is slow, we feel relaxed. Emotions, therefore, emerge partly as a quality generated by the interactions of the heart and brain (Barrett, 2017; Critchley & Garfinkel, 2017). Measuring variability of the heart rate at rest can provide us with useful information regarding the state of the organism as a whole, such as how well it is managing its metabolic resources and how flexibly it can respond to challenges. Perhaps even more usefully, measuring the heart's *response* to such challenges can indicate one's ability to appropriately match these resources to the demands of the environment. In sum, HRV marks the efficiency of the heart-brain link: certainly, a hallmark of allostasis and adaptive human functioning.

The Physiological Response to Threat

Threat and fear are overlapping but distinct phenomena which should be differentiated. Stimuli that potently signal *threat* are those that represent a danger to the survival of an organism — they are phylogenetically relevant. Potent examples include

heights, snakes, and spiders. However, threat stimuli can also be social in nature. Social mammals such as the primates, humans included, depend heavily on conspecifics for survival, and so the integrity of social relationships is crucial. In light of this dependence a socially evaluative context can be highly threatening to humans, and it is unsurprising that public speaking is one of the most common sources of fear in humans. The physical response an organism has to any perceived environmental threat involves a set of synchronised processes in the brain and body, which enable it to respond quickly and adaptively to the threat and increase the probability of survival. This response is generic and non-specific to the stimulus itself – I will refer to this as the *threat response.* It involves behaviours and reflexes that are phylogenetically old and preserved across species.

In the central nervous system, the threat response involves a network of subcortical and cortical regions (Friedman, 2015; Kredlow et al., 2021). The basolateral nucleus of the amygdala processes and recognise incoming sensory data as novel and as a threat. The dorsal anterior cingulate cortex is involved in expressing threat and upregulating resource allocation, and has bidirectional connection with the amygdala. Activation of the central and lateral nuclei of the amygdala, and subsequent activation of the hypothalamic-pituitary-adrenal axis (HPA), initiate a cascade of events: the secretion of hormones such as cortisol and adrenaline from the adrenal glands, and release of monoamine neuromodulators from brain stem nuclei such as norepinephrine (locus coeruleus) dopamine (ventral tegmental area) and serotonin (dorsal raphe nuclei). These events upregulate the metabolic processes necessary to serve the increased needs of the brain and body.

These changes in the central nervous system are accompanied by a response in the autonomic nervous system that is characterised by a shift towards sympathetic dominance, achieved through withdrawal of parasympathetic activity first, and if necessary, upregulation of sympathetic activity. Physiologically, the threat response is reflected in dilated pupils,

sweat produced by the eccrine glands in the skin, and increased heart rate and respiratory rate. Collectively, these changes facilitate a behavioural state known as the "fight, flight or freeze" response.

It is thought that simultaneously to these changes, a response to threat also includes a decrease in activity within circuits in the *prefrontal cortex* (PFC). Activity in the PFC — particularly in the ventromedial portion, by virtue of GABAergic projections to the amygdala — is known to tonically inhibit subcortical activity. The higher the PFC activity, the lower the activity in the amygdala. This tonic inhibition is thought to show phasic withdrawal during threat. Further, dorsolateral regions of prefrontal cortex are involved in cognitive and top-down control, mechanisms that are crucial in guiding adaptive behaviour. Facilitating these functions, ventromedial PFC has bidirectional, continual communication with the basolateral amygdala and the dorsolateral PFC. While the PFC is heavily implicated in the extinction and suppression of threat responses, an acute threat response also has the potential to impair the function of circuits in the prefrontal cortex, because of the deleterious impact of catecholamine signalling — including norepinephrine signalling in the locus coeruleus-amygdala-medial prefrontal cortex pathway (Arnsten, 2009; Iaizzo & Fitzgerald, 2015). Chronically, activation of catecholamine signalling involved in the threat response can cause lasting changes in prefrontal-amygdala inhibition, a mechanism that may underlie difficulties with emotion regulation in psychopathology (Wilber et al., 2011). Evolutionarily, reduced prefrontal activity during threat makes intuitive sense: higher-order cognitive functions are likely to be unnecessary in promoting survival while facing a life-or-death threat. If anything, top-down control may be disadvantageous under such conditions. Prolonged activation of a threat response, particularly a diminished level of tonic prefrontal inhibition, is a proposed explanation for chronic stress and anxiety disorders (e.g. Brosschot et al., 2018), and is reflected in reduced Resting HRV (Thayer & Lane, 2000; 2009).

To summarise, the physiological response to threat consists of a network of activation in subcortical regions (traditionally known as the limbic system), reduced activity in prefrontal cortex, decreased parasympathetic activity, and increased sympathetic activity.

Fear and Emotions

The physiological threat response is therefore a crucial, but automatic and non-specific, evolutionary strategy for survival. On the other hand, emotions are a more complex phenomenon. An emotion is constituted by coordinated patterns of activity occurring across the brain-body system which serve the organism in adaptive functioning in its environment. Viewing a human being as a dynamical system — a complex system characterised by complexity and loosely-coupled sub-systems — an emotion can be understood and measured at several levels of analysis. These levels, or domains, constitute the emotion in a part-whole, or mereological, manner, much as the varied ingredients of a cake give rise to its flavour, an *emergent* quality which is not present in any of the ingredients (de Haan, 2021).

Fear is an example of an emotion, and in keeping with the view described above, can be measured by changes in three domains: physiological (i.e. the threat response), behavioural, and subjective/experiential (Grimshaw, 2018). The combination of changes in physiological systems, behaviours (both internal and external) and the subjective experience, constitute what we describe as fear. Emotions can further be considered a property of the interactions between an organism and its environment which unfolds *across time*. Gross' (1998) process model of emotion regulation describes an emotion-generative process in a temporal sequence (see figure 1, below). In this model the antecedent for an instance of emotion is a situation, broadly defined, which can include external and internal stimuli. This situation is then subject to the individual's perception and attention, and then an appraisal, followed by a behavioural output. For instance, an external situation such as being near a

steep precipice, or an internal situation such as an imagined argument with a friend, reaches the level of conscious awareness via attention. The situation is then appraised in a certain way, by means of cognitive attributions directed at the situation (e.g. “this is terrifying, what if I fall”; or, “Why are they always so misunderstanding?”), which determine the trajectory of the emotion that is generated. Lastly, the emotion is instantiated in a behavioural response, such as backing away from the precipice or perhaps by avoiding social interactions.

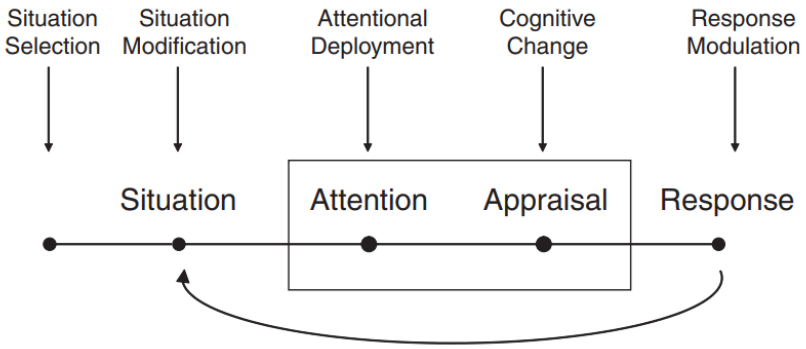

Figure 1: The Process model of emotion regulation (retrieved from Gross, 2014, p.7)

There are great differences between individuals in the intensity of emotional responses to the same stimulus. These differences can be assessed within any of the three domains of emotion. Using self-report measures researchers can ask participants to rate their conscious experience, and thereby index individual differences at the level of conscious awareness. For example, people may differ in their appraisal of the same event due to cognitive processes. Behavioural measures most often involve measuring the observable responses to an event and inferring an underlying emotional process. Physiological changes, however, are the easiest to objectively measure. In particular, measurements of the autonomic nervous system, obtained through electrical recordings of peripheral organs, are non-invasive and easily obtained.

Emotion-Regulation: The Cognitive Control of Emotion

Emotion regulation refers to efforts to alter the experience of emotions. The study of emotion regulation has been a major field of research for several decades (Gross, 1998). While emotions in general are certainly advantageous, the ability to control emotions is often necessary in order to behave in line with our goals. Emotion regulation therefore is a crucial component of human functioning. By the same token, an individual is emotionally *dysregulated* if they exhibit "a pattern of emotional experience and/or expression that interferes with appropriate goal-directed behaviour" (Beauchaine, 2015, p.43).[1] Returning to the Process Model (Gross, 1998), I will discuss one perspective on how volitional cognitive control mechanisms can be leveraged to downregulate negative emotions such as fear.

An important concept in the process model is that emotion regulation strategies can intervene at different points in the sequence of generating an emotion. The first option involves selecting the situations one places themselves in, so as to avoid generating certain emotions altogether. Once in a given situation, one can modify the situation to add or remove certain stimuli. Further, one can control which aspects of the situation they are attending to, either in the external environment (exteroception) or internally (interoception; introspection). However, two specific strategies have received the most attention in research.

The first of these, *cognitive reappraisal* (hereafter shortened to reappraisal), can be implemented at the point where a stimulus has already been perceived and attended to, by altering one's *appraisals* or attributions regarding the stimulus. This strategy involves attempting to mentally reframe the stimulus in such a way that reduces its emotional relevance. Reappraisal is of particular interest as a proposed mechanism underlying so-called

[1] It seems pertinent to note that a substantial degree of between-subject variance in emotion regulation/dysregulation is likely explained by factors entirely outside of conscious awareness. For instance, a view of emotion as psychologically constructed holds that emotion regulation is more about shaping the process of generating an emotion (i.e. interoception, metabolic health, and emotional granularity) than it is about altering one's thoughts or behaviour to change an emotion (Barrett, 2017; Gross & Barrett, 2011) – however, the latter is the focus of interest in this thesis

cognitive restructuring, a central feature of Cognitive Behavioural Therapy (e.g. Beck et al., 2005) and widely used in treating mental health disorders. Reappraisal is generally considered an adaptive strategy for down-regulating negative emotions, belonging to a category known as antecedent-focused strategies. Conversely, *behavioural suppression* (hereafter shortened to *suppression*) occurs at the final stage of generating an emotion and belongs to a category of response-focused strategies. Suppression involves attempting to mask bodily expressions of emotion through controlling one's behaviour. Suppression is associated with the colloquial use of the term stoicism[2] and is thought to be a relatively ineffective strategy for downregulating negative emotions, in part due to its reactive nature (Gross, 2002). The process model holds that reappraisal and suppression have divergent outcomes across the three areas of measurement described above (subjective, behavioural and physiological) in favour of reappraisal. This claim has been borne out in many empirical studies (see Gross, 2015 for review). Therefore, it is predicted that reappraisal is more effective in downregulating negative emotion than suppression, and that suppression is likely to have adverse consequences on physiology.

There are two ways in which these strategies and their effects have been studied. Firstly, individual differences in the habitual use of these strategies are often assessed as a trait, using self-report questionnaires. While several measures have been developed for this purpose, the *Emotion Regulation Questionnaire* (ERQ; Gross & John, 2003) is the most prominent, and is derived specifically from Gross' Process model (1998). The ERQ is a 10-item global assessment of the degree to which people tend to attempt to regulate their emotions in their daily lives using suppression (four items) and reappraisal (six items). The measure produces a separate score for each strategy. A large body of research finds that individuals who score highly on the reappraisal subscale enjoy better outcomes, such as

[2] Distinct from the ancient philosophy of *Stoicism*.

higher self-reported positive emotion and lower psychopathology, relative to those who use suppression more frequently (John & Eng, 2014; Hu et al., 2014). Studies have found that forms of suppression are associated with cardiovascular disease (Appleton et al., 2014; Bahremand et al., 2016) and systemic inflammation (Williams et al., 2019), suggesting that it may have deleterious long-term effects mediated by the autonomic nervous system.

The efficacy of suppression and reappraisal has also been compared in laboratory experiments, whereby participants are instructed to use reappraisal or suppression during an experimental emotion induction. Emotion inductions often consist of emotional images (e.g. IAPS), emotional video clips, interpersonal interactions (e.g. the Trier Social Stress paradigm), and rumination/worry manipulations. Gross (1998) first tested for differences in the effects of these two strategies by showing a disgust-inducing film to 120 participants. While both strategies led to a decrease in expressions of disgust relative to a control group, reappraisal led to less self-reported feelings of disgust, and suppression did not. Interestingly, participants in the suppression group also showed increased sympathetic arousal on certain measures (pulse amplitude, skin conductance, and skin temperature). The latter observation suggests that suppression use may actually have adverse effects on the body, conferred via enhanced sympathetic activity.

Webb et al**.** (2012) conducted a meta-analysis of such laboratory experiments. It was found that both suppression and reappraisal seem to be effective in decreasing negative emotion, as measured by a combination of subjective, behavioural, and physiological domains (effect sizes $d_+ = 0.32$ & $d_+ = 0.36$, respectively). When focusing solely on physiological outcomes however, the results are less clear; in a more recent meta-analysis of 68 studies (Zaehringer et al., 2020), mixed results were found regarding (1) whether either strategy is effective, relative to uninstructed control conditions, or (2) whether one strategy is more effective than the other. This uncertainty is reflected in a large heterogeneity of effects

(both direction and magnitude) found in the included studies. Zaehringer and colleagues highlight that this heterogeneity can be attributed to the wide range of methods employed in experiments, and the predominance of inductions of mild-to-moderate emotional states. In the absence of a strong emotional response being induced in participants, it may be difficult to capture subtle effects of emotion regulation on physiological measures. In the Zaehringer (2020) meta-analysis, a small but significant effect was revealed whereby reappraisal decreased heart rate, whereas suppression showed no overall effect on any measure. So, while it is clear that trait individual differences in suppression and reappraisal use predict psychological and physical outcomes, laboratory experiments have failed to find convincing evidence for their effects on the autonomic nervous system. It appears that more work is needed to test the effects of these strategies on the autonomic nervous system, using more sensitive measures such as HRV.

To establish a hypothesis regarding the effects of reappraisal and suppression on HRV, an examination of the involved cognitive mechanisms is required. *Cognitive control* refers to a collection of higher-order cognitive functions which involve top-down control of other cognitive and behavioural processes. Encompassed by the category of "executive functions", cognitive control includes the volitional control of thought and bodily expression — a capacity that is supported by the PFC and its connectivity to subcortical and parietal regions. A review by Ochsner and colleagues (2012) concluded that both suppression and reappraisal are subserved by cognitive control. The authors proposed a *Model for the Cognitive Control of Emotion* that includes the following components responsible for implementing cognitive control strategies: the ventrolateral PFC (selecting or inhibiting responses), dorsolateral PFC (selective attention and working memory), dorsal anterior cingulate cortex (conflict monitoring) and dorsomedial PFC (attributing emotional states in self or others). These exert control over the ventromedial PFC/medial orbitofrontal cortex

(value of stimulus in context), insula cortex (interoception), ventral striatum (encoding reward value) and the amygdala (arousal/threat value of stimuli) — which collectively are involved generating emotional states.

Functionally, the medial PFC is known to support the processing of emotionally relevant stimuli (Diekhof et al., 2011) and has structural connections to the amygdala (Devinsky et al., 1995). Indeed, activity in the PFC and amygdala is often negatively correlated (e.g. Davidson, 2000), particularly during regulation of negative emotion (Urry et al., 2006; Wager et al., 2008). In one fMRI study (Goldin et al., 2008), a within-subjects design was used to test the effects of suppression and reappraisal against spontaneous regulation while watching negative-emotion inducing video clips. Both strategies strongly activated the PFC more than using no strategy and decreased subjective negative emotion and behaviour. However, the time course of neural activation differed, such that reappraisal led to early PFC responses and decreased amygdala and insula responses, whereas suppression led to late PFC responses and an *increase* in amygdala and insula response. Butler et al. (2019) conducted a very similar experiment using fMRI, which found that reappraisal led to greater medial PFC activation during negative emotion than suppression or control.

Converging evidence from these studies, and basic research on the structural connections between these regions, points to a common pathway by which cognitive control mechanisms are leveraged in the service of volitional emotion regulation. This pathway consists of PFC regions involved in the processing of emotionally-relevant information held in working memory, which then lead to downregulation of responding in the amygdala and insula (or ventral striatum), probably via medial PFC and orbitofrontal cortex.

Cardiac Vagal Control

I now return to HRV for a summary of the mechanisms underlying short-term HRV, the kind that is usually studied in relation to emotion. The *vagus nerve* is the Xth cranial nerve and is known to relay information bidirectionally between the central nervous system and the visceral organs. The name "Vagus" comes from the latin for "wandering"; true to its name, the vagus originates in the medulla oblongata of the brain stem and innervates much of the upper body via its multiple branches. Notably, myelinated portions of the vagus terminate at the sinoatrial (or sinus) node of the heart. The vagus is famed for its role in interoception — 70-80% of the nerve fibers in the cervical portion of the vagus are primary afferent axons, sensing inflammatory mediators and other processes (Zanos et al., 2018). Here I focus on vagal efferent fibers and their role in neural control of cardiac functions.

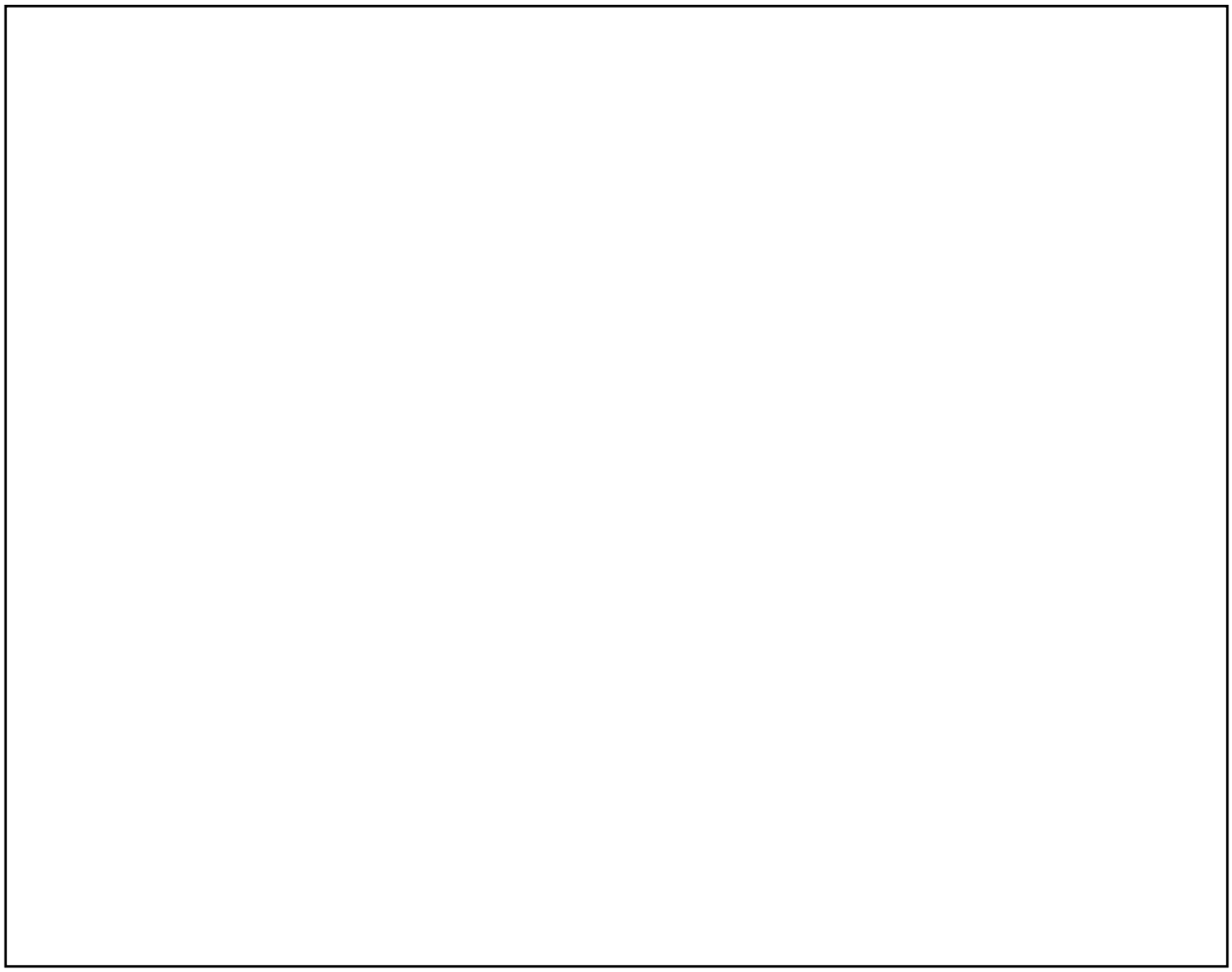

Figure 2: the vagus nerve (left) and QRS complex in ECG trace (right)

A sketch depicting the "wandering" or multi-branched vagus nerve (from Karemaker, 2022); The electrocardiogram (ECG) trace, depicting the QRS complex, P, and T waves of sinus rhythm, as well as the systole and diastole components of blood pressure oscillations with each beat.

The healthy heart rhythm (for review: Berntson et al., 2017), known as sinus rhythm, is primarily controlled by the spontaneous depolarisation of the sinoatrial node, a bundle of nerve fibres in the upper-left atria. Action potentials generated by this intrinsic firing travel through the atria, and into the ventricles, resulting in contractions of the left and then right chambers of the heart. This process is represented in the electrocardiogram as the "QRS complex" (see figure above). Following this contraction, repolarisation occurs, represented by the T-wave. This cyclic pattern of contraction and refilling gives rise to an associated blood pressure oscillation, with two phases: systole is the contraction phase in which blood is expelled from the chambers of the heart, and diastole is the phase in which the chambers are refilling.

The heart (specifically the sinoatrial node) is under dual innervation of the two autonomic branches: the sympathetic and parasympathetic systems. Therefore, the heart's rate is governed by the combination of intrinsic firing at the sinoatrial node (a steady rate) *and* the complex interplay of both autonomic influences. The sympathetic input originates in the stellate ganglion in the spinal cord and is conferred via adrenaline, which agonizes beta-adrenergic receptors (thereby depolarizing sinoatrial cells). Sympathetic activity is also conferred via smooth muscles surrounding the vasculature, by decreasing the size of arteries to create resistance and compressing the volume of blood in veins to fill the heart more — thereby increasing stroke volume. Through both mechanisms, sympathetic activity is excitatory and *increases* heart rate. Conversely, the parasympathetic system confers an inhibitory effect via the vagus nerve, through acetylcholinergic transmission. Vagal activity polarizes sinoatrial cells, thereby *slowing* the heart rate. This inhibitory parasympathetic effect on heart rate is referred to by some authors as the *vagal brake* (Porges et al., 1995), a

useful metaphor to convey how the heart rate is quickly adjusted — by applying and withdrawing what I will term hereafter *cardiac vagal control*. At rest, vagal influences on the heart rate prevail; it has been demonstrated that when the vagus nerve is severed or pharmacologically blocked, heart rate rises to around 140 beats per minute, but under vagal influence it is slowed to around ~75bpm at rest.

A major source of HRV is the phenomenon known as the baroreceptor reflex (or baroreflex), a negative feedback system that maintains blood pressure homeostasis. The baroreflex is present in all vertebrates, probably with origins as a fine-tuning mechanism to protect the functioning of the gills, which require a narrow range of blood pressure (Karemaker, 2022). Mammals also require blood pressure to remain within a range of values to adequately transport metabolic resources, particularly to the brain, and also to remain low enough to not cause excess damage to vasculature. Blood pressure represents the combined influences of pulse pressure (cardiac output), blood volume, and vascular resistance (size of blood vessels). In systole, pressure in blood vessels increases as blood is expelled from the heart. Specialised mechanoreceptors in the carotid artery and the aortic arch called baroreceptors fire when the blood vessels stretch. Signals from baroreceptors first reach cardiac vagal motor neurons within the ventrolateral medulla oblongata, specifically in the nucleus ambiguus, nucleus of the solitary tract and the dorsal motor nucleus of the vagus. Here, action potentials initiate vagal efferent activity which very quickly slow the heart rate so as to normalise blood pressure. Simultaneously, sympathetic nuclei in the brain stem are inhibited, minimizing sympathetic influence on the sinoatrial node. This baroreceptor afferent activity is quickly silenced during diastole — blood pressure drops as blood refills the heart, vagal efferent activity reduces, and sympathetic outflow from the brain stem is released.

So, all else remaining constant, a constant blood pressure level and pulse pressure at each heartbeat would give rise to an unchanging inter-beat interval and no HRV. In actuality,

the oscillating frequency of afferent action potentials from the baroreceptors leads to variable vagal efferent activity, and arrhythmic inter-beat intervals. Furthermore, how the brain stem regions translate this incoming baroreceptor data is determined by factors "higher" in the central nervous system[3], including circuits involved in emotion and cognitive control (Thayer & Lane, 2009). Also, note that sympathetic activity is released when baroreceptors stop firing, but this alone does not determine the magnitude of sympathetic influence. Rather, the magnitude depends on other factors such as chemoreceptors and psychological phenomena.

Because the vagus nerve (being myelinated) exerts its effects on heart rate so quickly, on the order of milliseconds, the variation in inter-beat intervals that occurs from one beat to the next (i.e., high frequency variation) is understood to quantify the rate of vagal firing (i.e. cardiac vagal control).[4] Because sympathetic activity has a delayed influence on the heart rate, a low frequency variation in heart rate at the rate of around 10-second cycles (0.1hz) is often attributed to sympathetic sources. This pattern maps onto the blood pressure oscillations known as Mayer waves and is thought to reflect vasomotor function[5]. However, attributing low-frequency variability solely to sympathetic sources is controversial, and some evidence suggests it may be instead related to vagal activity (Kromenacker et al., 2018) possibly the unmyelinated vagus nerve (Porges, 2007).

The heat rate variability mechanism described above, determined by the baroreflex, is exaggerated by respiratory mechanics. Even at rest, cardiac vagal control is not constant but is intimately coupled to the respiratory cycle. Upon inhalation, the diaphragm contracts and

[3] I discuss the effect of other brain regions on vagal activity when I describe the Neurovsiceral Integration Model in the following section

[4] Note that because the vagus nerve has many fibers and branches, it is unclear whether it is accurate to assume that "vagal tone", as measured by HRV, generalizes throughout the body as is often assumed, or if it is specific to the activity of cardiac vagal axons.

[5] Vasomotor refers to the action of the smooth musculature surrounding blood vessels which are controlled by the sympathetic nervous system

descends into the abdomen, increasing the thoracic volume and decreasing the blood pressure (due to greater space for blood to flow through). During exhalation, the diaphragm relaxes and rises again, and the associated increase in blood pressure elicits baroreceptor firing and compensatory slowing of the heart through increased vagal control. This influence of breathing on heart rate via the vagus nerve is known as respiratory sinus arrhythmia (RSA). Due to this respiratory gating of vagal transmission and therefore heart rate, certain statistical measures which capture instantaneous and respiration-coupled variability in heart rate are known to index an individual's level of cardiac vagal control. These are the measures I refer to as HRV, and their relationship to psychological phenomena will be discussed below.

The Heart of Emotion Regulation: Theories from Psychology

Several theoretical frameworks have been developed by psychologists which seek to explain the phenomenon whereby HRV is associated with emotion regulation (indicated by psychopathology, questionnaire measures, and laboratory-induced emotional reactivity). In these theories, researchers causally link cardiac vagal control to emotion regulation, based on the existence of certain neural networks and features of the vagus nerve. Here I will briefly review the four theories that are most prominent, and relevant to generating my predictions.

Polyvagal Theory. The Polyvagal theory (Porges, 2018) takes an evolutionary approach to explaining the role of cardiac vagal control in emotion, emphasizing the role of the vagus nerve in regulating social behaviours. As the name "Polyvagal" implies, this theory is grounded in physiological evidence that there are subdivisions of the vagus nerve which differ in their phylogenetic recency and roles. The dorsal vagal complex was evolved first and is present in lower phylum such as reptiles and is conserved in humans. It is unmyelinated and thus exerts slow, tonic inhibitory influences over the heart to allow for freezing behaviours in response to threat. Conversely, a more phylogenetically recent ventral

vagus complex is claimed to be present only in mammals; this myelinated vagus allows for fast acting influences over the heart and causes the resulting high-frequency HRV.

A core concept is the idea that this ventral vagal complex allows humans to rapidly inhibit, or disinhibit, autonomic activity in the periphery in order to behave adaptively. Other autonomic influences such as sympathetic activity are therefore only recruited when the withdrawal of the ventral vagus is insufficient to meet predicted metabolic needs. This autonomic flexibility is key in adaptively responding to fluctuating environmental demands, particularly in the complex social environment in which humans live. The constantly changing metabolic demands of behaviours such as nonverbal communication, emotional expression, sexual courting, and verbal communication, require this sensitive adjustment capacity. Further, a key proposition is that the ventral vagus complex has neuroanatomical connections to other cranial nerves which control the muscles of the face involved in communication. Finally, it is thought that HRV captures the activity of this fast-acting branch of the vagus. In this model, HRV measures the capacity of the organism to quickly engage and disengage the inhibitory vagal brake and therefore respond adaptively to environmental demands. In contrast to the other theories, interestingly, Porges and colleagues posit that a greater magnitude decrease in HRV ins response to a threat (Reactivity) is adaptive.

Neurovisceral Integration Model. The Neurovisceral Integration model (Thayer & Lane 2000; Thayer & Lane, 2009) links cardiac vagal control to emotion regulation and cognitive function. This account incorporates a dynamical systems approach to explain the link between emotions, cognitive control and emotion regulation with HRV. Thayer and colleagues posit that vertically organised components of the central nervous system, peripheral nervous system and the visceral organs comprise a dynamic system. Within this system, two features are emphasized. Firstly, the *central autonomic network* (Benarroch, 1993) is a network of brain nuclei in which the generation of emotional responses, self-

regulatory control of behaviour, and control of the autonomic nervous system are co-localised. Second, negative feedback mechanisms including the baroreflex are crucial in regulating emotional states through inhibition.

The central autonomic network consists of structures in the forebrain (ventromedial prefrontal cortex, anterior cingulate cortex, insular cortex, central nucleus of the amygdala,), hypothalamic structures (lateral hypothalamus and paraventricular nucleus), rostral brain stem (periaqueductal gray, nucleus ambiguous), and lower brain stem (nucleus of the solitary tract). The key claim is that this network coordinates autonomic output to shape somatic reactions to emotionally relevant events. One important premise in the theory is that the PFC exerts top-down inhibition of the subcortical components. Ultimately, because the PFC has both direct and indirect influences on brainstem (autonomic control) nuclei its activity is directly reflected in cardiac vagal control. This link between PFC activity and efferent vagal activity is central to the Neurovisceral Integration explanation for observed relationships between emotion regulation (and related phenomena) and Resting HRV.

Several lines of empirical evidence substantiated the above claim. Firstly, pharmacological deactivation of the frontal cortices[6] is shown to elicit immediate increases in heart rate and a decrease in HRV, an effect that was faster and of greater magnitude in participants with right hemisphere inactivation relative to those with left hemispheric inactivation (Ahern et al., 2001). Convergent observations when researchers compared patients who experienced right-hemispheric damage from strokes, to those with damage in the left hemisphere: the former group had much higher rates of clinically significant tachycardia symptoms (Lane et al., 1992). Manipulations of PFC activity using transcranial

[6] This deactivation is initiated by the infusion of sodium amobarbital, a sedative agent, into the introcarotid artery. This procedure (known as the Wada test) is used to deactivate individual hemispheres of the brain during surgical procedures to assess the risk of causing significant cognitive impairment in neurosurgery.

magnetic stimulation have yielded similar results. Specifically, excitatory pulses to the dorsolateral PFC were shown to increase HRV in a dose-dependent manner (Iseger et al., 2021), while elsewhere, inhibitory pulses to the same region decreased HRV (Era et al., 2021). Further, a meta-analysis of fMRI studies examined the relationship between neural activity and HRV during emotional states, and cognitive and motor tasks (Thayer et al., 2012). Overall results found that medial PFC was strongly and consistently associated with HRV across all three types of tasks. Additionally, during emotional states, the lateral amygdala was *inversely* associated with HRV. In other studies with both young and older participants, a positive, linear relationship was found between resting HRV and PFC – amygdala functional connectivity (Sakaki et al., 2016). Finally, in a randomised–controlled trial where slow, rhythmic breathing was given to one group as an intervention, pre–post increases in HRV were observed. These improvements were accompanied by significant increases in resting-state functional connectivity between the medial prefrontal cortex and amygdala in the left-hemisphere (Nashiro et al., 2021). Taken together, these findings all point to HRV as a measure of the output of the central autonomic network — and particularly the integrity of PFC top-down influences.

Generalised Unsafety Theory of Stress. Brosschot and colleagues (2018) extend on the Neurovisceral Integration account. The authors claim that the threat response, rather than being an anomalous process that occurs only occasionally — in response to threat — is always in fact potentially active. In this view, the threat response is the *default* response when cues of safety are absent. A threatening context is therefore characterised by the lack of perceived safety, rather than the presence of threat. Instances of perceived unsafety elicit a withdrawal of vagal activity, which represents the release of a system that inhibits the threat response. As in other theories, HRV provides an insight into this system's integrity. Brosschot et al (2018) predict that momentary contextual shifts in an individual's perceptions

of unsafety elicit phasic HRV decreases. In other words, perceived *unsafety* is associated with phasic decreases in HRV, due to a disinhibition of the threat response mediated by the PFC becoming decoupled from the amygdala. A corollary of this view (as in the previous theory) is that higher phasic HRV during threat (or a smaller magnitude HRV decrease), would indicate greater perceived safety and more effective emotion regulation. Actively regulating emotions (e.g. reappraising a situation as safe) should be associated with smaller phasic HRV decreases during threat.

A Biomarker for Psychopathology. HRV has also been directly implicated in mental disorders. The research domain criteria (RDoC) approach to psychopathology research (Cuthbert & Insel, 2013) seeks to link brain-behaviour mechanisms to clinical phenomena, and has a focus on cross-cutting mechanisms (i.e. those that play a causal role across diagnostic categories). In line with this philosophy, one of RDOC's specified research goals is to delineate endophenotypes. In psychiatric genetics, an endophenotype explains behavioural symptoms in terms of underlying mechanisms that have a heritable, genetic basis. An implication of an endophenotype is that it should have a measurable, typically biological, signature that differentiates those with the endophenotype from those who do not. This measurable signature is often called a *transdiagnostic biomarker,* in that it is a bodily signature that represents the vulnerability to general psychopathology conferred by the endophenotype.

Several mental disorders that are characterised by impaired emotion regulation have a well-established association with HRV. For instance, individuals diagnosed with major depressive disorder, general anxiety disorder, panic disorder, phobia, and substance use disorder have all been demonstrated to exhibit lower resting HRV relative to control participants. These observations have led to claims that reduced cardiac vagal control is a central phenomenon in psychopathology — or an endophenotype — and that significantly

low resting HRV is an associated biomarker (Beauchaine & Thayer, 2015). A recent meta-analysis examined relationships between resting HRV and major depression (Koch et al., 2019). It was found, across 21 studies, that reductions in RMSSD and HF-HRV were found in MDD patients relative to controls. This link has been corroborated in other reviews (Kemp et al., 2012), while lower HRV may be associated with more severe depressive symptoms (Sgoifo et al., 2015). Similar findings in anxiety have been reported in reviews (Paniccia et al., 2017) and meta-analyses (Chalmers et al., 2014). Finally, HRV has been shown to have a strong genetic basis, with heritability estimates in twin studies ranging from 47-64% (Golosheykin et al., 2017). Taken together, it seems that reduced resting HRV is linked to impaired emotion regulation, however, it is uncertain whether this association holds in HRV *Reactivity*. Beachaine (2015) proposes that over-reactivity of HRV during emotional challenge (greater phasic decreases) is indicative of emotion dysregulation. A meta-analysis of 37 studies (total n = 2, 347) found no evidence for a main effect of psychopathology diagnosis on HRV reactivity, though a relationship emerged whereby externalising (but not internalising) disorders showed greater HRV reactivity (larger decreases).

Vagal Tank Theory. Lastly, Laborde and colleagues (2018) attempt to integrate explanations of HRV with the self-control literature. Vagal Tank Theory is especially useful due to its explicit differentiation of tonic and phasic HRV constructs, which will be discussed in the following section. The core of the Vagal Tank theory is the claim that HRV, to the degree that it captures vagal activity, represents the efficiency with which an individual can deploy *self-regulation resources*. Self-regulation is broadly construed, in reference to effortful and deliberate control of behaviour, thought and emotion. The use of the term "resources" is intentional — self-regulation is thought to map onto social and cognitive psychology theories of self-control[7] (Baumeister et al., 1998; Baumeister et al., 2018; Kotabe

[7] This tank analogy is akin to the well-known concept of ego-depletion, from social psychology.

& Hofman, 2015) in that self-regulation is viewed as a resource that may be both depleted with use and replenished, as it were, with rest/recovery. Hence the analogy to a "tank". Laborde et al (2018) propose HRV as an indicator of the current state of the tank and therefore the capacity of self-regulation. Importantly, this theory is the only one to make concrete predictions regarding HRV Reactivity and emotion regulation (outlined below).

HRV Paradigms: Resting, Reactivity, and Recovery

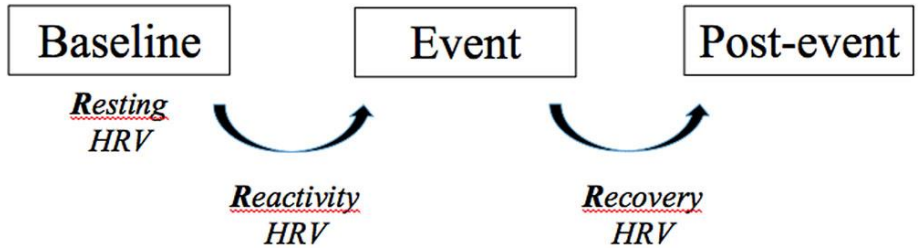

Figure 3: the 3-R approach to studying HRV (retrieved from Laborde et al., 2018)
Resting HRV as a measurement taken from a Baseline epoch, and HRV Reactivity/Recovery as change measures between Baseline-Event, and Event-Recovery epochs

Laborde and colleagues (2018) recommend the "3 R's" of HRV research: Resting, Reactivity, and Recovery. In doing so the authors highlight an asymmetry in the literature — while there has been a lot of research on resting HRV, the other aspects and their relationships have been under-explored. A prevalence of research uses *resting* HRV measurements as a trait-like individual differences, between-subjects variable. Resting HRV is widely used to assess *tonic* vagal activity, a relatively stable pattern of vagal activity (likened to a personality trait; a resource; and a biomarker; an index of PFC activity). This method involves taking recordings of cardiac activity under resting conditions, either in supine or seated positions. Participants may be asked to breathe as they naturally would or instructed to follow paced breathing instructions to control for respiratory influences. Additionally, studies are variable in whether pure resting conditions are used or whether a "vanilla" baseline is used, involving a cognitive task which requires minimal mental

resources/effort. These are often used in clinical populations to avoid rumination or anxiety over what the participant should be doing. A less commonly studied element of HRV is the change associated with an event, such as an emotional challenge. This method involves studying the within-subject changes in HRV that occur, such as change from baseline to a stressor. I will refer to this as HRV *reactivity*[8]. A third way in which HRV is studied examines cardiovascular recovery from a stressor, i.e. the change in HRV that occurs following an emotion induction, when resting conditions are restored. I will refer to this as HRV *recovery.*

According to the Vagal Tank theory, the direction and magnitude of HRV reactivity in response to an event is related to adaptivity, which is in turn derived from the demands imposed by the environment. Therefore, it is necessary to determine (1) whether high levels of metabolic resources are required to face a situation and (2) to what degree top-down prefrontal activity is required. It follows that when faced with a context that demands action, a large withdrawal of cardiac vagal control is adaptive. Conversely, when the context requires a high degree of executive functioning/top-down control, a smaller decrease or an increase is adaptive. The predictions regarding HRV *recovery* are more clear-cut. In situations where HRV reactivity consisted of a decrease in HRV, an adaptive response is for HRV to increase to (or above) baseline levels following the event. This would indicate that the individual has the means to restore cardiac vagal control and fully recover. If there is an increase in HRV during the event, the adaptive response would be for HRV to remain elevated during recovery.

A Review of Empirical Data

[8] Some authors also refer to the resting-reactivity distinction as tonic and phasic. I choose to use the "3 R's" for clarity.

Several studies have shown that HRV reactivity during the induction of negative emotion consists of a reduction in HRV, indicating withdrawal of vagal activity, and a subsequent increase in HRV during recovery. This has been shown in several studies. For instance, Beauchaine (2001). This pattern is replicated in other work (Beauchaine et al., 2007; El-Sheikh et al., 2011; Schwerdtfeger & Derakshan, 2010; Berna et al., 2014). Additionally, experimental inductions of worry have shown similar decreases in HRV (Thayer et al., 1996; Lyonfields et al., 1995). The meta-analysis by Beauchaine (2015) investigating HRV reactivity and psychopathology found that there was large heterogeneity in findings between studies but found evidence that the variability may be explained by several methodological factors. The following factors showed larger effect sizes of HRV reactivity (larger phasic HRV decreases): negative emotion inductions versus positive emotion/physical stress/cognitive tasks (stimulus type), women versus men (gender), higher ECG sampling rates versus lower rates (data quality), and vanilla versus stimulus-free baselines (baseline type).

Some studies have found that the degree of HRV recovery is influenced by individual differences variables. For instance, Berna et al (2014) showed that both participants, split into those with low and high levels of self-reported trait emotion regulation difficulties, exhibit decreases in HRV during emotional challenge. However, in recovery, the group with low emotion regulation difficulties showed an increase in HRV, whereas the group withhigh difficulties did not recover to baseline levels. Weber et al. (2010) found that lower resting HRV was associated with a delayed HRV recovery after stress, suggesting that HRV recovery may depend on resting HRV levels.

Perhaps, the least understood phenomenon in HRV research in psychology is the relationship or interaction between the three Rs. Laborde (2018) predicts that because high resting HRV is known to represent an adaptive capacity for emotion regulation, high levels

should be associated with a more adaptive pattern of reactivity and recovery. Some studies **(**Beauchaine, 2001; Beauchaine et al., 2007; El-Sheikh et al., 2011**)** show a negative association whereby higher resting levels of HRV are associated with greater decreases during emotional challenge. Park et al. (2014) is one of the few studies to explicitly test this relationship. Participants in this study were tasked with identifying a target letter among letter strings which were superimposed on faces, which were manipulated to be either fearful or neutral. A median split was performed based on resting HRV into low and high HRV groups. Results showed that for the high HRV group, HRV reactivity comprised an increase (as opposed to the decrease typically reported) during fearful faces relative to neutral faces, whereas no change was observed in the low HRV group. The authors interpret the phasic HRV increase as a reflection of emotion regulation processes in the group with high resting HRV, a capacity which may be impaired in the lower HRV group and evidenced by their lack of phasic HRV increase.

Another outstanding question is how HRV is modulated by emotion regulation strategy use. A meta-analysis of 123 studies (total n = 14,347) tested the relationship between HRV and top-down self-regulation (Holzman & Bridgett, 2017). HRV included resting and reactivity measurements, while self-regulation included measures of executive function, emotion regulation and effortful control. In support of the theories described above, a small but significant effect was found (r = 0.09) indicating that higher HRV was associated with better top-down self-regulation. Another smaller meta-analysis of 24 studies found a small, significant correlation between resting HRV and a broad range of self-control tasks (Zahn et al., 2016). However, these studies include a wide range of cognitive tasks, and they do not directly address how strategy use alters HRV reactivity.

Here I will review the handful of studies which have tested this question directly. Jentsch and Wolf (2020) induced a threat response in participants using the Trier Social

Stress Test and assigned them to either suppression or reappraisal instruction conditions. Results showed that the reappraisal group exhibited more pronounced decreases in HRV during the stressor, relative to those in the suppression group, but not compared to the control group. The magnitude of this effect was moderated by the level of trait reappraisal reported by participants on the ERQ. Another study (Butler et al., 2006) used dyadic interactions in which pairs of participants discussed a distressing film they viewed, with content focused on the Hiroshima nuclear bombing. Use of both strategies were associated with higher HRV during the conversation relative to uninstructed controls, though the reappraisal group showed a more pronounced effect, and reactivity was not assessed as a change score. Denson et al. (2011) elicited a state of anger in participants, who watched brief video clips of an actor (purportedly a fellow student) discussing a politically inflammatory topic (either immigration, university fees, or climate change) that was selected to be opposite to the participant's personal views. Participants were assigned to either a reappraisal, suppression or control condition and given instructions accordingly. Reappraisal increased HRV relative to suppression and control conditions. A study using a public speech as an emotional challenge (Nasso et al., 2018) found that engaging in reappraisal, but not rumination, in the lead-up to the speech increased HRV throughout the challenge, but only for participants low in trait rumination. More support for the effect of reappraisal on increasing phasic HRV comes from a different methodology: Schwerdtfeger et al. (2019) used an ecological momentary assessment paradigm, and found that the use of reappraisal was positively associated with HRV during the day, but only in participants who scored highly on a test of heartbeat-detection accuracy (a measure of interoceptive accuracy).

One study (Di Simplicio et al., 2012) showed limited evidence that use of suppression increased HRV, but only in low-neuroticism participants. 33 participants, split on trait neuroticism (13 high & 20 low) viewed negative emotional images and were asked to either

passively view or attempt to emotionally regulate. In the low neuroticism group, emotion regulation via suppression increased HRV relative to passive exposure. This effect was small and limited to the low neuroticism group, as the high neuroticism showed no relationship between suppression and HRV.

Table 1: Summary of relevant empirical studies
Studies that measure phasic HRV and use of emotion regulation strategies.

Author/s	Year	Sample	Method	Finding/s
Butler et al.,	2006	Healthy controls	RCT, instructed sup. & reapp. During distressing film	Both strategies ^ HRV, more so in reapp.
Denson et al.,	2011	Healthy controls	Instructed Supp. Or Reapp. Or control During anger-inducing clip	Reapp. ^ HRV compared to Supp. And Control
Di Simplicio et al.,	2012	HC, median-split on trait neuroticism	Told to regulate, or to passively watch, while viewing negative emotional images	Supp. ^ HRV, but only in low neuroticism participants
Nasso et al.,	2018	HC, median-split on trait rumination	Reapp. Or catastrophizing prior to giving public speech	Reapp. ^ HRV, but only for low ruminators
Schwerdtfeger et al.,	2019	HC, split on interoceptive accuracy (heartbeat detection)	Ecological momentary assessment	Reapp. ^ HRV during day, only in high interoceptive accuracy group
Volokhov & Demaree	2010	HC	Positive and negative film clips, post-hoc ERQ admin	Reapp ^ HRV, but not during neg. condition, only pos. condition
Jentsch & Wolf	2020	HC	Reapp. Vs Supp. & Control groups	Reapp. -> more Reactivity/Recovery than Supp, moderated by trait levels of Reapp.
		Other Studies		
Ingjaldsson et al.,	2003	Abstinent alcoholics	Cue exposure	^ HRV while exposed to cues for alcohol
Garland et al.,	2012	Abstinent alcoholics	Cue exposure	^ HRV during cue-exposure predicted relapse (indicates need to regulate)

Segerstrom & Nes,	2007	HC	Gave participants a choice of palatable vs unpalatable food, then gave them difficult anagram, tasks to solve	^ HRV while exerting self-control predicted persistence on anagram task
Jamieson et al.,	2012 /2013	HC	Arousal reappraisal vs attention reorientation and control	Reapp. of arousal -> greater cardiac output & less vascular resistance

A handful of other studies have indirectly addressed the question of how emotion regulation affects phasic HRV. These studies, from the addiction and self-control literature, support the notion that successful regulation of emotions increases phasic HRV (Ingjaldsson et al., 2003; Garland et al., 2012; Segerstrom & Nes, 2007; Gesler et al., 2016). To summarise, some evidence is present for the hypothesis that emotion regulation increases HRV during an emotional challenge. However, few have tested this idea using ecologically valid paradigms, few use reactivity/change measures of HRV, and none (to my knowledge) have examined this relationship during paradigms that elicit a state of fear.

HRV Measures and Methods

Data Acquisition

In this thesis, several cardiac variables will be analysed. Calculating all of these variables requires the cardiac time series*:* the continuous sequence of R-R intervals representing the time elapsed between two successive R peaks. This series of R-R intervals is subjected to one of several techniques to quantify the variability within it (HRV). The R-R series is often averaged to quantify the mean R-R interval in milliseconds, known as heart period. A nonlinear transformation[9] is used to convert this mean R-R interval to heart rate in beats per minute, known as HR (Goldberger et al., 2014). The quality of this initial RR data is

[9] HR x RR interval = 60,000; these two measures of rate are not interchangeable, due to their non-linear relationship at certain rates. See correlation tables in Appendices A-C.

therefore crucial and depends on the data collection method. The highest-quality recording method is electrocardiography (ECG), which involves recording the electrical activity of the heart via passive electrodes (usually placed on the ribcage). While a 12-lead ECG is the gold-standard in medical research, a more convenient 3-lead version may be used with good results. A sampling rate of at least 256hz has been recommended for HRV measurement (Berntson et al., 1997; Malik., 1996). One caveat is noted: a meta-analysis of phasic HRV found that studies using higher sampling rates (>1000hz) were more likely to find significant results of phasic HRV changes during emotion (Beauchaine et al., 2019). While traditional ECG with high sampling rates is ideal, it presents limitations for certain experiments due to the constraints it imposes on movement for the participant. To address this concern, other methods have been developed for cardiac measurement under ambulatory (freely moving) conditions. Photoplethysmography (PPG) is commonly used in wearable consumer technology such as smart watches. However, as this measures changes in light reflected from the skin in the periphery, it is subject to influences other than the heart beat (blood pressure etc.), does not capture R peaks due to a lower sampling rate, and is not suitable for HRV assessment. Recent innovations in ambulatory technologies have seen the development of technologies such as the Equivital LifeMonitor (ADInstruments). This product consists of a vest which has electrodes woven into the fabric such that it records similarly to a 3 lead ECG. It records data to a portable module and is capable of streaming this data to a PC in real time using wireless connection. With a sampling rate of 256hz this allows for the possibility of reliably measuring HRV during laboratory tasks which require a participant to move around.

The simplest cardiac variable is the heart rate (HR). HR is measured in beats per minute (BPM) and is typically averaged across a time window, or *epoch*. The HR is the most commonly used measurement of cardiac function and is especially common in psychophysiological research due to its simplicity. In psychology, it is assumed that an

increase in HR reflects an increase in physiological arousal. Due to the dual-innervation of the heart from both sympathetic and parasympathetic origins, an increase in HR can arise due to either (1) enhanced sympathetic activity and/or (2) reduced parasympathetic activity. Therefore, it is useful to consider other measurements to tease apart changes in these two systems in response to an emotion induction.

Measuring HRV is one way in which researchers may attempt to separate these influences. HRV refers to the beat-to-beat variations that characterise the heart rate across time, and encompasses many different measurement techniques. Here I will describe the major HRV measures in the frequency, time, and nonlinear domains, with a focus on those which capture vagal activity. For review of HRV measures and norms, and discussion of the mechanisms they capture, see the following: Shaffer and Ginsberg (2017), Pham et al. (2021), Malik (1996), Berntson (2017), or Laborde et al. (2017).

Time Domain

This category involves various statistical techniques to quantify the variability in the R-R interval across time. One such method, the root mean square of successive R-R intervals (RMSSD) is considered the most statistically robust and is thought to be a measure of CVC. It is the HRV metric that is most widely used in research (Pham et al., 2021). In simple terms, deriving this metric involves calculating the differences between successive R-R intervals, squaring each of these differences, before taking an average and obtaining its square root (see figure 4, below). RMSSD is reported in milliseconds and is often subjected to natural log transformation (Shaffer & Ginsberg, 2017).

Figure 4: Formula for calculating RMSSD

Frequency Domain

Heart rate varies across different time scales. It is accepted that the mechanisms that underly HRV at different frequencies differ, and using components of the frequency domain affords researchers a more fine-grained assessment to tease apart these influences. Frequency domain measures are usually derived from a fast-fourier transform of the RR time series, allowing an analysis of spectral density, and the quantification of HRV within different frequency bands. The HRV frequency domain is traditionally split into three spectrum bands. The high-frequency band (HF) is usually set to 0.15-0.40 hz, for the reason that this range maps onto the human respiration cycle, i.e. 9 – 24 breaths per minute. Therefore, HF is considered a surrogate measure for RSA and thought to quantify vagal activity. The low-frequency band (LF) is usually set to 0.04 – 0.12 hz. The use of this frequency is controversial; LF was once commonly thought to measure the sympathetic influence on HRV, however, more recent data has revealed a more complicated picture. LF likely reflects the influences of both these branches, plus circadian rhythms and other metabolic factors. An ultra-low-frequency band (ULF) is also occasionally described in the literature, and is thought to be primarily mediated by circadian and thermoregulatory influences. When reporting frequency-derived HRV metrics, the total power in the band of interest is reported usually in milliseconds-squared[10] (ms^2), and often log transformed similarly to RMSSD. Further still, the ratio of LF to HF (LF:HF) is commonly used – once thought to indicate the

so-called sympathovagal balance in the nervous system, a practice which has largely fallen out of favour because the source of LF remains unclear. Note that using the ratio provides equivalent information to reporting HF and LF in normalised units (for further explanation see: Burr, 2007).

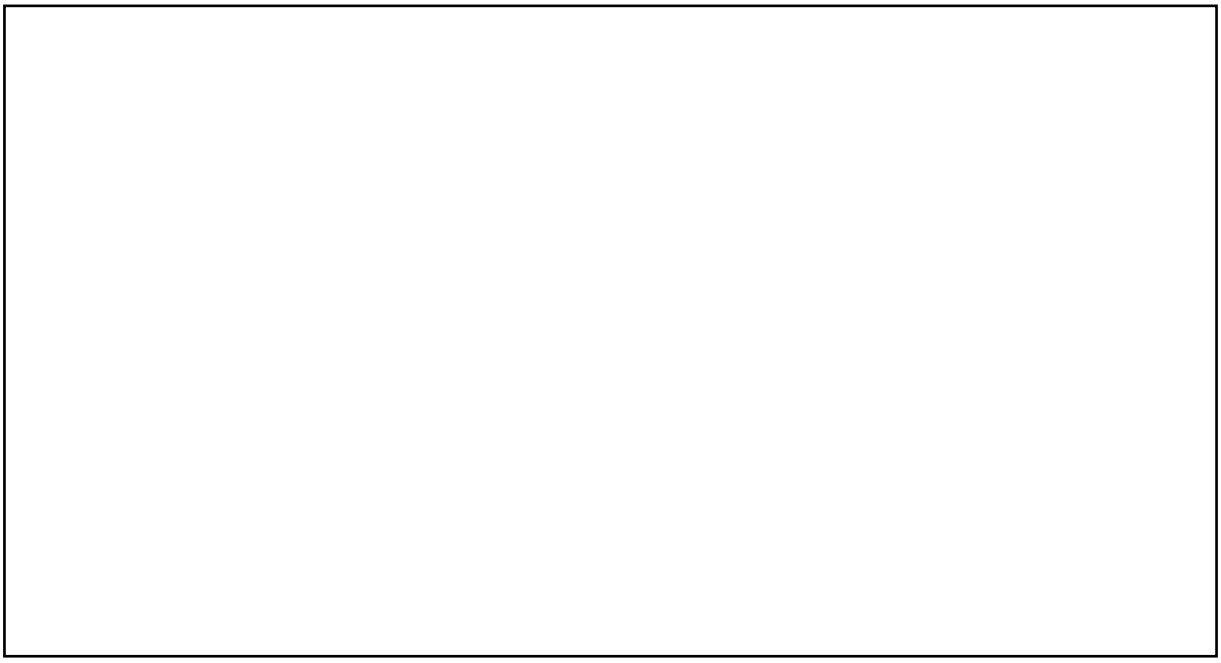

Figure 5: the relationship between heart rate (left) and HRV frequency (right)
From top to bottom panels: high frequency, low frequency, and total power of variability.

To summarise, there are two commonly used and well-validated metrics of HRV which are used to measure vagal activity (RMSSD and HF). Other variables can provide useful information but as less is known about their properties and sources they should be interpreted with caution. In general, the gold-standard measurement duration for both RMSSD and HF is 5-minutes — although ultra-short measurement durations have also been used, and will be discussed in Study 2. HRV measures are highly sensitive to measurement length, as they attempt to capture subtle changes in variability, and so longer measurements with more RR intervals are preferred. A common concern here is validity, i.e. whether shorter measurement durations correlate with subsequent measurements, given that resting HRV is often intended to measure trait-level individual differences. For HRV reactivity (within-

subjects designs) this concern is less, as the greater concern is whether shorter measurements are sensitive enough to capture emotion-related variance.

Other physiological variables

Heart rate will be used to measure the overall state of arousal, without differentiating the constituent autonomic influences. HRV will be used to quantify vagal activity. Additionally, it is of interest to tease apart the sympathetic component in the physiological response to our lab stressors. Measuring electrodermal activity is one way to assess sympathetic activity, because the sweat glands at the surface of the hands are innervated solely by the sympathetic nervous system. During states of sympathetic arousal more sweat is secreted. By placing two electrodes at this surface and recording the electrical conductivity between them, the relative change in conductivity can be measured; as more sweat is produced, the conductivity increases. Therefore, a skin conductance level (SCL) can be recorded and has been widely used as an index of sympathetic activation (Boucsein, 2004; Boucsein et al., 2012).

Research Questions

To return to the overarching theme of this thesis — it is abundantly clear that the coupling between the brain and heart is crucial in both emotion and cognition, and that this relationship is observable in HRV measurement (theoretically due to the mediating role of the vagus nerve and the central autonomic network). However, three questions pertaining to the link between HRV, emotion, and emotion regulation remain unanswered:

Question 1

How does the parasympathetic nervous system respond under conditions of acute threat? It is known that both autonomic changes occur as part of the response to acute threat, and it is thought that a withdrawal of cardiac vagal control specifically mediates the

allocation of metabolic resources to deal with challenges. However, there is a need to better characterise the changes that occur in cardiac vagal control during threat, and how it recovers following restoration of non-threatening conditions.

Question 2

How does resting parasympathetic activity relate to the parasympathetic response under conditions of acute threat? Since resting HRV measurements have been related to an adaptive capacity for emotion regulation (evidenced by self-reported emotional dysregulation and to psychopathology), it is of interest to investigate the relationship between resting HRV and the HRV reactivity and recovery that occurs in response to threat.

Question 3

How is parasympathetic reactivity modulated by emotion regulation strategies? Emotion regulation has been linked to cardiac vagal control through self-report measures and resting HRV measures. However, it is yet to be established whether the use of emotion regulation strategies influences HRV Reactivity during emotional challenge. It is unknown whether attempting to regulate emotions causes greater or less HRV reactivity, or whether different strategies (i.e. suppression and reappraisal) have divergent consequences on HRV reactivity and recovery.

Hypothesis

The convergence of several theories forms my *top-down regulation hypothesis of cardiac vagal control* (hereafter the regulation hypothesis). This hypothesis is based on a neural system whereby PFC inhibition (of subcortical and brainstem nuclei activity) is positively associated with RMSSD and HF measures of HRV, via cardiac vagal control. Deliberate engagement in effortful cognitive control strategies, such as reappraisal or suppression, enhances HRV through the same pathway. However, because of the differences

in how these strategies intervene in the process of emotion generation, reappraisal (a resource-intensive, proactive and effective strategy) should show an enhanced effect in this regard, i.e. more HRV relative to suppression (a reactive, less resource-intensive and less effective strategy). This hypothesis generates the following predictions in answer to the three overarching questions addressed in this thesis.

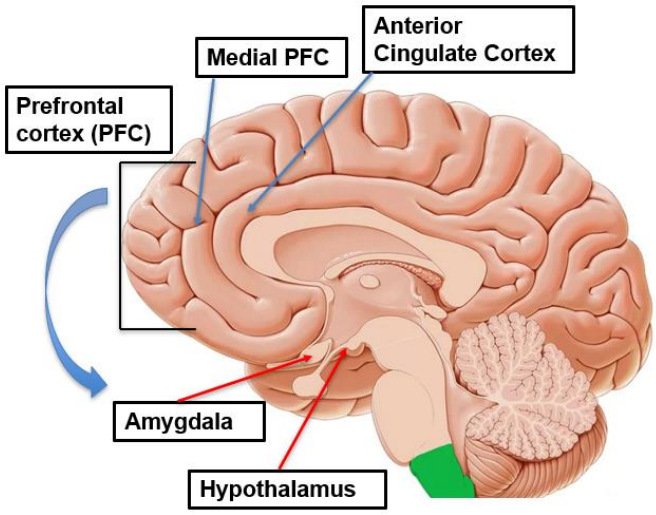

Figure 6: neural structures involved in regulation hypothesis
Hypothesized mechanisms common to emotion regulation and HRV; the central autonomic network and how it is hypothesized to relate to top-down inhibition in cognitive control and efferent vagal activity.

Predictions

Prediction 1

In light of the hypothesis detailed above, i.e. if it is the case that part of an adaptive response to threat is a withdrawal of cardiac vagal control (and decreased prefrontal activity) I predict that during threat, HRV will substantially decrease, and recover to baseline levels afterward.

Prediction 2

If it is the case that resting cardiac vagal control reflects an adaptive capacity for emotion regulation, then higher resting HRV should predict smaller magnitude decreases in HRV (i.e. less HRV Reactivity).

Prediction 3

If it is the case that emotion regulation strategies rely on cognitive control mechanisms subserved by prefrontal networks, and that prefrontal activity is translated into HRV through the central autonomic network, then use of reappraisal and suppression during exposure to threat should be associated with higher HRV (and therefore less HRV Reactivity). More specifically, to the degree that reappraisal is a more effective strategy (because it is situated earlier in the process of emotion generation) then the association between HRV and reappraisal use should be stronger than that of HRV and suppression use.

The Present Studies

In this thesis I will present three studies in which I test the above hypothesis. Studies 1 and 2 use archival data from studies of emotional challenge conducted in the Laboratory for Cognitive and Affective Neuroscience. Study 3 is a new preregistered study designed to specifically address how dynamic changes in cardiac vagal control are affected by emotion regulation strategy use.

Crucially, these studies employ ecologically valid paradigms to induce stress in participants, across two types of context: social stress (Study 1), and extreme height-exposure in virtual reality (Study 2 and 3). I will use HRV measurement to index cardiac vagal control (complimented by other physiological measures), namely the measures RMSSD and HF. Further, study designs will make use of resting, reactivity, and recovery[11] periods to assess

[11] All three studies include Resting, Reactivity, and Recovery measures of HRV with the exception of Study 2 which has no recovery period.

the time course of the HRV response to threat. Finally, in all three studies, self-report questionnaire measures will assess the use of reappraisal and suppression strategies during the threat exposures, while emotion ratings will be obtained throughout to capture the subjective experience of negative emotion.

Study 1 uses archival data from the Trier Social Stress Test in a laboratory environment. This will allow me to investigate the time course of HRV responding across resting, reactivity and recovery, in relation to a *social* threat. Further, self-reported history of non-suicidal self-injury behaviour will allow me to test whether HRV reactivity and recovery varies based on this index of psychopathology. Studies 2 and 3 will use a *Virtual Reality* paradigm in which participants are exposed to extreme heights in a realistic, simulated environment, which has been shown to elicit a strong experience of fear. In Study 2 I aim to establish the pattern of HRV responding in relation to *physical* threat using archival data. In Study 3 I will use a similar virtual reality simulation, this time experimentally manipulating the use of emotion regulation strategies by randomly assigning participants to a reappraisal, suppression, or control conditions.

Study 1: Social-Evaluative Threat

Trier Social Stress Task

Social situations in which people feel evaluated by others are often experienced as threatening. For this reason, the *Trier Social Stress Test* (TSST; Kirschbaum et al., 1993) has been widely used as a paradigm for studying the stress response in humans and is considered a gold-standard laboratory stress induction (Shields & Slavich, 2017). The paradigm typically involves the preparation and delivery of a five-minute speech, followed by an observed serial subtraction task, both of which are observed by a panel of confederate judges. Experimental designs using the TSST usually employ a structure that includes baseline, stressor, and recovery components. The presence of unresponsive judges creates a context of perceived social judgement, and participants in this paradigm often self-report heightened levels of negative affect, stress, and anxiety (Yim et al., 2010; Rimmele et al., 2009). In addition to these subjective parameters, a large body of research has also demonstrated marked physiological reactivity to the paradigm. Importantly for the study of cardiac responses to stress, heart rate is shown to increase by 15-25 bpm during the stressor, usually returning to baseline in the subsequent recovery period (Kudielka et al., 2007). Activation of the hypothalamic-pituitary-adrenal axis (a neuroendocrine substrate of the threat response) is observed during the TSST, as marked by a doubling or tripling of salivary cortisol levels in most participants (Kirschbaum et al., 1993). Enhanced activity in the sympatho–medullary pathway is indicated by elevated salivary a-amylase levels[12] (Graef et al., 2003).

Trier Social Stress Task and HRV

[12] an enzyme involved in the sympathetic response

Studies have also assessed the effect of the TSST on measures of cardiac vagal control, in most cases demonstrating a significant decrease (Lackschewitz et al., 2008; Jentsch & Wolf, 2020), suggesting that the TSST elicits concurrent sympathetic activation and vagal withdrawal. In a series of studies with healthy participants, significant decreases in RMSSD and HF were observed during the stressor component (Petrowski et al., 2010; Petrowski et al., 2017). Further, a review of the effect of the TTST on physiological reactivity in youth samples also found that HRV significantly decreases during the task (Seddon et al., 2020).

The pattern of HRV responding to the TSST in clinical groups was recorded in a study by Kircanski et al. (2016). They administered the task to healthy controls, and to participants diagnosed with depression, anxiety, or both. In healthy controls, there was a sharp decrease in HRV upon onset of the stressor, followed by a sharp increase again during the recovery period afterwards. Interestingly, all three clinical groups demonstrated a pattern that differed from controls: while they were no different at baseline, the clinical groups all showed a smaller change in HF across the task. Relative to healthy controls, the clinical groups all showed a smaller decrease during the stressor and a smaller increase in the recovery period. So, despite finding no evidence for differences in *resting* cardiac vagal control, Kircanski and colleagues reported a diminished response (less HRV reactivity) to social stress, in their samples from a population that tends to present with impaired emotion regulation. This finding is consistent with the notion that affective disorders are marked by a reduced flexibility in cardiac vagal control (Beauchaine, 2015; Mulcahy et al., 2019).

However, the effect of the TSST on cardiac vagal control in healthy controls is inconsistent across studies. At least one study has found an average *increase* in HRV during the stressor portion of the paradigm (Yim et al., 2015). Other studies have found no evidence for a change in HRV (Altemus et al., 2001; Rohleder et al., 2006). Gender is one factor that

may contribute to this difference. A meta-analysis (Hamidovic et al., 2020) examined 17 studies in healthy controls where public speaking tasks were used to induce social stress (including but not limited to the TSST). Evidence was found in support of a gender difference in HRV during social stress. Women and men were no different at resting time points, however, *during* social stress women exhibited lower HRV than men, suggesting a greater decrease. Women also had marginally lower HRV during both anticipation and recovery from social stress. Gender may therefore be a moderating variable in the effect of social stress on cardiac vagal control.

Taken together, it seems that changes in cardiac vagal control are a part of the normative response to socially-evaluative situations which are perceived as threatening. However, the nature of these changes is not yet fully understood. Given the emphasis given to the role of cardiac vagal control during social interactions (i.e. Porges, 1992), the TSST affords a unique opportunity to explore the nature of this dynamic response. The three-part design of the TSST allows the relationship between Resting, Reactivity and Recovery levels of measurement to be assessed. There is also some evidence that cardiac vagal control under social-evaluative threat may vary as a function of between-subjects factors, specifically psychopathology diagnosis and gender, but more investigation is warranted. Finally, it is yet to be tested whether emotion regulation strategies alter HRV during the TSST.

Robinson, 2021 (PhD Thesis)

A previous study from our lab explored whether there were differences in the overall emotional response to social stress in individuals who engage in *non-suicidal self-injury* relative to healthy controls[13]. Evidence from self-report data suggests that non-suicidal self-

[13] This was Study 2 from Robinson's PhD thesis: Robinson, K. (2021). *Emotion in non-suicidal self-injury: A contradiction between global self-reports and real-time responses* (Doctoral dissertation, Open Access Victoria University of Wellington| Te Herenga Waka).

injury behaviour is associated with elevated emotional reactivity and difficulties in trait self-regulation (You et al., 2018; Robinson et al., 2019). Theorists argue that self-injury may be driven or maintained by emotion reactivity, understood as a negative reinforcement cycle whereby the behaviour provides relief from aversive states in lieu of less harmful self-regulation strategies (e.g. the *Integrated Theoretical Model*; Nock et al., 2009). Maladaptive patterns of emotion regulation during social-evaluative threat in particular may be relevant in individuals who engage in non-suicidal self-injury (Groschwitz et al., 2016), and cardiac vagal control may play a role in these patterns (Crowell et al., 2005).

In this study, 101 female participants experienced the TSST. Participants were recruited based on their responses to pre-screening questions regarding history of non-suicidal self-injury (NSSI) to form two groups: NSSI and Control. Multimodal measures of emotional responding were recorded including self-reported discrete emotions, heart rate (derived from ECG), and skin conductance. The stress induction was found to be effective as indexed by significant increases in both subjective (self-reported negative affect) and physiological (heart rate and skin conductance level) measures. Surprisingly, no evidence was found for a difference between NSSI and Control groups on measures of emotional reactivity. Robinson's initial publication of this study in her PhD thesis included heart rate data but did not include HRV measures. As high-quality ECG data were available this study presented an opportunity to address the questions in this thesis. All participants in this study are female, thereby avoiding gender as a potential source of variability. Additionally, a subset of participants completed a modified ERQ measure which asked them to report their use of suppression and reappraisal during the TSST.

Planned Analyses

Firstly, the hypothesis that cardiac vagal control decreases and returns to baseline in response to social stress will be tested using two repeated-measures ANOVAs. These ANOVAs will test for the effect of *Epoch* (3 levels; Baseline, TSST, Recovery) on RMSSD and HF, with also a between-subjects factor of *Group* (2 levels; NSSI vs Control). A significant within-subjects effect of time on RMSSD and/or HF would provide support for this hypothesis. In the case of significant effect of time, post-hoc tests will be used to confirm within-subject differences between time points (i.e. significant differences in HRV should be found between baseline and stress, and/or stress and recovery). The interaction between Group and Epoch would provide converging evidence for the regulation hypothesis.

Secondly, to test the relationship between Resting HRV and HRV Reactivity and recovery, regression models will be used where baseline RMSSD and HF predict *changes* in RMSSD and HF. These change scores will be calculated by subtracting HRV measures during the Baseline from those during the TSST (ReactivityΔ), and Recovery minus TSST (RecoveryΔ[14]). Specifically, if the regulation hypothesis is correct, higher Resting HRV will be associated with smaller ReactivityΔ values.

Thirdly, to test the hypothesis that cardiac vagal control Reactivity and Recovery vary based on regulation strategy use, regression modelling will be used to predict RMSSD/HF Reactivity and Recovery scores. Self-reported use of reappraisal and suppression will be predictors. Use of reappraisal should predict less Reactivity during the TSST relative to suppression.

Method

[14] I use "RecoveryΔ" to differentiate this measure, a change score, from the tonic measurement taken from the recovery epoch. Both ReactivityΔ and RecoveryΔ are change scores.

Study 1 uses previously collected data which tested for differences in emotional responding between individuals who report engaging in non-suicidal self-injury and those who do not. The method section here will describe only procedural information that is relevant to the present study (for the full description, see Robinson, 2021). The original study was preregistered at https://osf.io/px534/. For the purposes of this thesis, the existing cardiac data will be processed to derive HRV and other measures. The HRV analyses in the present study were exploratory and were therefore not preregistered.

Participants

Participants were 101 undergraduate psychology students recruited from Victoria University of Wellington (*M* age = 18.72, *SD* = 1.29). A survey prior to recruitment was used to select women who were between the ages of 17 and 25, fluent in English, capable of using a computer screen and mouse, with normal or corrected to normal vision, and who consented to participating in self-injury research. Participants were also recruited on the basis of their self-reported history of engaging in non-suicidal self-injury. On a pre-screening measure, 51 participants reported engaging in non-suicidal self-injury in the past year, and 50 reported never having done so. All participants in the sample identified as female, with the exception of one who identified as gender-fluid. Participants received mandatory course credit for taking part.

Procedure

Participants first completed a "vanilla baseline" task in order to measure baseline physiological indices and subjective affect. The vanilla baseline epoch comprised a minimally challenging colour-counting task which lasted five-minutes while seated still and alone in the testing room (Jenning et al., 1992). A vanilla baseline is preferred over a

stimulus-free baseline in research with clinical groups, as it elicits less anxiety than simply sitting quietly.

Next, the social-evaluative stress induction was induced using the mathematics component of the Trier Social Stress Test (Kirschbaum et al., 1993). In this task the lead researcher (Robinson) informed participants that they were to complete a mental arithmetic task that assesses working memory and verbal intelligence, and that this would be administered by an evaluator who was trained in the assessment of verbal and non-verbal behaviour (actually an older male confederate). The confederate maintained a neutral, professional manner while instructing the participant to count aloud backwards from 2023 in intervals of 17 as quickly and as accurately as possible. Participants were instructed to restart every time that they made a mistake, and those who performed well were encouraged to count faster.

Following this stress-induction, participants were told that the working memory task was completed and that their task was to sit and relax. This phase was designed to assess recovery while participants were left to regulate their own emotions (i.e. spontaneous regulation), and so participants were not given a vanilla baseline task in the recovery phase. Participants were left to sit alone in the testing room for five minutes.

After the Recovery period was completed, participants watched an excerpt from a nature documentary, and then rated photographs of natural scenes, both designed to induce positive emotion and restore mood to baseline levels. Participants were finally asked to complete some questionnaire measures which assessed their use of self-regulation strategies both during the TSST and during Recovery, and also assessed their emotion reactivity and dysregulation in daily life.

Physiological recording

Electrocardiography was recorded continuously throughout the duration of the experimental procedure, using ADInstruments ML408 Dual Bio Amp. Raw ECG was sampled at 1000hz and converted from analogue to digital by the PowerLab 16/30 Amplifier (ML880; ADInstruments, Australia). Electrodermal response (EDR) was also recorded throughout, using ADInstruments MLT116F EDR dry electrodes, placed on the medial phalanx of the index and ring fingers of the right hand at a sampling rate of 1000hz, amplified via the ML116 EDR amplifier (ADInstruments, Australia). Both the ECG and EDR signals were recorded directly into LabChart Pro 8.0 software.

Self-Reported Emotion

Self-reported emotion ratings were collected at the end of each phase (Baseline, TSST, and Recovery). Ratings were collected using visual analogue scales where participants rated the degree to which they were currently experiencing each of nine emotions. Responses were made on a 17.8cm visual analogue scale on a computer screen which ranged from *'0 – Not at All' to '100 – Extremely'.* Participants responded by moving a marker from the midpoint of the scale corresponding to 50. The nine emotions were presented in a randomised order for each assessment and each participant. Instructions stated: *"This scale consists of a number of words that describe different feelings and emotions. Read each item and then rate how much it applies to you at the present moment. Right now I feel…"*. The emotion words used were: *Happy, Sad, Angry, Anxious, Stressed, Jittery, Frustrated, Embarrassed, Ashamed.* Responses corresponding to the 8 negatively-valenced emotions were averaged together for each participant to create a mean negative affect value at Baseline, TSST, and Recovery time points. To create an index of the change in negative emotion attributable to the TSST, Baseline levels were subtracted from TSST levels for each participant.

Questionnaires

An adapted version of the *Emotion Regulation Questionnaire* (Gross & John, 2003) was administered following participation in the TSST and the positive mood induction. The standard 10-item scale was used here, except that participants were asked to reflect back to their time during the TSST and the Recovery period when responding to the items. Responses are made on a 1-7 Likert scale where 1 is "strongly disagree" and 7 is "strongly agree". For example, an item used to capture reappraisal is item 3: "*When I want to feel less negative emotion (such as sadness or anger), I change what I'm thinking about."* Item 9, intending to capture the use of suppression, reads: "*When I am feeling negative emotions, I make sure not to express them."*. Scores were averaged for two facets: the six items measuring reappraisal and the four items measuring suppression, to create average scores (with a range of 1 to 7) for the use of each strategy (hereafter 'reappraisal use' and 'suppression use'). The purpose of this measure was to assess the degree to which participants used cognitive reappraisal and expressive suppression strategies in *real-time* (rather than their global/trait-levels) during the TSST and Recovery.

Two additional questionnaire measures were administered following the positive emotion induction. The Emotion Reactivity Scale (Nock et al., 2008) which has 21 items, and the brief version of the Difficulties in Emotion Regulation Scale (Bjureberg et al., 2016) which has 16 items, both of which use 5-point Likert scales. These were global/trait-level measures and will only be used to compare self-reported emotional responding between groups.

Physiological Data Processing

For all physiological variables, participants' values were calculated for each of the three epochs (Baseline, TSST, Recovery). EDR was converted from volts to micro-Siemens (μS) offline and smoothed at 999 samples per second using a median filter, with the first 30s

of each epoch being removed to account for the participants' habituating to the room and to the experimenter leaving the room.

HRV Processing[15]

The raw ECG signal was viewed in LabChart and was pre-marked (during data collection) with comments denoting the start and end of each portion of the TSST. Segments selected from this signal were 260 seconds of each of the three five-minute epochs (Baseline, Stressor, recovery), excluding the first and last 20s of data. This exclusion was to account for the habituation of the participant and also to exclude the effects of the experimenter leaving and re-entering the room. ECG pre-processing involved two steps: first, an algorithmic classification allowed for the selection of good beats for further processing and constituted a first-pass for the detection of both physiological and technological artifacts. The HRV function in LabChart, using the default "human" settings, automatically detected R peaks: the highest inflection on the ECG signal representing maximal depolarisation of the heart's ventricles. LabChart's "beat classifier" view was used to allow a graphical representation of each beat (R peak) along the dimensions of complexity (shape of the QRS complex) and R-R interval (latency since the preceding beat). Second, a manual process of visual inspection of the ECG signal followed to further ensure the rejection of artefacts. A high-pass digital filter was applied at 8hz for ease of visualisation. In cases where LabChart had detected an R peak where there was none (e.g. erroneously detecting a T-wave), this whole beat was excluded. R peaks which appeared to represent ectopic beats (abnormally long or short R-R interval) were excluded if they fell outside of the criterion of 350-1600ms. Sections or beats which contained clear technological artifacts, such as where there was evidence that an ECG pad had lost contact from the skin, were also excluded.

[15] This protocol for processing ECG data and subsequently calculating HRV measures was repeated for Studies 2 and 3 – the only differences pertain to the length of epochs.

Following this inspection and artifact detection process, LabChart modules were used for the following calculations. Mean HR (in beats per minute) and mean R-R interval (in milliseconds) were calculated as measures of heart rate and heart period. Several HRV variables were then calculated using the R-R series. The time-domain measure RMSSD, the root mean square of successive R-R differences, was calculated as the main index of cardiac vagal control. Spectral analysis was also performed using a fast-fourier transform, to derive the high-frequency component (HF), using the spectral band 0.15-0.4hz.[16]

Results

Exclusions

All analyses included the full dataset of 100 participants (control n = 49, NSSI n = 51) except for those using ERQ data, as only 55 participants provided scores for both Suppression and Reappraisal subscales.

Manipulation Check

The within-subjects manipulation of social-evaluative threat was shown to elicit strong changes in emotional arousal, as measured by self-reported negative affect, skin conductance level, and heart rate. This robust emotional response was not modulated by NSSI status as shown in figure 9 (below).

[16] Several other HRV measures were calculated; correlations between all physiological variables are reported in Appendices A-C

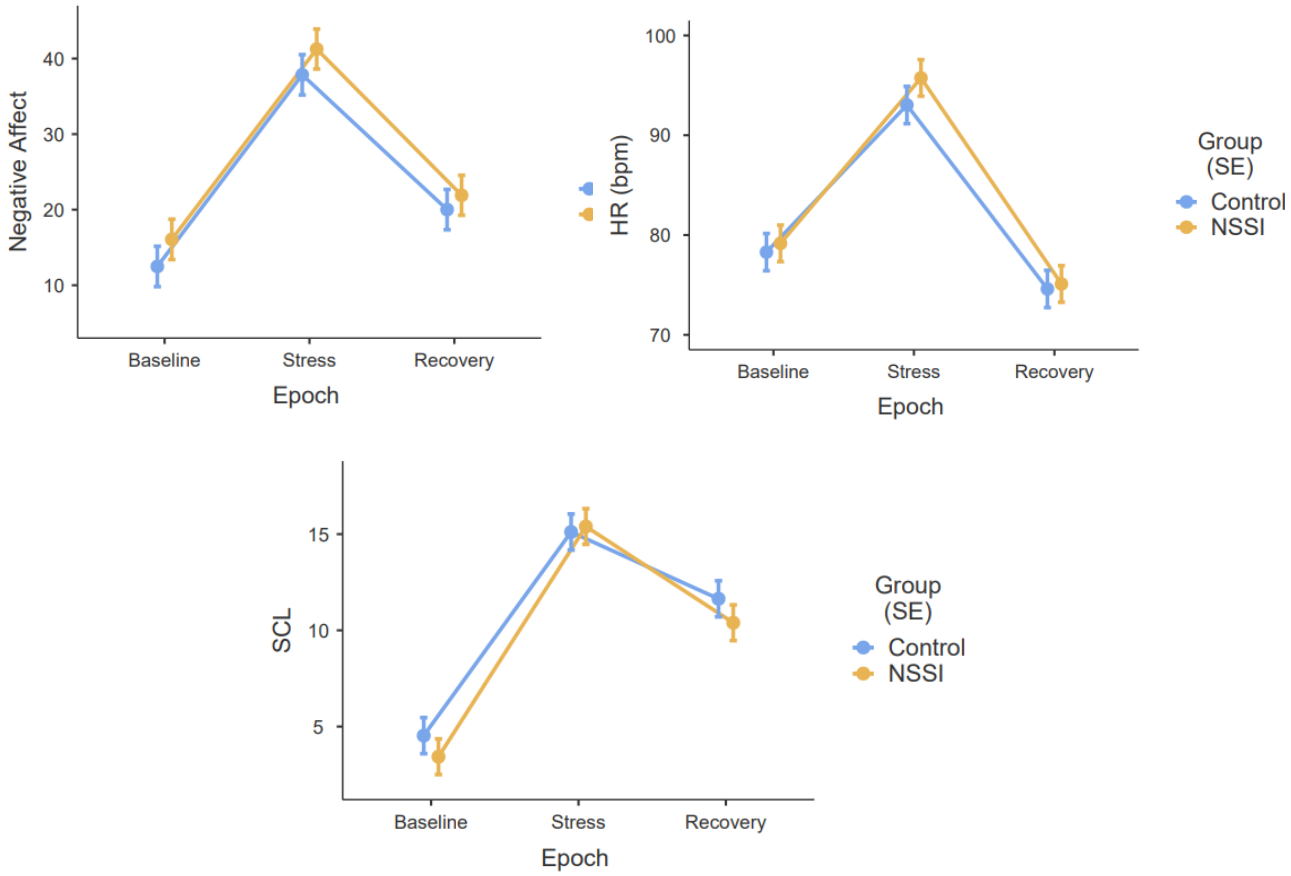

Figure 7: measures of emotion, split by Group
Measures of emotion across Baseline, TSST, and Recovery epochs as a function of Group. From left to right: Self-reported negative affect ratings; skin conductance level (microsiemens); heart rate (bpm).

Between-Group Tests

The between-subjects factor Group (NSSI vs controls) was determined based on self-reported behavioural history. However, the groups did indeed differ in terms of their trait emotion-regulation: the NSSI group scored significantly higher on self-reported measures of emotion reactivity (Emotional reactivity Scale; ERS) and emotion dysregulation (Difficulties in Emotion Regulation Scale; DERS), see figure 8 below. No between-group differences were observed on the modified ERQ subscales for suppression and reappraisal use.

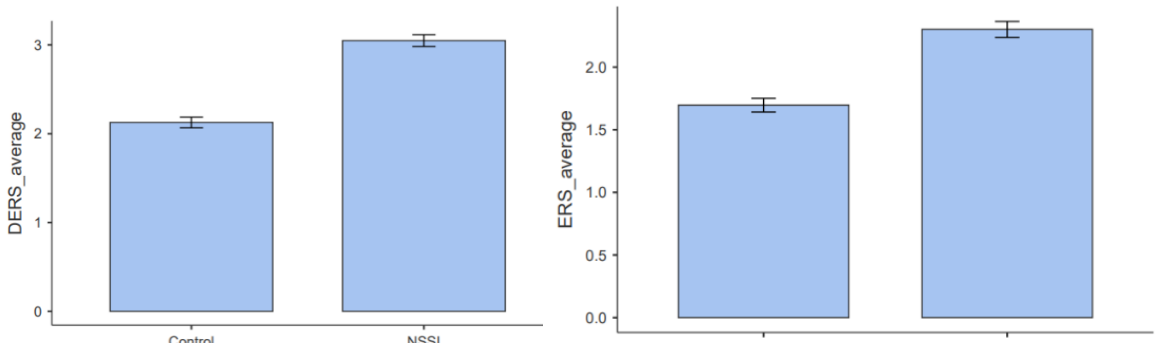

Figure 8:Manipulation Checks
Average scale scores for the DERS and ERS scales, split by group

Time Course - lnRMSSD

As anticipated, RMSSD differed from a normal distribution at all time points, exhibiting a substantial right skew. It was submitted to natural log transformation. The resulting lnRMSSD data were normally distributed.[17]

Inferential analysis of lnRMSSD data found that on average, cardiac vagal control decreased from the resting Baseline epoch during the Stressor epoch, and subsequently was restored to resting levels. Statistically, a Greenhouse-Geisser corrected repeated-measures ANOVA[18] revealed a significant effect of time on lnRMSSD, $F(1.44, 1) = 55.6$, $p < .001$, $\eta_p^2 = .362$. Follow up Tukey's tests showed a significant decrease in lnRMSSD from Baseline ($M = 3.66$, $SD = 0.61$) to Stressor ($M = 3.38$, $SD = 0.62$) epochs, and a significant increase from Stressor to Recovery epochs ($M = 3.86$, $SD = 0.59$). Interestingly, lnRMSSD during Recovery was significantly higher than during Baseline (all p's $< .001$) indicating a substantial "rebound" phenomenon in cardiac vagal control following the stressor.

[17] Throughout the studies in this thesis, natural log transformations will be used for RMSSD and HF data, as is conventional in the literature. Nonetheless, the repeated-measure ANOVA is considered robust to such deviations and so they are reported as main analyses.

[18] For the repeated-measures ANOVAs in studies 1-3, I adopted corrections for violations of sphericity using the Greenhouse-Geisser and Hyun-Feldt methods, depending on the epsilon statistic in line with Girden's (1992) recommendations. Both reduce the likelihood of type-1 error via adjusting degrees of freedom. Huynh-Feldt is used where epsilon > .75, and Greenhouse-Geisser when epsilon < .75

Experimental (*M* = 3.61) and control (*M* = 3.72) groups did not differ at baseline, and there was no main effect of group (*p* = .544). Moreover, the interaction term for *group* x *time* was not significant (*p* = .533), confirming that there was no evidence in favour of a difference in time course of changes in vagal control between control and NSSI groups.

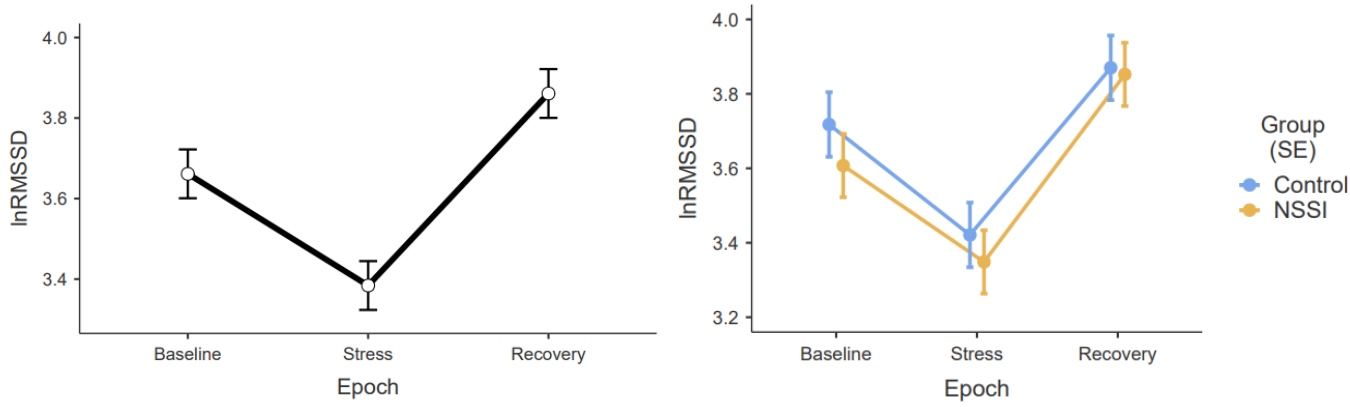

Figure 9: lnRMSSD in ms, by epoch (left), split by experimental group (right)

Time Course - lnHF Power

Following natural log transformation, lnHF power data were normally distributed. Mirroring the pattern of lnRMSSD, a significant decrease during emotional challenge and subsequent reinstation of cardiac vagal control was observed in lnHF. A repeated measures ANOVA (Huynh-Feldt corrected) revealed a significant effect of Epoch on lnHF, F(1.56, 1) = 30.9, $p < .001$, $\eta_p^2 = .240$. Follow-up Tukey's comparisons[19] for lnHF showed a significant decrease (*pTukey* = .008) from baseline (*M* = 6.47, *SD* = 1.33) to stressor (*M* = 6.17, *SD* = 1.15) epochs. A significant increase was observed from the stressor to the recovery (*M* = 6.93, *SD* = 1.24) period, and lnHF was significantly higher during Recovery than Baseline (*p*s < .001). No difference in lnHF showed at Baseline between Experimental (M = 6.57) and Control (M = 6.37) groups, and no main effect of Group was found (*p* = .541). Finally, the

[19] Tukey's T-tests are used in all cases where post-hoc comparisons are required

interaction term for Group and Epoch was not significant [F(1.54) = .346, p = .652, η_p^2 = .004], providing no evidence for a difference in the time course in changes in cardiac vagal tone between participants who had engaged in self-injury and controls was found.

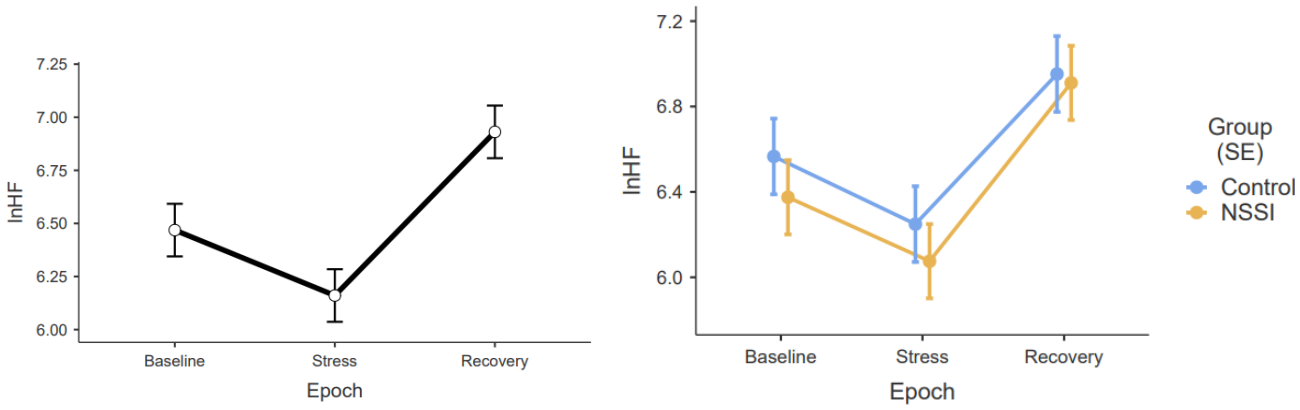

Figure 10: lnHF in ms^2, by epoch (left), split by group (right)
The natural log of lnHF power ms^2, as a function of epoch, and split by experimental group (controls and non-suicidal self-injury)

HRV Reactivity

HRV *Reactivity*Δ was measured by calculating change scores, whereby the Baseline HRV value was subtracted from the TSST value. Reactivity/change scores for both lnRMSSD and lnHF were calculated for each participant. Positive values represent an increase in HRV during the TSST, relative to Baseline, while negative values represent a decrease in HRV. On average participants exhibited a decrease in HRV during the TSST, a pattern which was true for both lnRMSSD (M = -0.277, SD = 0.529) and lnHF (M = -0.308, SD = 1.14). The data for these ReactivityΔ scores were normally distributed and show large between-subject variability: for example lnHF reactivity ranged from -3.64 to 2.65. This range indicates that many participants showed an *increase* in cardiac vagal control during the TSST and suggests that the effect of social-evaluative threat on HRV is modulated by other variables. HRV Reactivity (for lnRMSSD) did not differ between NSSI and control groups (see figure 28A, Appendix A), T(98) = -.35, p = .724.

A hierarchical design was used to predict HRV Reactivity$_{\Delta}$ using linear regression modelling. Models predicting lnRMSSD reactivity are described and reported here (see Appendix for models using lnHF). Three linear regression models were employed. *Model 1* predicted lnRMSSD reactivity from Baseline lnRMSSD (resting HRV) and Group (NSSI vs Controls); *Model 2* entered Suppression and Reappraisal use (averaged scores from the ERQ subscales).

Model 1, predicting lnRMSSD reactivity from Baseline lnRMSSD and Group accounted for a significant amount of variance, R^2adj. = .328, $F(2,52) = 12.70$, $p < .001$. However, the addition of Suppression and Reappraisal scores in Model 2 did not significantly increase the variance accounted for in lnRMSSD reactivity, $R^2\Delta = .052$, $F(2,50) = 2.10$, $p < .133$. Model 2 did account for a significant amount of variance in lnRMSSD reactivity, R^2adj. = .331, $F(4,50) = 7.67$, $p < .001$, but an increased BIC relative to Model 1 (72.9 to 76.5) indicates a worse model fit upon the addition of strategy use as additional predictors.

Model 2 included all four predictors — Baseline lnRMSSD, Group, Suppression, and Reappraisal — and overall accounted for approximately 38% of the variance in lnRMSSD reactivity. The independent variable Baseline lnRMSSD was a significant predictor independently ($p < .001$), as was Reappraisal use ($p = .046$), whereas Group and Suppression use were not. When all five predictors are equal to 0, Reactivity$_{\Delta}$ is expected to be 1.55. For every 1 unit increase in Baseline lnRMSSD, lnRMSSD reactivity is expected to decrease by 0.4 units (std.error = .08), holding all other predictors constant. Additionally, for every 1 unit increase in Reappraisal use, lnRMSSD reactivity is expected to decrease by 0.08 units (std.error = .043), holding all other predictors constant.

In sum, hierarchical modelling for both measures of HRV indicate that individuals with higher levels of resting HRV/cardiac vagal control exhibited greater decreases in

HRV/cardiac vagal control during the TSST. Further, a small proportion of HRV reactivity is independently explained by use of Reappraisal, but not Suppression; however, adding these two variables into the model led to a worse model fit and did not significantly increase the amount of overall variance explained. Lastly, experimental Group membership (non-suicidal self-injury) did not predict HRV reactivity relative to control Group membership — perhaps unsurprising, given that group differences did not emerge for any other aspect of emotional response. Overall, these analyses provide significant evidence in opposition to the Regulation hypothesis regarding the Resting-Reactivity relationship — as higher Baseline HRV predicted greater *decreases* during TSST — and showed no relationship between reactivity and either strategy use or psychopathology. Note that Model 2 had less power than model 1, with only 55 of the total participants (see Table 1, below).

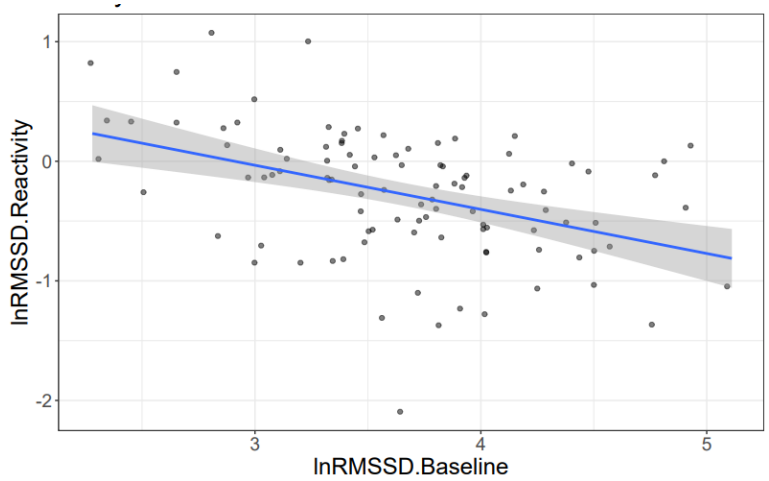

Figure 11: Scatterplot, Resting lnRMSSD predicting lnRMSSD Reactivity

HRV Recovery

HRV *Recovery*Δ was similarly measured by calculating change scores, whereby the TSST value was subtracted from the Recovery value. RecoveryΔ scores for both lnRMSSD and lnHF were calculated for each participant. Positive values represent an increase in HRV during the Recovery epoch, relative to the TSST epoch, while negative values represent a

decrease in HRV. On average participants exhibited an increase in HRV during the Recovery epoch, a pattern which was true for both lnRMSSD (M = 0.477, SD = 0.506) and lnHF (M = 0.771, SD = 1.06). These data show less between-subject variability, e.g. lnRMSSD Recovery$_{\Delta}$ ranged from -0.53 to 1.78, a range that indicates that most participants showed a restorative increase in HRV during the Recovery period, and few showed a decrease. HRV Recovery (for lnRMSSD) did not differ between NSSI and control groups, T(98) = -1.66, p = .101.

A hierarchical design was used to predict HRV Recovery$_{\Delta}$ using linear regression modelling. Models predicting lnRMSSD Recovery$_{\Delta}$ are described and reported here (see Appendix for models using lnHF). Three linear regression models were employed. *Model 1* predicted lnRMSSD Recovery$_{\Delta}$ from Baseline lnRMSSD (resting HRV) and Group (NSSI vs Controls); *Model 2* entered Suppression and Reappraisal use (averaged scores from the ERQ subscales).

Model 1, predicting lnRMSSD Recovery$_{\Delta}$ from Baseline lnRMSSD and Group accounted for a significant amount of variance, R^2adj. = .135, $F(2,52) = 5.21$, p = .009. The addition of Suppression and Reappraisal scores in Model 2 did not significantly increase the variance accounted for in lnRMSSD reactivity ($R^2\Delta$ = .011, $F(2,50) = .341$, p = .713). Model 2 did account for a significant amount of variance in lnRMSSD reactivity, R^2adj. = .112, $F(4,50) = 2.71$, p = .040, but an increased BIC relative to Model 1 (70.8 to 78.1) indicates a worse model fit upon the addition of Strategy use as additional predictors.

Model 2 included all four predictors — Baseline lnRMSSD, Group, Suppression, and Reappraisal — and overall accounted for approximately 18% of the variance in lnRMSSD Recovery$_{\Delta}$. The independent variable Baseline lnRMSSD was a significant predictor independently (p = .003), although Group, Suppression and Reappraisal use were not. When

all five predictors are equal to 0, Recovery∆ is expected to be -.589. For every 1 unit increase in Baseline lnRMSSD, lnRMSSD Recovery∆ is expected to increase by .255 units (std.error = .08), holding all other predictors constant.

In sum, hierarchical modelling for both measures of HRV indicate that individuals with higher levels of Resting HRV/cardiac vagal control exhibited greater increases in HRV/cardiac vagal control during the Recovery epoch. Group membership (NSSI vs. controls) did not predict HRV Reactivity∆ , and the use of neither Suppression or Reappraisal had an effect on this change during the Recovery epoch. The results of these analyses are consistent with the Regulation hypothesis insofar as higher Baseline HRV predicted greater Recovery from social evaluative stress. However, the findings pertaining to emotion regulation strategies and psychopathology are counter to the predictions of the Regulation hypothesis.

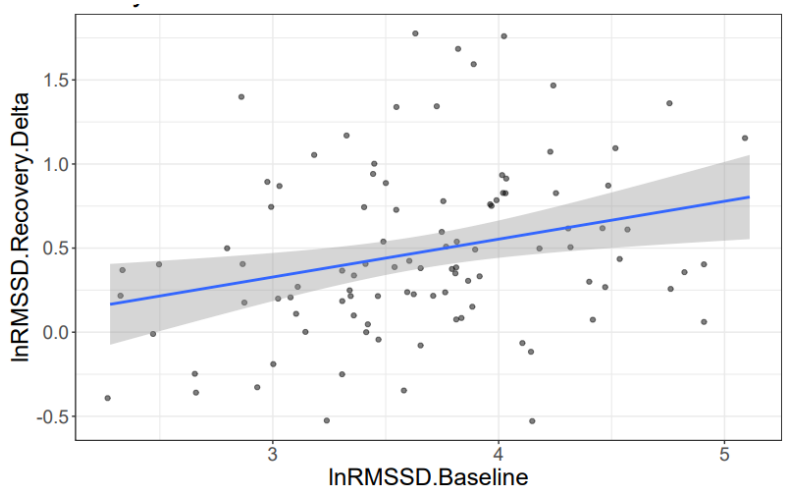

Figure 12: Scatterplot, Resting lnRMSSD predicting lnRMSSD Recovery∆

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Interim Summary

Data from study 1 demonstrated that social-evaluative stress elicits a strong within-subjects effect on HRV, as reflected in a decrease in cardiac vagal control. As predicted, HRV measures showed significant decreases between the Baseline and TSST epochs, followed by a significant increase during the Recovery epoch. These observations support the

idea of a quick release and restoration of vagal control being part of a coordinated physiological response to threat.

Further, Study 1 data support the hypothesis that Resting cardiac vagal control determines the pattern in this time course — although not in the expected direction. Interestingly, higher levels of HRV at Baseline predicted *greater decreases* during the TSST, while lower levels at Baseline were associated with smaller decreases, and sometimes increases. This is counter to the predictions of the Regulation hypothesis, which states that individuals with higher Resting cardiac vagal control more effectively self-regulate under threat, a process which is reflected in less Reactivity$_{\Delta}$ (i.e. smaller decrease in HRV during threat). Higher HRV at Baseline also predicted greater increases during Recovery following the TSST, indicating that individuals with higher tonic cardiac vagal control also recover more quickly from stress. These findings are more consistent with the predictions of Porges' Polyvagal Theory, than with the Regulation hypothesis, in that higher Resting HRV was associated with more dynamic range, or autonomic flexibility.

The effect of self-reported emotion regulation strategy on HRV Reactivity in Study 1 was limited. No interaction effect was found in the repeated-measures ANOVA between Group and Epoch, suggesting that NSSI status was not related to a different pattern of HRV Reactivity$_{\Delta}$. Reappraisal use had a significant, but small, relationship to HRV Reactivity$_{\Delta}$ such that higher self-reported Reappraisal predicted greater magnitude decreases in HRV during TSST, however, adding in the two strategies to the model did not improve model fit or variance accounted for. It is worth noting that the regressions used to test the hypotheses regarding strategy use included only a subset of the whole sample, and therefore it is entirely possible that there was insufficient statistical power to detect the effect of Reappraisal use which seemed to emerge. No variance in HRV Reactivity or Recovery was explained by whether participants reported a history of non-suicidal self-injury either, and in this case,

there was a sizeable sample combining Control and NSSI groups lending adequate statistical power.

Taken together, Study 1 shows that cardiac vagal control is reduced during emotional stress, but provided no substantial evidence that this dynamic response is modulated by a history of self-injury or the use of emotion regulation strategies. These findings argue against the hypothesis that top-down mechanisms subserving cognitive control are reflected in different patterns of HRV during social-evaluative threat.

Table 2: Descriptive Statistics for lnRMSSD, Strategy Use, and Negative Affect

Variable	*N*	Mean	SD	Min.	Max.	Skew	Kurtosis	Shapiro-Wilk *p*
Resting	100	3.67	0.61	2.28	5.11	-0.02	-0.190	.860
TSST	100	3.38	0.615	1.53	5.05	-0.1361	0.524	.665
Recovery	100	3.86	0.589	2.55	5.22	0.0486	-0.407	.791
ReactivityΔ	100	-0.28	0.53	-2.09	1.07	-0.35	0.95	.123
RecoveryΔ	100	0.48	0.51	-0.53	1.78	-0.50	0.09	.037
Reapp.	55	4.18	1.34	1.00	7.00	-0.05	-0.38	.828
Supp.	56	3.71	1.14	1.75	6.00	0.04	-0.88	.107
NegAffectΔ	99	25.14	22.13	1.89	86.67	0.79	-0.23	< .001

Table 3: *Correlation Matrix*

	Resting	TSST	Recovery	ReactivityΔ	RecoveryΔ	Supp.	Reapp.	ΔNegAffect
Resting	—	—	—	—	—	—		
TSST	.629***	—	—	—	—	—	—	—
Recovery	.891***	.648***	—	—	—	—	—	—
ReactivityΔ	-.428***	.433***	-.279**	—	—	—	—	—

RecoveryΔ	.273**	-.461***	.377***	-.853***	—	—	—	—
Suppression	-.192	-.133	-.187	.118	-.103	—	—	—
Reappraisal	-.140	-.290**	-.241	-.145	.030	.205	—	—
NegAffectΔ	.057	-.093	.050	-.175	.172	-.295*	-.388**	—

Note: Pearson's *r* reported in table. * $p < .05$, ** $p < .01$, *** $p < .001$

Table 4: *Resting lnRMSSD and Group as Predictors (Model 1)*

Coefficient	**Estimate**	**SE**	***p*-Value**
Intercept	1.19	.30	<.001
Resting	-0.39	0.08	<.001
NSSI – Control	-0.05	0.11	.630

Table reports unstandardised coefficients. $F(2,52) = 12.70$, $p < .001$. $R^2 = .328$, R^2adj. = .302, BIC = 72.9.

Table 5: *Entering Suppression and Reappraisal Use as Predictors (Model 2)*

Coefficient	**Estimate**	**SE**	***p*-Value**
Intercept	1.55	0.42	< .001
Resting	-.40	.08	< .001
NSSI-Control	.03	.11	.787
Supp.Avg	.02	.05	.664
Reapp.Avg	-.08	.043	.046**

Table reports unstandardised coefficients. $F(4,50) = 7.67$, $p < .001$. $R^2 = .380$, R^2adj. = .331, BIC = 76.5.

Model 1 – 2 comparison: $R^2\Delta = .052$, $F(2,50) = 2.10$, $p = .133$.

Table 6: Resting lnRMSSD and Group as Predictors (Model 1)

Coefficient	**Estimate**	**SE**	***p*-Value**
Intercept	-492	0.29	.106
Resting	.250	.078	.002**
NSSI-Control	.070	.111	.529

Table reports unstandardised coefficients. $F(2,52) = 5.21$, $p = .009$. $R^2 = .167$, R^2adj. = .135, BIC = 70.8.

Table 6: Entering Suppression and Reappraisal Use as Predictors (Model 2)

Coefficient	Estimate	SE	*p*-Value
Intercept	-.589	0.43	.175
Resting	.255	0.08	.003**
NSSI-Control	.081	0.11	.478
Supp.Avg	-0.02	0.05	.718
Reapp.Avg	0.03	0.04	.430

Table reports unstandardised coefficients. $F(4,50) = 2.71$, $p = .040$. $R^2 = .178$, R^2adj. = .112, BIC = 78.1.

Model 1 – 2 comparison: $R^2\Delta = .011$, $F(2,50) = .341$, $p = .713$.

Study 2: Insights from a Virtual Reality Height-Exposure Paradigm

Fear and Acrophobia

Having studied the dynamics of HRV in the context of social-evaluative threat, the question remains whether the findings of Study 1 replicate, and whether they generalise to other threat types. Therefore, in Study 2, I explore how HRV changes in the context of height-exposure — a context that signals physical danger.

Height-exposure is known to elicit a threat response in most people, usually accompanied by the experience of fear. While individuals vary in their emotional response to heights, from clinical levels of fear (known as *acrophobia;* Emmelkamp et al, 2002*)* to even positively-valenced "excitement", the normative fear of falling is considered an adaptive response (known as *basophobia;* Nesse, 2019). Most people experience some level of physiological arousal and subjective fear during exposure to heights. Responding in this way is likely a product of evolutionary pressures and therefore heights present a good context in which to study the role of cardiac vagal control in the response to threat and its relationship to emotion regulation. Because heights signal the presence of a very real physical threat, an individual must often deploy emotion regulation strategies in order to engage in goal-directed behaviour despite the threat. There are obvious ethical issues in using height-exposure *in vivo*; however, virtual reality can be used to give participants the feeling of height exposure without the physical danger.

Emotion in Virtual Reality

Virtual Reality is an emerging tool used in psychology research to study emotional states. For the purposes of studying mechanisms involved in fear and emotion regulation, virtual reality is the best currently available approach. Most studies of emotion regulation and

HRV have traditionally measured responses to emotionally charged stimuli, for example from the International Affective Pictures System (IAPS), which are rated for arousal and valence. While responses to emotionally relevant stimuli tell us a lot about the cognitive processes involved in emotion, they do not elicit strong emotional reactions. Other studies use video clips, audio, conversation or imagination, or threat of electric shock. These methodologies also allow for rigorous experimental control. However, they cannot reliably induce a realistic emotional state that is representative of a real-life state, indicating a lack of ecological validity (Siedlecka & Denson, 2019). On the other hand, Virtual Reality allows researchers to study genuine states that are analogous to real-life fear – therefore achieving ecological validity, while retaining the crucial elements of experimental control. One piece of evidence that supports this claim comes from measuring self-reported "presence" during immersion in virtual reality simulations: by asking participants how present they feel, we can gauge the degree to which our paradigm is representative of the real context we are attempting to emulate, and thereby improve ecological validity (Felnhofer et al., 2015). Virtual Reality is a methodology that has not been used extensively to study HRV or emotion regulation. In Study 2 I begin to address this gap in the literature.

In this study, I draw on archival data that was collected in participants who experienced exposure to height in virtual reality. In the simulation, participants were seated for a five-minute baseline, and then were immersed in a VR environment in which they were exposed to extreme heights — participants were asked to walk along a plank suspended from the side of a skyscraper building. At several points throughout the procedure, participants provided ratings of their subjective emotion.

Ambulatory HRV and Ultra–Short Measurements

While virtual reality is an ideal methodology for ecological validity and experimental control, it presents some challenges for physiological data collection. For the study of fear, it is crucial that we allow the participant to be ambulatory and to have freedom in the virtual space to generate a sense of immersion and presence. Presence, specifically, is known to be a crucial component in the success of Virtual Reality in inducing emotional states (Riva et al., 2007). The requirement for free ambulation leads to two considerations with HRV data collection. The first is that cardiac vagal control is known to be highly dependent on posture, such that even the difference between sitting and standing can have very large effects on HRV measures. This is the reason that the so-called orthostatic test is often used in clinical assessments of autonomic function. In this test HRV is recorded during seated conditions and then while standing, to measure the autonomic response to postural changes. The implication of this postural effect is simply that the change between a seated baseline, and standing for subsequent VR time points must be accounted for, whereas in Study 1 we could simply compare measures between time points as participants stayed seated throughout.

The second consideration is that even using gold-standard and reliable ECG devices, the calculation of HRV metrics is very sensitive to artefacts caused by movement. The effect of movement on data quality is anticipated and controlled for: Study 2 is designed to measure HRV under stationary conditions at pre-specified time points at which emotion ratings are also collected. The drawback to this is that these time points only require the participant to be stationary for short periods of time (Study 2 was not designed for HRV analysis). Therefore, the maximum duration of stationary epochs for HRV calculation is 20 seconds in the present study design. The reliability of HRV metrics is known to vary as a function of epoch length. The research is clear that in general, the more beats the better the validity of HRV assessment. While five-minute measurements are considered the gold-standard for measuring HRV (Malik, 1999) there is also some available research using so-called ultra-short

measurements (sub-1-minute). The findings in this literature are mixed, leading to disagreement as to the shortest minimum epoch length that can reasonably be used. However, it seems likely that 10-20 seconds can be justified for RMSSD (Salahuddin et al., 2007; Nussinovitch et al., 2011; McNames & Aboy, 2006). HF power on the other hand may require longer epochs. Because it is a measure of the variability in the signal within a specific frequency band (frequency-domain) it requires a minimum number of cycles at that frequency to be informative. Some studies indicate that 20-second epochs are sufficient (Baek et al., 2015; Salahuddin et al., 2007; McNames & Aboy, 2006) while others disagree (Munoz et al., 1997; Shaffer et al., 2016). As noted in a recent review (Shaffer et al., 2020) most studies assessing ultra-short-term measurement for HRV measures do so by correlating them against standard 5-minute measurements, but without *a priori* specifying a minimum *r* value that would reflect acceptable agreement between measurements. Shaffer and colleagues found insufficient evidence to recommend ultra-short epochs for clinical practice; however, for within-subjects analyses they may be sufficient. It is therefore useful to test the sensitivity of ultra-short-term measurements of RMSSD and HF in detecting threat-related changes in cardiac vagal control.

Maymon et al. (manuscript in preparation)

Study 2 involved a set of exploratory analyses using previously collected ECG data from a study investigating ‘presence’ and fear in a novel Virtual Reality paradigm (Maymon et al., 2022, manuscript in preparation). The original study, which did not involve HRV analysis, was pre-registered at the Open Science Framework (https://osf.io/6s3mf/). The shared data include heart-rate data derived from ECG. Preliminary findings show that virtual height exposure elicited subjective fear, alongside increases in heart rate and skin conductance that indicate physiological arousal. Data were collected in this study that permit analysis of HRV Reactivity (although not Recovery), and therefore this dataset presents an

ideal opportunity to build on Study 1 by exploring HRV during exposure to a fear-inducing virtual reality paradigm.

The Present Study

In Study 2 I aim to extend on Study 1 by addressing the same three questions, to test whether the findings from Study 1 replicate and generalise to a different threatening context. The differences from my first study are that Study 2 uses height-exposure as opposed to a social-evaluative context, that the Epochs will be shorter and measurements while participants are standing (both due to necessity). Additionally, there is no between-subjects manipulation, and there is no Recovery epoch.

The first aim of Study 2 is to further address the question of how cardiac vagal control changes as a response to threatening contexts – by capturing these changes in response to heights-exposure and exploring whether the pattern of this response differs compared to the response to social evaluation in Study 1. If the hypothesis is correct that dynamic changes in cardiac vagal control (i.e. Reactivity) play a role in the threat response to heights, then we should observe a significant decrease in HRV during the threat relative to a non-threatening portion of the VR simulation. A repeated-measures ANOVA will be used to test this hypothesis: a significant effect of time should be observed on RMSSD and HF measurements of HRV during the threat, whereby RMSSD and HF are lower during threatening portion of the simulation relative to the non-threatening portion.

Secondly, I aim to test the relationship between Resting HRV and HRV Reactivity. If it is the case that higher levels of Resting cardiac vagal control are associated with a more adaptive Reactivity, due to greater capacity to self-regulate, then higher levels of RMSSD/HF at rest should be associated with smaller decreases during threat. This hypothesis will be tested by calculating Reactivity scores RMSSD and HF for each participant (negative values

indicate a decrease in HRV), and then using Baseline values to predict the change scores in a regression model. A significant positive relationship would support this hypothesis.

Method

Participants

Participants were 65 (42 female) undergraduate psychology students from Victoria University of Wellington, ages 18-53 (M =20.37, SD = 5.59), who took part in the study in exchange for course credit. Participants were right-handed, with no known hearing impairments, normal or corrected-to-normal vision, limited experience with VR, no current diagnosis of depression or anxiety, and no history of neurological disorder. Participants provided written informed consent prior to taking part. This study was approved by the Human Ethics Committee of the School of Psychology at Victoria University of Wellington (Approval number: RM0025873).

Questionnaires

All questionnaires were adapted for presentation using Qualtrics (Qualtrics, Provo, UT). The Discrete Emotions Questionnaire (DEQ; Harmon-Jones et al., 2016) was used to assess discrete emotional states before and after participants completed the VR simulation. The DEQ comprises 32 items and 8 emotion subscales (4 items per subscale): *Anger, Disgust, Fear, Anxiety, Sadness, Desire, Relaxation, and Happiness.* Participants indicate to what extent they are currently experiencing the emotions denoted by items on a 7-point scale.

The *Presence Questionnaire* (PQ; Witmer & Singer, 1998) was used to assess presence. The revised PQ (Witmer, Jerome, & Singer, 2005) comprises 24 items and 4 subscales: Involvement, Sensory Fidelity, Adaptation/Immersion, and Interface Quality. Participants characterize their experience in the virtual environment using a 7-point scale

based on the semantic differential principle, where each item is anchored at the ends by opposing descriptors based on question content.

A modified version of the Emotion Regulation Questionnaire (Gross & John, 2003) was used following the completion of the threat-exposure, as in Study 1. Following the Recovery period, the full 10-item scale was given as per the standardised method, except that participants were asked to reflect back to their time on the plank. This questionnaire therefore allowed for the measurement of self-reported suppression and reappraisal use while in the threat-exposure.

Emotion and Presence Ratings

Participants provided verbal ratings of the extent to which they were experiencing seven subjective states (anger, anxiety, sadness, relaxation, happiness, fear, and presence), on a 10-point scale (1: *Not at all;* 10: *Extremely)* at five locations during the VR simulation. These are taken from the DEQ, with presence substituted for "desire". The time points for emotion ratings were at: the curb, the bottom of the elevator, the top of the elevator, the start of the plank, and the end of the plank. Experimenters prompted participants to give ratings at each time point by asking: *'what is your fear/anxiety/happiness/etc. rating?'*. Importantly, "presence" was described as "the extent to which you feel like you are present and immersed in the virtual environment." Rating-order was randomised at each time-point, and the experimenter recorded responses in real-time using a Qualtrics survey on a smartphone.

Physiological Recording

Electrocardiography was recorded continuously using ADinstruments ML138 Octal Bio Amp using a 3-lead setup. Disposable adhesive silver/silver chloride (Ag/AgCl) ECG electrodes were placed below the right clavicle and lower left ribcage, referenced to the left

clavicle[20]. HR was determined by the inter-beat interval between consecutive R-wave spikes, converted to beats per minute (BPM). HRV measures were calculated in LabChart from the ECG signal (see below). SCL was recorded using ADInstruments bipolar dry stainless steel GSR electrodes (MLT116F) and an ML116 AC GSR amp, attached to the medial phalange of the index and ring fingers of the participant's left hand. ECG and SCL were converted from analogue to digital signals at 1 kHz, using ADInstrument's Powerlab 16/30, and recorded in LabChart 8.0.1. During the study procedure, a second experimenter was tasked with placing pre-set comments in LabChart to mark the onset of specific events. The nature of these events and associated time windows will be elaborated on in the data processing section below

VR System

The VR simulation for this study was taken from the VR game *Richie's Plank Experience* (ToastVR, 2017). The simulation is set in a busy city street. The simulation was presented via an HTC Vive VR headset, equipped with headphones providing three-dimensional audio, and participants used an HTC Vive controller in their right hand. Study 2 was conducted in a room that was 3.6 meters long and 2.9 meters wide. Two HTC Vive base stations were fastened to the opposite corners of the rectangular room, at a height of 2.44 meters, and were 4.62 meters apart. The VR system was driven by a PB Everyday Home PC with an ASUS ROG Strix GeForce GTX 1080 graphics card,110 GB SSDs, 16 GB RAMs; Intel Core i7-7700 CPU @ 3.60 GHz running Windows 10. A wooden plank was used for the height-exposure, which was 208 cm long, 18.5 cm wide, and 4.5 cm thick.

Procedure

[20] Note that the ECG apparatus is identical to that used in Study 1, while a different system was used in Study 3

The full procedure requires a lead experimenter (E1) who maintained nearly all of the interaction with the participant, and an assisting experimenter (E2) who was tasked with operating the PC running LabChart and the PC running the simulation. The entire procedure took place within a period of roughly an hour, in the following sequence:

The participant was first asked to respond to a series of questionnaires on a desktop PC running the online software Qualtrics. Following the completion of these, it was explained to the participant that the study will involve them walking along a plank and that a physical plank would be included to make the task more realistic. The participant was asked to remove their shoes and to complete a practice walk of the plank (ensuring balance). The plank was removed, and the participant was instructed on how to attach the ECG torso and SCL finger electrodes. A 5-minute seated resting baseline of the physiological measures was recorded, with instructions given for participants to remain still for the baseline period, breathe naturally, and to no use their cell phone or other devices.

The headset is fitted to the participant, as well as a controller in their right hand. The initial experience of the VR environment was a city-street scene, at which point they are asked to walk to the curb to explore the environment, and to look around as they go. A first set of verbal emotion ratings was acquired at the curb (Curb epoch). The participant was directed to walk back into the elevator where a second set of ratings is acquired (Bottom of Elevator epoch). Here, participants were informed via the headphones in the VR headset that they would see a virtual wooden plank when they reached the top, which "corresponds to the real plank" they walked on earlier. At the instruction of Experimenter 1, the participant pressed a red button on the elevator panel which begun the elevator's ascent. The doors to the elevator opened, revealing that they were at the top the building, and that the plank stretched out in front of them — off the side of the building. While still standing in the elevator, a third set of ratings (Top of Elevator epoch) was taken. Then, they were instructed to step onto the plank with both feet,

and to remain there while the fourth set of ratings was taken (Start of Plank epoch). Experimenter 1 then prompted them to walk to the end of the plank when they were ready. Upon completion of the plank walk, one final set of ratings is taken as they stood at the end of the plank (or at whichever point they reached, for those who did not complete). Experimenter 1 then gave the participant the option to either step off the side of the plank if they so wish (in which case they would experience a falling simulation) or to be "teleported" back to the street (in which case the VR would fade to black and they would reappear back at ground floor level).

Experimenter 1 then removed the headset and asked the participant to complete the post-VR questionnaires, plus some questions regarding participants' previous experiences using VR .

Data Processing

For the purposes of this thesis, descriptions of data processing and analysis will focus on data from physiological measures, fear ratings and modified ERQ. ECG and SCL channels were analysed in LabChart software, with these signals processed visually by the author.

Custom-made macros were run in LabChart to select eight epochs of data in succession. The first was the resting *"Baseline"*, which was taken outside of VR. This epoch was a standard duration of 260s, as the first and last 20 seconds of the 5-minute resting period were removed due to a prevalence of movement artifacts during these periods. This epoch is longer because a stable measurement of Resting HRV was desired. The following five epochs were extracted from the periods during which the participant was stationary while providing verbal emotion ratings in VR. These were standardised to 20s in length, as this was the maximum possible duration which included data from all participants. The selection began at a marker which was manually inputted by E2 in LabChart, which indexed the time at which E1 initiated the collection of ratings, and extended for 20s from there. The data across the

three channels was then visually inspected for each epoch and used to calculate cardiac variables following the same method as described in Study 1.

Results

Manipulation Check

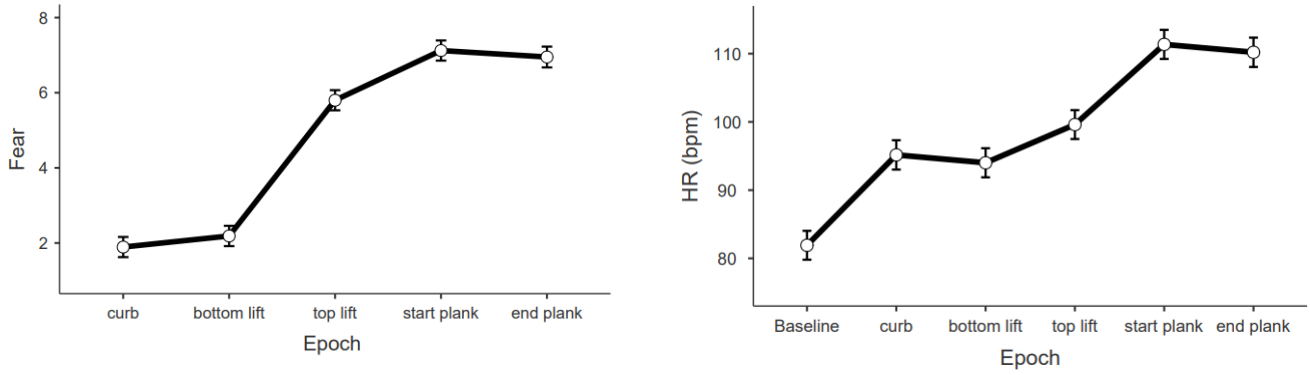

Figure 13: Fear ratings (left) and heart rate in bpm (right) by epoch
Plots showing time course of subjective fear ratings (left) taken during VR, measured on a 1-10 scale of intensity, and heart rate in bpm (right), as a function of Epoch (Baseline, Stress, recovery).

The exposure to heights in VR was shown to be an effective induction of physiological and subjective emotional arousal, see figure 14 above.

Time Course — lnRMSSD

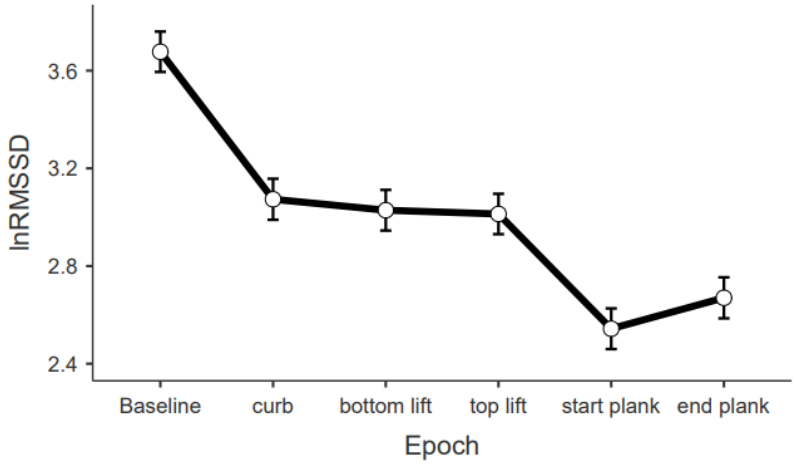

Figure 14: lnRMSSD by epoch

RMSSD was found to violate the assumption of normality even following natural log transformation as indicated by a significant Shapiro-Wilk test at the Curb, Top of Elevator, and Start of Plank epochs. A repeated-measures ANOVA using the Greenhouse-Geiser correction for non-sphericity was performed on all six time points to test the effect of time on lnRMSSD[21]. A significant effect of time was found, $F(3.34, 57) = 40.4$, $p < .001$, $\eta_p^2 = .415$. Post-hoc comparisons using Tukey's HSD method confirmed that RMSSD changed during the VR procedure, and was significantly lower at both plank epochs relative to Curb epoch. RMSSD decreased from the Baseline ($M = 3.68$, $SD = 0.537$) to Curb epochs ($M = 3.073$, $SD = 0.559$), as expected due to the change from sitting to standing. RMSSD did not change from the Curb to the Bottom of Elevator epochs ($M = 3.029$, $SD = 0.678$), or from the Bottom of Elevator to the Top of Elevator epochs ($M = 3.013$, $SD = 0.758$). lnRMSSD values decreased from the Top of Elevator to the Start of Plank epochs ($M = 2.543$, $SD = 0.758$) — the greatest magnitude difference observed in the time course of the study. Although visual inspection suggests a rise in lnRMSSD when participants were at the end of the plank, comparisons revealed no difference from the Start of Plank epoch to the End of Plank epoch ($M = 2.67$, $SD = 0.751$).

For a more sensitive test of the effect of height exposure on lnRMSSD, another repeated-measures ANOVA was performed on four time points (Bottom of Elevator, Top of Elevator, Start of Plank, and End of Plank), from which individual's Curb values had been subtracted. This subtraction controls for any potential effect of VR itself on autonomic arousal, which would be unsurprising due to the novelty of being in a simulation. Negative values therefore represent a decrease in HRV at each timepoint, with more negative values indicating greater decreases in HRV. The Greenhouse-Geisser correction was used on this

[21] A non-parametric version of this test, the Friedman's ANOVA (which uses median values to minimise the influence of deviations from normality), was run in the interest of thoroughness, and the results were the same.

analysis due to non-sphericity. A significant effect of time was found, F(3, 171) = 16.3, p <.001, η_p^2 = .223. Post hoc Tukey's tests found no difference in vagal withdrawal between the Bottom (*M* = - 0.0388, *SD* = 0.623) and Top of Elevator epochs (*M* = -0.0623, *SD* = 1.69), but a significant difference between the Top of Elevator and Start of Plank (*M* = -0.526, *SD* = 0.725) epochs. The latter difference represents a substantially lower HRV when participants were standing at the beginning of the plank prior to walking to the end. There was no difference between HRV measurements at the Start and End (*M* = -0.4032, *SD* = 0.728) of Plank epochs. Both plank time points showed a significantly greater degree of vagal withdrawal when compared to the bottom of the elevator (*p* < .001).

Time Course — lnHF Power

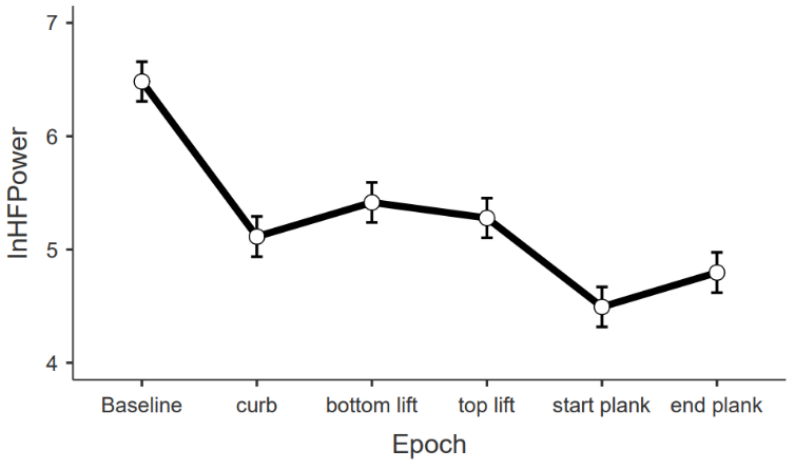

Figure 15: lnHF in ms2 by epoch

HF power in ms^2 deviated from a normal distribution at all 6 time points prior to log transformation. A repeated-measures ANOVA using the Greenhouse-Geiser correction for non-sphericity was performed on all six time points to test the effect of time on lnHF. A significant effect of time was found, F(3.86, 57) = 22.7, *p* <.001, η_p^2 = .285. Post-hoc comparisons using Tukey's HSD method confirmed that lnHF values decreased from Baseline (*M* = 6.48, SD = 0.919) to Curb (*M* = 5.11, SD = 1.29, *P*tukey < .001) epochs, but did not change from the Curb to the Bottom of Elevator epoch (*M* = 5.42, SD = 1.37) or from

the Bottom to the Top of Elevator epochs (*M* = 5.28, SD = 1.31). lnHF values showed a decrease from the Top of Elevator to the Start of Plank epoch (*M* = 4.49, SD = 1.80, *P*tukey = .025), which was the greatest observed change in lnHF through the paradigm. There was no difference from the Start of Plank epoch to the End of Plank (*M* = 4.80, SD = 1.51) despite the visual inspection suggesting a slight increase. lnHF values at the Curb epoch were not significantly different from either the Start, or the End of Plank epochs, contrary to the difference seen in lnRMSSD data. This surprising null effect here suggests insufficient evidence to conclude that HF (frequency domain measure) is strongly decreased by threat, while a strong effect is seen in lnRMSSD (time domain measure).

For a more sensitive test of the effect of height on lnHF, another repeated-measures ANOVA was performed on four time points (bottom of elevator, top of elevator, start of plank, and end of plank), from which individual's curb values had been subtracted. This subtraction was performed to control for the effect of the VR environment on autonomic arousal. Negative numbers therefore represent a withdrawal of cardiac vagal control which can be attributed to the fear manipulation, with more negative numbers indicating greater withdrawal. Another Greenhouse-Geisser corrected repeated-measures ANOVA was employed for this purpose. A significant effect of Epoch was found, $F(3, 168) = 7.15$, $p < .001$, $\eta_p^2 = .113$. Post hoc Tukey's tests found no difference in Curb-controlled lnHF values between the Bottom of Elevator (*M* = 0.32, *SD* = 1.33) and Top of Elevator epochs (*M* = 0.172, SD = 1.25). A significant difference was however observed between the Top of Elevator and Start of Plank epochs (*M* = -0.518, *SD* = 1.69). This significant difference further corroborates the finding from the other ANOVAs showing that HRV does in fact decrease when participants are exposed to heights, even when controlling for Curb levels of HRV. There was no difference between Start and End (*M* = -0.345, *SD* = 1.28) of Plank

epochs. Both the Start and End of Plank time points however did show a significantly greater degree of vagal withdrawal when compared to the bottom of the elevator.

HRV Reactivity

HRV *Reactivity*Δ consists of *change* scores for both lnRMSSD and lnHF as used above, specifically the score created by subtracting values at the Curb epoch from values at the Start of Plank epoch. Therefore, positive numbers on this measure indicate that HRV increased from the Curb to the Start of Plank Epoch, whereas negative values indicate a decrease across the same two measurements. As indicated in the above analysis, reactivity was shown to typically involve a decrease for lnRMSSD (M = -0.526, SD = 0.725) and lnHF (M = - 0.602, SD = 1.79). These reactivity measures differed from normality due to a strong left skew where a few participants had dramatic decreases and most had small decreases (and on occasion, increases). However, HRV ReactivityΔ scores on both measures are evidence of a substantial withdrawal of cardiac vagal control during height-exposure.

A hierarchical design was used to predict HRV ReactivityΔ using linear regression modelling. Models predicting lnRMSSD ReactivityΔ are described and reported here (see Appendix for models using lnHF). Two linear regression models were employed. *Model 1* predicted lnRMSSD ReactivityΔ from Baseline lnRMSSD (resting HRV); *Model 2* entered Suppression and Reappraisal use (averaged scores from the ERQ subscales).

Model 1, predicting lnRMSSD ReactivityΔ from Baseline lnRMSSD failed to account for a significant amount of variance, R^2adj. = .028, $F(1,57) = 2.66$, $p = .108$. The addition of Suppression and Reappraisal scores in Model 2 did not significantly improve the model ($R^2\Delta = .023$, $F(2,55) = .688$, $p = .507$); Model 2 did not account for a significant amount of variance in lnRMSSD reactivity, R^2adj. = ., $F(3,55) = 1.34$, $p = 2.72$, and an increased BIC

relative to Model 1 (139 to 146) indicates a worse model fit upon the addition of strategy use as additional predictors.

These analyses found no evidence for a relationship between Resting and Reactivity levels of HRV in a height-exposure paradigm. Further, no relationship was found between use of emotion regulation strategies and HRV Reactivity. This was a surprising null finding which deviates from predictions.

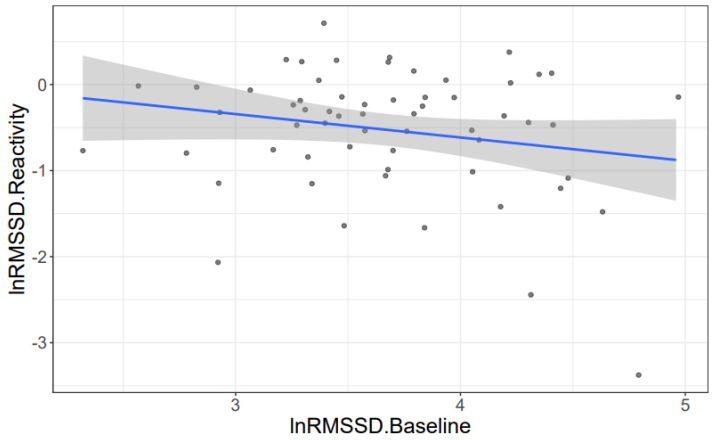

Figure 16: Scatterplot, Baseline lnRMSSD predicting lnRMSSD Reactivity
Depicts fit of data predicting HRV Reactivity, as measured by log transformed RMSSD change scores (Start of Plank epoch – Curb epoch), using log transformed RMSSD at Baseline as a predictor (a measure of Resting HRV).

Interim Summary

Results from Study 2 demonstrate that HRV shows significant decreases during threat in a virtual reality height-exposure paradigm. This decrease appears to be robust across both time domain and frequency domain (RMSSD and HF power) metrics, consistent with the hypothesis that cardiac vagal control withdrawal is a part of the response to threat. These findings are of interest as they demonstrate that HRV can be used to detect autonomic correlates of fear, when fear is induced in ambulatory participants in virtual reality. The data in Study 2 suggest that ECG measurements as short as 20 seconds may be sufficient to assess these changes in HRV, a claim that has been subject to controversy among researchers. These

data are therefore helpful in addressing methodological considerations for future research using HRV in psychology.

Interestingly, despite the relationship found between HRV Resting and Reactivity measurements in Study 1, no such relationship emerged in the data of Study 2. Not only did Resting HRV not predict HRV reactivity (the change from Curb to Start of Plank epochs), but it also did not predict *absolute* levels of HRV at the Start of Plank epoch (see table 10 below). Although Study 1's results in this regard were opposite to the predicted relationship, they can be explained in terms of alternative theoretical accounts (i.e. Porges' Polyvagal theory). The null relationship observed in Study 2 leads to three speculative explanations. The first of these is that the relationship differs for a strong fear induction relative to the former study using a social-evaluative context. The second is that perhaps the relationship is moderated or mediated by an unknown variable that was not considered, which explains why some participants exhibit and increase, and some a decrease, in HRV during height-exposure. The third explanation is simply that the HRV measurement used in Study 2, while sufficient to detect threat-induced changes in HRV, are not adequate to fully capture the dynamic between-subjects effects needed to illuminate the resting-reactivity relationship. A key factor in support of this latter idea is that the measurement for Resting HRV was 5 minutes in length, while the measurements for reactivity were only 20 seconds. It seems possible that this methodological/statistical flaw could explain the lack of relationship seen here, while it was seen in Study 1 where both resting and Reactivity HRV measures were created using 5-minute measurements.

Further, none of the variance in HRV Reactivity was explained by the self-reported use of either Suppression or reappraisal strategies during the plank walk. However, Suppression use was shown to independently predict absolute levels of HRV during height-exposure (as opposed to change from Curb), such that higher levels of Suppression use

predicted higher levels of HRV while at the Start of Plank timepoint. Interestingly, no such relationship was found for Reappraisal use. While the planned regression modelling revealed no evidence in support of the Regulation hypothesis, this exploratory analysis did provide limited support, indicating that greater use of a response-focused strategy is associated with higher cardiac vagal control during threat.

Table 7: Descriptive Statistics

Variable	*N*	Mean	SD	Min.	Max.	Skewness	Kurtosis	Shapiro-Wilk *p*
Resting	63	3.678	0.537	2.320	4.959	0.0183	-0.009	.965
Height	62	2.54	0.758	0.730	3.83	-0.598	-0.131	.033
Reactivity	60	-0.526	0.725	-3.375	0.714	-1.4798	3.407	<.001
ΔFear	60	4.508	2.042	0.333	8.500	-0.2394	-0.691	0.116
Supp.Avg	65	2.912	1.155	1.000	5.750	0.1553	-0.573	0.134
Reapp.Avg	64	4.352	1.422	1.000	6.667	-0.4769	-0.2596	0.072

Note: Resting and Height refer to lnRMSSD during Baseline and Start of Plank time points. Reactivity is the change in lnRMSSD calculated (Start of Plank minus Curb).

Table 8: Correlation Matrix

	Resting	Height	Reactivity	ΔFear	Supp.Avg	Reapp.Avg
Resting	—	—	—	—	—	—
Height	.156	—	—	—	—	—
Reactivity	-.206	.717***	—	—	—	—
ΔFear	-.002	-.238	-.194	—	—	—
Supp.Avg	-.010	.345**	.150	-.153	—	—
Reapp.Avg	-.097	.118	.025	.106	.144	—

Note: Pearson's *r* reported in table. * $p < .05$, ** $p < .01$, *** $p < .001$

Predicting HRV Reactivity

Table 9: Model 1 - Resting lnRMSSD

Coefficient	Estimate	SE	*p*-Value
Intercept	.499	.637	.437
Resting lnRMSSD	-.281	.172	.108

Table reports unstandardised coefficients. $F(1,57) = 2.66$, $p = .108$. $R^2 = .045$, R^2adj. = .028, BIC = 139.

Table 10: Model 2 – Adding Suppression and Reappraisal during Plank-Walk

Coefficient	Estimate	SE	*p*-Value
Intercept	.265	.761	.729
Resting lnRMSSD	-.282	.174	.110
Supp.Avg	.095	.081	.246
Reapp.Avg	-.008	.071	.908

Table reports unstandardised coefficients. $F(3,55) = 1.34$, $p = .272$. $R^2 = .068$, R^2adj. = .017, BIC = 146.

Model 1 – 2 comparison: $R^2\Delta = .023$, $F(2,55) = .688$, $p = .507$.

Predicting HRV *During* Height-Exposure

Table 11: Model 1 - Resting lnRMSSD

Coefficient	Estimate	SE	*p*-Value
Intercept	1.742	.673	.012
Resting lnRMSSD	.218	.182	.235

Table reports unstandardised coefficients. $F(1,59) = 1.44$, $p = .235$. $R^2 = .024$, R^2adj. = .007, BIC = 150.

Table 12: Model 2 – Adding Suppression and Reappraisal during Plank-Walk

Coefficient	Estimate	SE	*p*-Value
Intercept	.835	.755	.273
Resting lnRMSSD	.229	.173	.191

Supp.Avg	.219	.080	.008**
Reapp.Avg	.051	.068	.460

Table reports unstandardised coefficients. $F(3,57) = 3.41$, $p = .024$. $R^2 = .152$, R^2adj. = .107, BIC = 150.

Model 1 – 2 comparison: $R^2\Delta = .128$, $F(2,57) = 4.31$, $p = .018$.

Study 3: An Experimental Manipulation of Emotion Regulation

The Present Study

Having initially explored the dynamic changes in HRV that occur in response to VR height-exposure in Study 2, in Study 3, I seek to replicate those findings and extend on them, with the addition of an experimental manipulation of emotion regulation strategy. Study 3 is part of a larger collaborative project studying the effects of emotion regulation strategies on fear[23] (larger project pre-registered at https://osf.io/z42th/). The aim of this experiment was to test the relative effects of Suppression and Reappraisal on measures of negative emotion during Height-Exposure in Virtual reality, using a variation on the now-validated fear-induction paradigm used in Study 2.

The same three research questions are being assessed in this study as in Studies 1 and 2: 1) how does HRV change during threat, 2) how are resting HRV and HRV reactivity related, and 3) how do emotion regulation strategies modulate HRV reactivity. The major difference is that a between-subjects experimental manipulation of emotion regulation strategy is added, in which participants are randomly assigned to either an instructed suppression, reappraisal, or control group. This manipulation is designed to further address question 3. Some minor methodological improvements are also incorporated to address limitations of Study 2. Main analyses for Study 3 — specific to HRV data — are preregistered at: https://osf.io/8maqw. Analyses that are not preregistered will be clearly demarcated as exploratory. The planned analyses are identical to that of Study 2, except for the accommodation of tests for interactions between experimental condition and HRV.

[23] While Study 3's data comes from a larger collaboration, I largely collected the data (alongside other researchers) and processed/analysed the physiological data. Manuscript in preparation as of this writing.

specifically, an interaction between condition and epoch should be seen if instructed use of emotion regulation strategies modulates dynamic changes in HRV.

Ambulatory HRV Assessment

Study 3 was designed with HRV analysis in mind, and so some specific changes were made. For one, because Study 2 was not designed for the measurement of HRV specifically, it did not include a recovery period following the completion of the Virtual Reality experience – in Study 3 this will be part of the study design. Further, in Study 3, 30 second epochs will be used for HRV analysis to improve sensitivity to capture within-subject changes. Finally, ambulatory assessment of ECG proved difficult in Study 2, as a conventional 3-lead ECG approach restricts movement and has the potential to break a participant's sense of presence in the virtual environment. Therefore, it is desirable to have a wearable sensor that allows freedom of movement, while capturing high quality data. This requires an ECG-recording device that it is wireless[24]. One such device is the *Equivital Lifemonitor 2,* which has been shown to have accuracy in measuring HRV under ambulatory conditions (Liu et al., 2013), comparable to Holter ECG, the gold-standard equivalent used in clinical cardiology. This is the device used in Study 3, and it has the added benefit of recording respiratory data via stretch sensors in the chest strap.

Method

Design

Study 3 employed a mixed, three-group design with condition (Suppression, Reappraisal, and Control) as a between-subjects variable and time (Curb, Outside VR, Bottom of Elevator, Top of elevator, Start of Plank, and End of Plank) as a within-subjects

[24] We tested a research-grade version of a photoplethysmography device in Study 2, the E4 device (Empatica, Milan, Italy), and found that the data were not comparable or accurate for HRV measurement

variable. In addition, two 5-minute epochs of seated ECG were collected for use in regression analyses (Resting and Recovery).

Participants

Participants were 99 (74 female, 23 male, 2 non-binary) undergraduate psychology students from Victoria University of Wellington, ages (*Mdn* = 18, *M* = 20, *min* = 17, *max* = 50) took part in the experiment in exchange for course credit. The same inclusion criteria were used as in Study 2 (i.e. ability to use a mouse, adequate mobility, normal or corrected-to-normal vision, English speaker, usage of VR technology not exceeding a weekly basis, no history of migraine or vertigo). Participants provided written informed consent prior to taking part. This study was approved by the Human Ethics Committee of the School of Psychology at Victoria University of Wellington (Approval number: 0000025873.v2; approval date 04.03.2021).

Questionnaires

As in Study 2, momentary/state-level emotion ratimgs were obtained at the specified epochs. The *Emotion Regulation Questionnaire* (Gross & John, 2003) was administered both prior to, and following the completion of, the VR procedure. In the first instance the scale was used as a trait-level assessment, while the post-VR administration was adapted such that it was specific to their time on the plank. The post-VR ERQ measure will further serve to test the integrity of the strategy manipulation, that is, whether the instructions to use expressive suppression or cognitive reappraisal were followed and if they elicit behavioural differences that can be captured by the ERQ.

Procedure

The VR simulation was created in Unity and is inspired by the commercially available VR game *"Richie's Plank Experience"* used in Experiment 2. The simulation was made to be

slightly more realistic than the one used in Experiment 2, but otherwise only changed to add instructions for the emotion regulation manipulation. The simulation is run in Steam VR, on the same PC described in Experiment 2. The commercially available wireless HTC Vive Pro headset was used for the VR, using two base stations mounted on the walls of the VR laboratory in the School of Psychology at Victoria University.

The procedure is largely consistent with that of Study 2. In Study 3, participants were randomly assigned to one of three experimental conditions: *suppression*, *reappraisal*, or *control*. These differed on the experimental manipulation of emotion regulation strategy instructions. All participants experienced an identical procedure apart from the instructions which were given auditorily while ascending in the elevator. Importantly, prior to entering the VR headset, participants are given a backpack to wear and told that it contains the recording equipment for the physiological recording. The backpack is of relevance to the reappraisal condition, but all participants wear it for consistency.

Participants in the *Cognitive Reappraisal Condition* heard the following instructions: "Welcome to Victoria Towers. When the doors open, you will see a plank extending from the elevator floor, 80 stories above the street. Your task is to walk to the end of the plank, and as you do so, please try to imagine that the backpack you are wearing is actually a jetpack which will automatically save you if you fall. I'll repeat that for you again. Your task is to walk to the end of the plank, and as you do so, please try to imagine that the backpack you are wearing is actually a jetpack which will automatically save you if you fall. The experimenter will now ask you to provide some ratings for how you are currently feeling."

Participants in the *Expressive Suppression Condition* heard the following instructions: "Welcome to Victoria Towers. When the doors open, you will see a plank extending from the elevator floor, 80 stories above the street. Your task is to walk to the end of the plank, and as

you do so, please try to behave in such a way so that anyone watching you would not be able to tell what you are feeling. I'll repeat that for you again. Your task is to walk to the end of the plank, and as you do so, please try to behave in such a way so that anyone watching you would not be able to tell what you are feeling. The experimenter will now ask you to provide some ratings for how you are currently feeling."

Participants in the *Control Condition* heard the following instructions: "Welcome to Victoria Towers. When the doors open, you will see a plank extending from the elevator floor, 80 stories above the street. Your task is to walk to the end of the plank. I'll repeat that for you again. Your task is to walk to the end of the plank. The experimenter will now ask you to provide some ratings for how you are currently feeling."

As in Experiment 1, the procedure is run by two experimenters. In Experiment 2, all but ~5 of the data collection sessions were run with the author as Experimenter 1, while one of four colleagues filled the role of Experimenter 2 for any given session. This consistency in Experimenter 1 was intended to minimise bias due to differences while interacting with participants across sessions. Further, Experimenter 1 was blinded to the condition of every participant — instructions presented to the participant through the VR headset were not audible to the experimenters, and all precautions were made to avoid Experimenter 1 being exposed to the condition of each participant. This was done to minimise the possibility of experimenter bias affecting the data, as Experimenter 1 continuously interacts with the participant and verbally elicits subjective ratings.

As this experiment used an ambulatory Equivital recording vest, measurements were obtained of the height and chest circumference of the participant, in order to properly fit them with an Equivital recording belt. This was done following the practice walk of the plank. A belt was then set up for them by Experimenter 2 (i.e. dampening the electrode pads, inserting a data

recording module, and connecting the GSR cord) and they were asked to change into the belt while the experimenters left the room. Once this is correctly fitted, LabChart is started so that data begins to be streamed via Bluetooth to the PC.

As opposed to study 2, when participants reached the end of the plank, no falling simulation was implemented in this study if participants chose to step off the plank, a change which avoids some participants experiencing differential levels of threat. The other change to the procedure in study 3 was the addition of a recovery period after the participant left the VR simulation. This consisted of a 5-minute duration immediately following the termination of the VR simulation. Participants were seated and given the same instructions as during the Baseline recording.

Psychophysiological recording

To address some of the limitations in the physiological recording equipment in Study 2 a wireless ambulatory system was used in Study 3. Participants were fitted with the Equivital Lifemonitor belt (ADinstruments), which records a two-channel ECG (averaged from two combinations of three electrodes which are embedded in the belt) at a sampling rate of 256hz. A chest circumference measure was obtained from each participant to ensure an optimal fitting belt was selected. This is fitted around the torso and has electrode pads built in for ECG recording, which were sprayed with a saline mixture prior to the participant putting it on, in order to enhance conductivity and therefore provide a cleaner ECG signal. An additional module in the Equivital system recorded SCL, with wet-gelled electrodes connected to the thenar and hypothenar eminences of the participant's left hand. SCL was recorded at a sampling rate of 4hz. Respiration data were also recorded, as the Equivital belts measure chest expansion. These four channels of data were recorded continuously and connected via Bluetooth to a PC running LabChart software for real-time monitoring.

Experimenter 2 used pre-set comments to mark events in LabChart during the simulation as in Study 2.

Data processing

The ECG data were processed using the same techniques described in Experiment 1 to derive HR and HRV indices. 30 second epochs were analysed from the periods where participants are providing verbal emotion ratings, in addition to the baseline and recovery periods (260s each). SCL was also recorded for each of these epochs, as was respiratory rate (in breaths per minute) and respiratory maximum (the peak inhalation).

Results

Exclusions

One participant from the Reappraisal condition had ECG artefacts that were substantial enough to preclude HRV analysis for all epochs, and one participant from the Control condition is missing data for all epochs except for Baseline and recovery for the same reason. Data from all other participants is included in all analyses reported here (see table 12 below).

Epochs

Data were extracted from 8 epochs which subdivided continuous electrocardiography, respiratory, and skin conductance recordings. The epochs consisted of a 260s Baseline and a 260s Recovery at the beginning and end of the procedure, plus six ratings epochs of 30s each: Outside VR, Curb, Bottom of Elevator, Top of Elevator, Start of Plank, and End of Plank.

Manipulation Checks

First, I used ANOVA models to test whether the experimental, between-subjects manipulation of emotion regulation strategy was effective. An ANOVA was used with

condition (Control, Suppression, Reappraisal) as the between-subjects variable to test for differences in two dependent variables: self-reported use of Suppression and Reappraisal while on the plank. If participants faithfully followed instructions, we would expect those in the Suppression group to report higher use of suppression use, and those in the Reappraisal group to report higher levels of reappraisal use, relative to the other two groups. Examining self-reported suppression use between conditions I used a one-way ANOVA, which revealed a significant effect of Condition, $F(2,94) = 3.21$, $p = .045$, $\eta_p^2 = .064$. Post-hoc tests indicate that the instructed Suppression condition was higher in self-reported suppression relative to control as expected (Tukey's corrected $p = .051$). Likewise, examining self-reported reappraisal use between conditions, a one-way ANOVA found no evidence for a group difference. This lack of concordance between assigned strategy and self-reported strategy use is noteworthy and will be discussed later. Using similar ANOVAs, I also tested whether trait levels of self-reported reappraisal and suppression differed between groups. Neither ANOVA showed a significant effect of group, indicating that random assignment achieved an even distribution of these traits.

As a second manipulation check, I tested whether the within-subject threat manipulation was successful in inducing a state of high emotional arousal. Emotional arousal is demonstrated by strong increases in subjective ratings of fear, in skin conductance level, and heart rate (see figure 18 below).

heart rate.

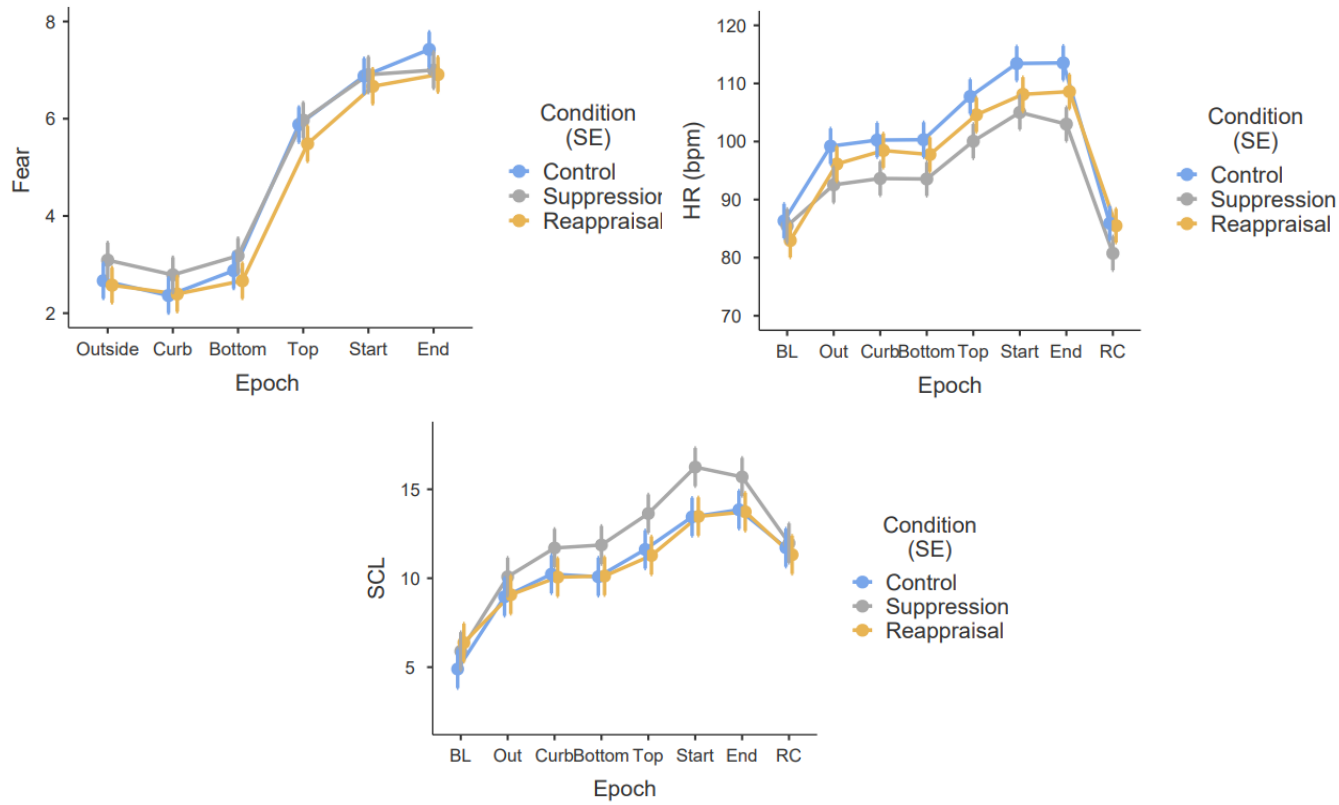

Figure 17: *Fear ratings (left), skin conductance level (bottom) and heart rate (right) by epoch, split by condition*

Time Course – lnRMSSD

A repeated-measures ANOVA using the Greenhouse-Geisser correction was used to test the effect of Epoch on lnRMSSD data across all 8 epochs, showing a significant effect, $F(4.76, 2) = 90.9$, $p < .001$, $\eta_p^2 = .511$. Entering condition into this model did not yield significant between subject ($p = .104$) or interaction effects [$F(9.52) = 1.30$, $p = .231$, $\eta_p^2 = .029$].

Tukey's corrected post-hoc tests show a large decrease in HRV from Baseline ($M = 3.60$, $SD = 0.735$) to Outside VR ($M = 3.06$, $SD = 0.60$). Further contrasts showed no consecutive change from outside VR to Curb ($M = 3.03$, $SD = 0.641$), but a substantial decrease in lnRMSSD occurred from the Bottom of Elevator epoch ($M = 3.02$, $SD = 0.727$) to

the Top of Elevator (M = 2.81, SD = 0.69; p = .003). Subsequently, lnRMSSD did not change from the Start of Plank (M = 2.76, SD = 0.776) to End of Plank (M = 2.78, SD = 0.742) epochs. A large increase in lnRMSSD was shown between the End of Plank and Recovery (M = 3.59, SD = 0.684), the reverse of the effect from Baseline to Outside VR Epochs — primarily reflecting orthostatic influences. It is interesting that the post-hoc contrasts find that the biggest change in cardiac vagal control occurred in the difference from the Bottom of Elevator to the Top — and not from the Top of Elevator to the Start of Plank (as was seen in Study 2).When comparing the Curb values of lnRMSSD (representing the HRV while at street-level and inside VR), to subsequent Epochs (at height), lnRMSSD was significantly lower at the Top of Elevator, Start, and End of Plank Epochs (all ps < .001) — an observation that indicates a significant impact of height-exposure on cardiac vagal control[25]. Baseline and Recovery epochs were not significantly different (p = .996), indicating that on average, participant's HRV recovered to resting levels following the virtual reality component of the study.

The effect of time on lnRMSSD was tested using a Huynh-Feldt[26] corrected repeated-measures ANOVA, this time using the Bottom and Top of Elevator, Start and End of Plank time points from which individual's Curb values had been subtracted. This was to control for the effect of VR and examine more closely the effect attributable to the fear induction. This test yielded a significant effect of time, F(2.77, 2) = 12.29, p < .001, η_p^2 = .118. Post-hoc tukey's tests showed a significant decrease in curb-controlled HRV from the Bottom (M = -0.0121, SD = 0.415) to the Top of Elevator (M = -0.221, SD = 0.303), but no consecutive differences from Top of Elevator to Start of Plank (M = -0.288, SD = 0.451), or from Start of

[25] The same differences were seen from Bottom of Elevator, ps = .003, .002, and .001 respectively

[26] The epsilon value for this test was higher/closer to 1 than for the other ANOVAs, indicating that repeated-measures exhibited less non-sphericity (although still violating the assumption) — justifying the use of a less conservative adjustment of degrees of freedom

Plank to End of Plank (*M* = -0.256, *SD* = 0.432). Curb-controlled HRV was higher at the Bottom of Elevator relative to all subsequent epochs (all *p*s < .001), providing further evidence in favour of an effect of height on HRV. Entering condition into this model did not yield a significant interaction effect of Epoch by Condition, (*p* = .296), however, there was a significant between subjects effect of condition however, F(2.7, 2) = 3.62, *p* = .031, η_p^2 = .073. The main effect is not relevant for hypotheses however, as any significant changes driving this result occur prior to the participants' receiving their emotion regulation instructions, and so are differences not attributable to the manipulation.

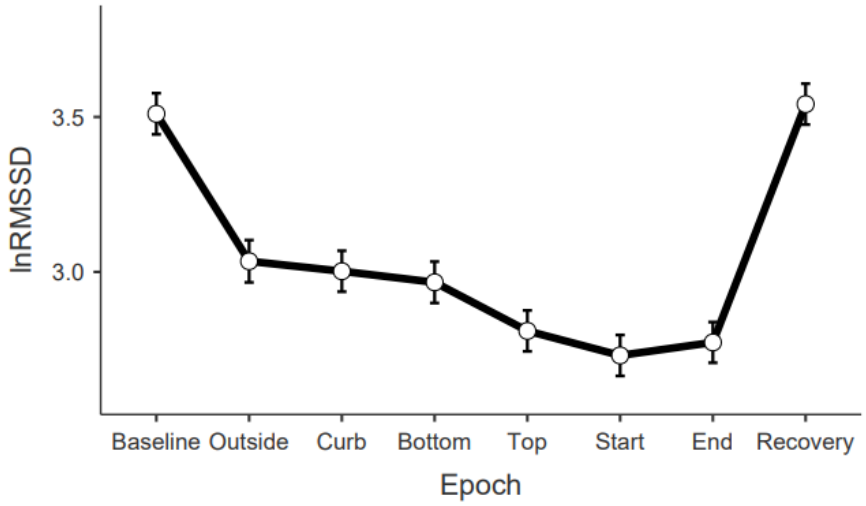

Figure 18:lnRMSSD by epoch

Time course of log transformed RMSSD data, using mean scores of whole sample (n = 99), with error bars depicting standard error of the mean.

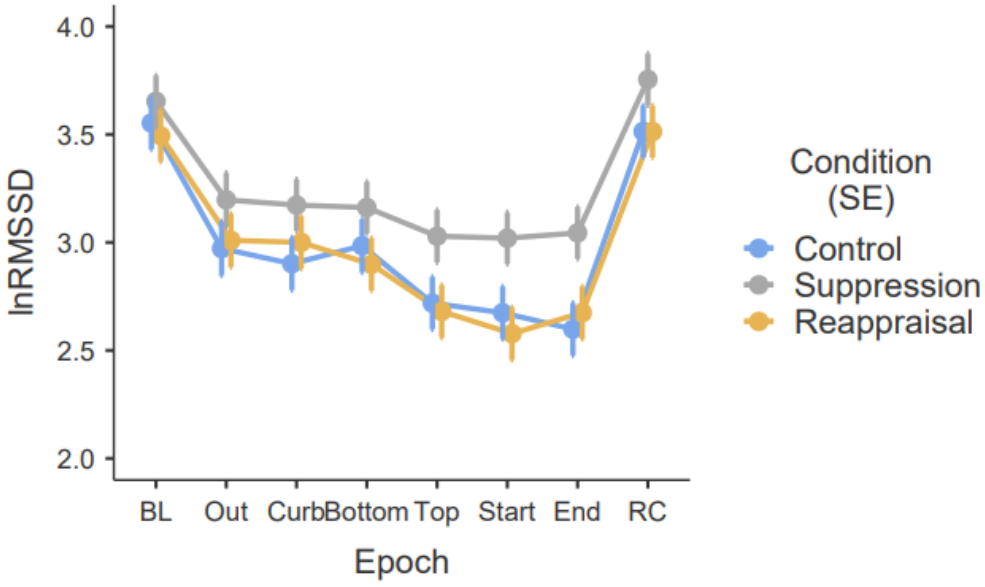

Figure 19: lnRMSSD by epoch, split by condition

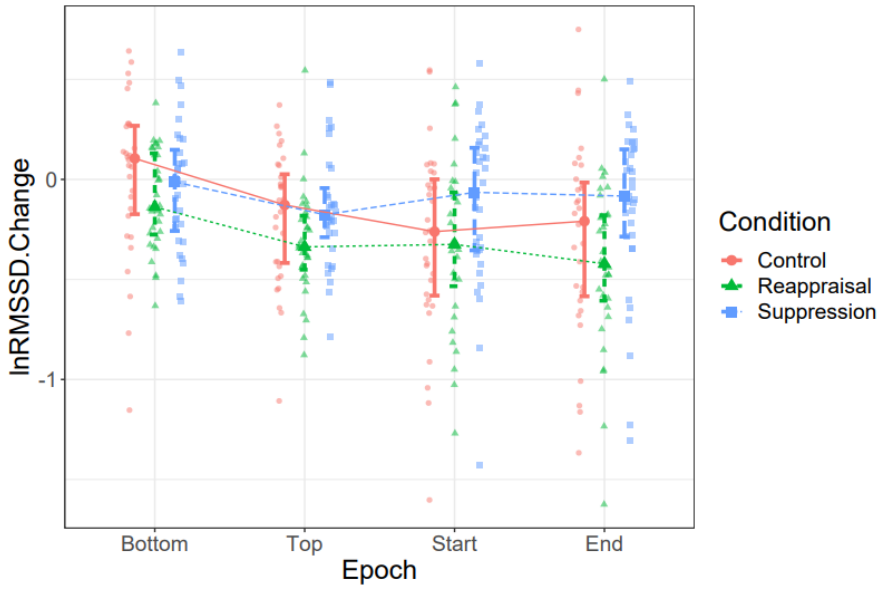

Figure 20: lnRMSSD change by epoch, split by condition
Time course of log transformed RMSSD data, as change scores (Bottom of Elevator, Top of Elevator, Start of Plank, End of Plank) with Curb values subtracted from each. Observed values plotted with standard error bars

Time Course – lnHF

A repeated-measures ANOVA using Greenhouse-Geisser correction was conducted on lnHF power data to test the effect of time across all 8 time points, showing a significant effect, $F(5.25, 2) = 56.01$, $p < .001$, $\eta_p^2 = .392$. Tukey's corrected post-hoc tests showed only

two significant changes between consecutive Epochs: a decrease from Baseline (*M* = 6.41, *SD* = 1.40) to Outside VR (*M* = 5.40, *SD* = 1.30), and an increase from the End of Plank to Recovery (*M* = 6.38, *SD* = 1.40), as expected due to orthostatic influences. No differences were observed between consecutive Epochs during the VR portion of the experiment: from Curb (*M* = 5.37, *SD* = 1.38) to Bottom of Elevator (*M* = 5.22, *SD* = 1.49), Top of elevator (*M* = 4.92, *SD* = 1.45) to Start of Plank (*M* = 4.89, *SD* = 1.76), or from the Start to the End of Plank (*M* = 5.01, *SD* = 1.61).

However, comparing the street-level measurement at Curb, to the measurements at height, lnHF was significantly lower at the Top of the elevator ($p < .001$), at the Start of the plank ($p = .011$), but not the End of Plank Epoch ($p = .069$) — probably because many participants showed an increase again at this last epoch. These decreases at height were not seen relative to the Bottom of Elevator, however, probably because many participants exhibited an increase here. In this model, Condition did not yield either a significant between-subject ($p = .172$) or interaction effect [$F(10.49) = 1.20$, $p = .285$, $\eta_p^2 = .027$].

The effect of time on lnHF was tested using another repeated measures ANOVA, this time using the bottom, top, start and end time points from which individual's curb values had been subtracted. This was to control for the effect of VR and examine more closely the effect attributable to the height-exposure. The assumption of sphericity was met in this case and so no correction was applied. The ANOVA yielded a small but significant effect of time on lnHF, $F(3, 2) = 3.35$, $p = .020$, $\eta_p^2 = .035$. Post-hoc Tukey's tests showed a significant difference in curb-controlled HRV between the Bottom of Elevator (*M* = -0.144, *SD* = 0.924) to the Top of Elevator (*M* = -0.448, *SD* = 0.809; $p = .025$), indicating a greater decrease in HRV relative to street level. No significant differences were found between the Top of Elevator and Start of Plank (*M* = -0.476, *SD* = 1.20), or from the Start to End of Plank Epochs (*M* = -0.356, *SD* = 1.05). Moreover, the Bottom of Elevator was not significantly different

from Start (p = .068) and End (p = .269) of Plank epochs. Entering Condition into this model did not yield either a significant between subject (p = .091) or interaction effect, F(3, 90 = 1.47, p = .187, η_p^2 = .031).

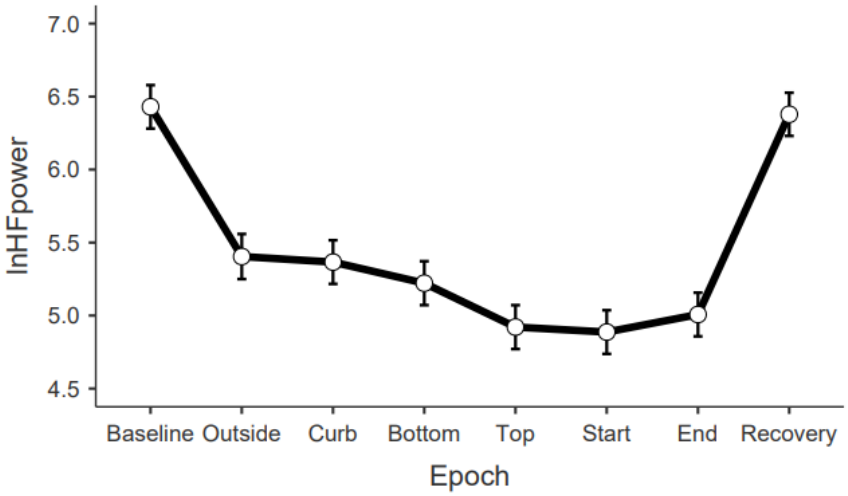

Figure 21: lnRMSSD by epoch
Time course of log transformed RMSSD data, using mean scores of whole sample (n = 99), with error bars depicting standard error of the mean.

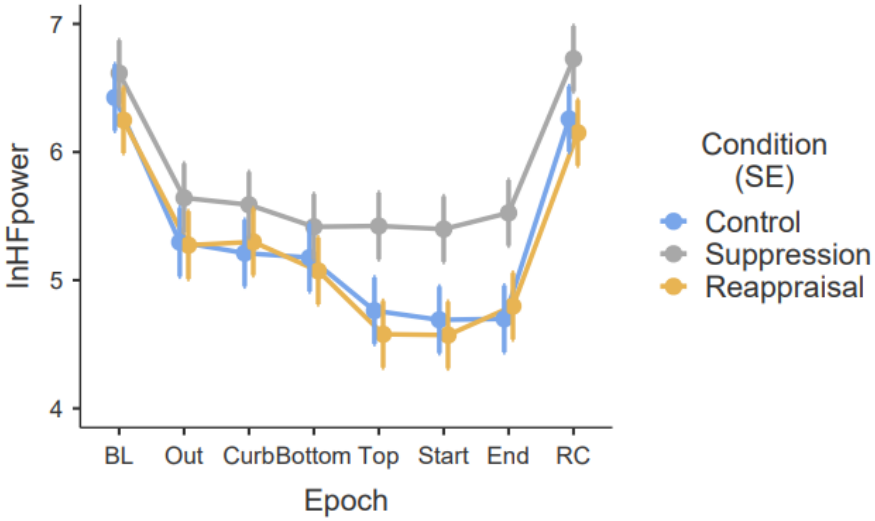

Figure 22: lnHF by epoch, split by condition
Time course of log transformed high-frequency HRV data, split by experimental condition (group n = 33), with error bars depicting standard error of the mean.

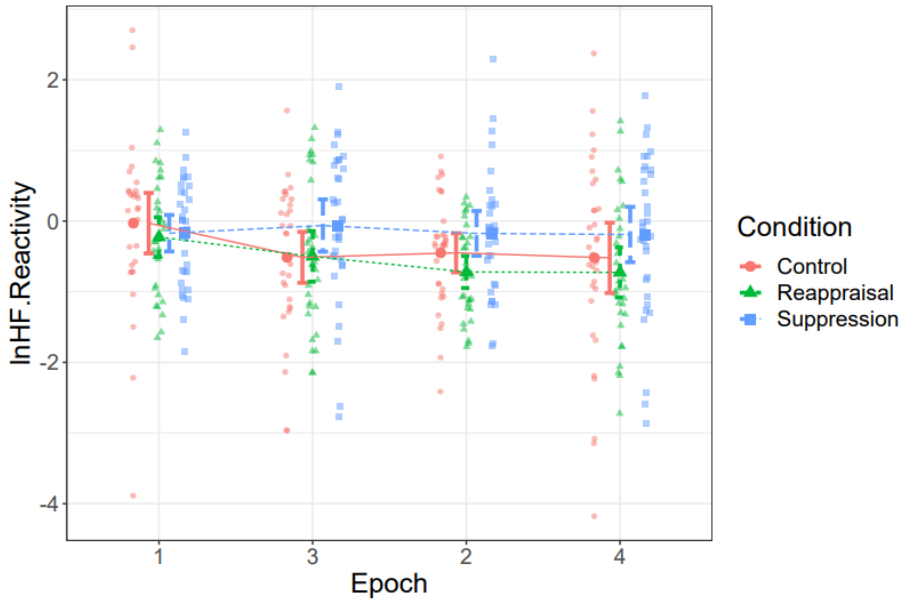

Figure 23: lnHF change by epoch, split by condition
Time course of log transformed high-frequency HRV data change scores (Bottom of Elevator, Top of Elevator, Start of Plank, End of Plank) with Curb values subtracted from each. Observed values plotted with standard error bars

HRV Reactivity

Reactivity scores were calculated in the same fashion as for Study 2 — the Start of Plank epoch is used, from which the Curb epoch value was subtracted for each participant. In Study 3, Reactivity was shown to typically involve a decrease for lnRMSSD (M = -0.288, SD = 0.451), and lnHF (M = -0.476, SD = 1.20). These reactivity measures differed from normality due to a strong left skew where a few participants had dramatic decreases and most had small decreases. However, HRV *Reactivity*Δ scores on both measures are evidence of a substantial withdrawal of cardiac vagal control during height-exposure.

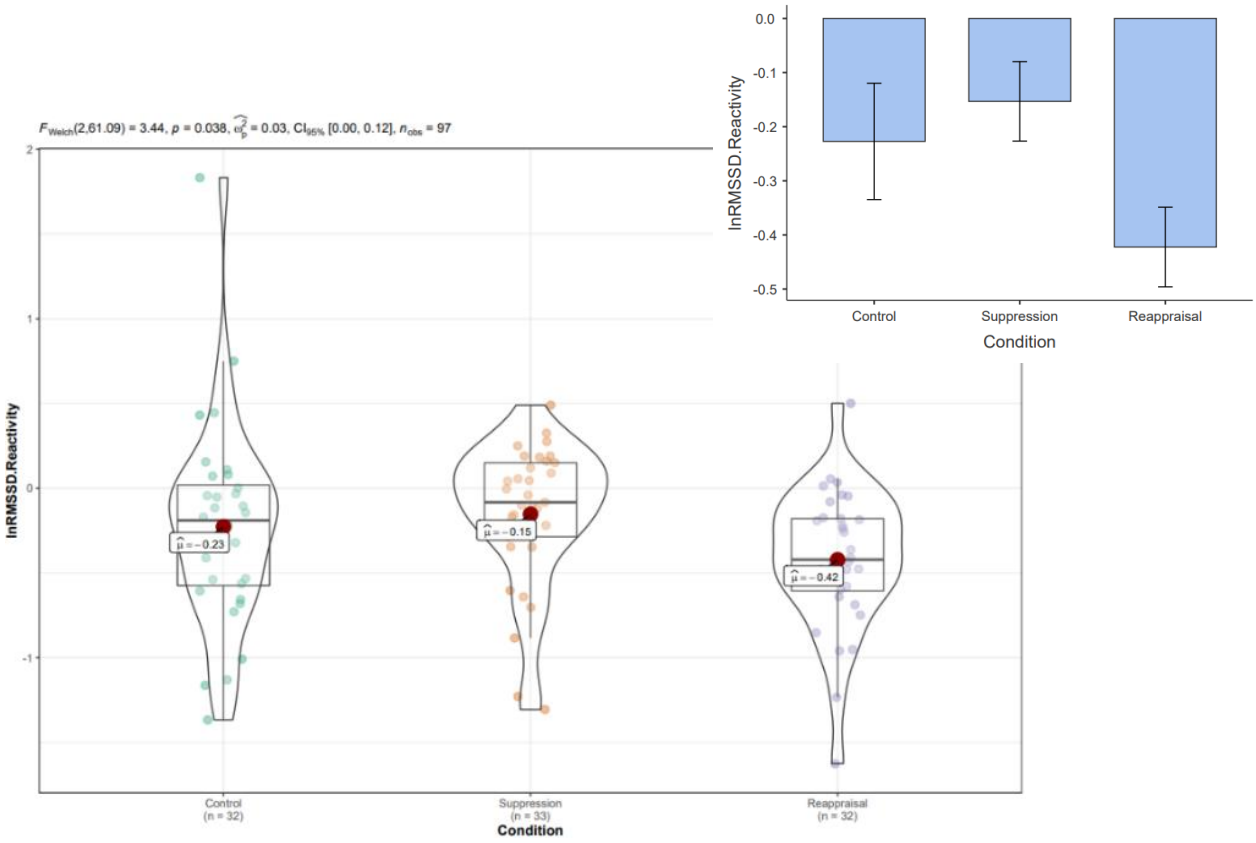

Figure 24:HRV Reactivity by group, bar graph (top) and violin (bottom)
HRV reactivity values represented by change scores of lnRMSSD (Start of Plank – Curb epochs) as a function of experimental condition. Reappraisal significantly different from Suppression condition.

A Welch's one-way ANOVA[27] was used to test for between-group differences in lnRMSSD Reactivity values. F(2.44, 61.1) = 3.44, p = .038. Descriptively, the Suppression group showed the least decrease in HRV, that is the smallest HRV Reactivity (M = - 0.15, SD = 0.42), followed by the Control group (M = - 0.23, SD = 0.61), while the Reappraisal group showed the greatest magnitude decreases (M = - 0.42, SD = 0.42). When using the Games-Howell method of correction (for unequal variances) the comparison yields a significant difference (mean difference = .27, p = .032)[28]. While the results of preregistered analyses do not provide convincing evidence in support of hypotheses, the discrepancy

[27] A non-parametric version of this ANOVA was also run to corroborate the results of the between-subjects test on HRV reactivity. The Kruskal-Wallis ANOVA results: $X^2 = 8.62$, p = .013. Dwass-Steel-Critchlow pairwise comparisons revealed a significant difference between Suppression and Reappraisal groups (W = - 4.27, p = .007).
[28] Post-hoc Tukey's tests revealed no significant differences between the groups, though the difference between Suppression and Reappraisal groups approached significance (mean difference = .27, p = .074).

between the two strategy groups is consistent with predictions, and is remarkably similar to the results of another recent study (Jentsch & Wolf, 2020).

A hierarchical design was used to predict HRV *Reactivity*Δ using linear regression modelling. Models predicting lnRMSSD *Reactivity*Δ are described and reported here (see Appendix for models using lnHF). Two linear regression models were employed[29]. *Model 1* predicted lnRMSSD reactivity from Baseline lnRMSSD (resting HRV) and Group (Suppression vs Control; Reappraisal vs Control); *Model 2* entered self-reported suppression and reappraisal use (from the modified ERQ).

Model 1, predicting lnRMSSD *Reactivity*Δ from Baseline lnRMSSD and Condition (emotion regulation instruction) failed to account for a significant amount of variance, R^2adj. = .023, $F(3,90) = 1.74$, $p = .165$. The addition of self-reported suppression and reappraisal scores in Model 2 did not significantly increase the variance accounted for in lnRMSSD *Reactivity*Δ, $R^2\Delta = .00372$, $F(2,88) = .74$, $p = .841$. Model 2 did not account for a significant amount of variance in lnRMSSD reactivity, R^2adj. = .00497, $F(3,88) = 1.09$, $p = .370$, and an increased BIC relative to Model 1 (149 to 158) indicates a worse model fit upon the addition of strategy use as additional predictors. Moreover, none of the four independent variables included in the model were significant predictors independently.

In sum, hierarchical modelling for both measures of HRV Reactivity, calculated as a change score from Curb to Start of Plank, indicates that there is no relationship found between Resting HRV and HRV Reactivity, nor can Reactivity be predicted by the experimental group a participant was assigned to, nor by the use of either Suppression or Reappraisal during the Plank-Walk. This is consistent with the findings of Study 2. Similarly, to in Study 2 this null finding begged the question of whether any relationships would emerge

[29] Assumptions of non-collinearity and normalised residuals were not violated in any cases

when doing similar investigations of the same relationships but with absolute values of HRV during Height-Exposure. However, a cursory examination of the correlations (See Table X) indicate no evidence for a relationship here either, and preclude justification for further regression modelling.

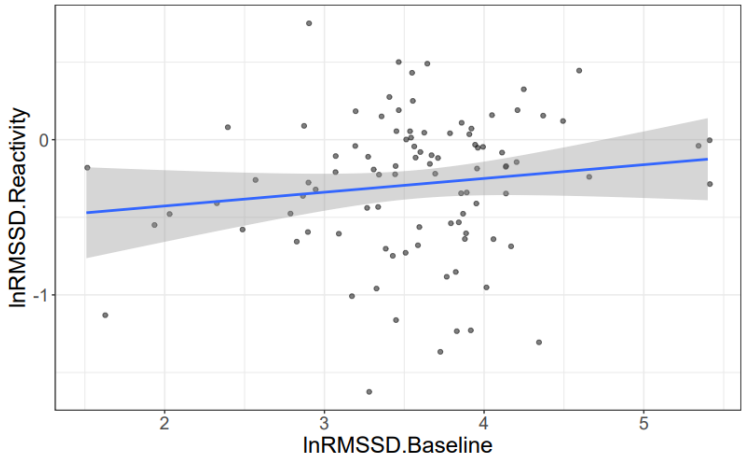

Figure 25: Scatterplot, lnRMSSD Baseline predicting lnRMSSD Reactivity
Scatterplot depicting fit of data predicting HRV Reactivity, as measured by lnRMSSD change scores (Start of Plank epoch – Curb epoch), using lnRMSSD at Baseline as a predictor

HRV Recovery

*Recovery*Δ values were calculated for each participant by subtracting Start of Plank values from Recovery values, thereby creating a value that reflects the change from threat to recovery. On average, participants showed a positive change from threat to recovery for both lnHF (M = 1.47, SD = 1.28) and lnRMSSD (M = 0.82, SD = 0.58). These data indicate that for most participants are substantial restorative effect occurred in HRV following the absence of height-exposure and restoration of safe, resting conditions.

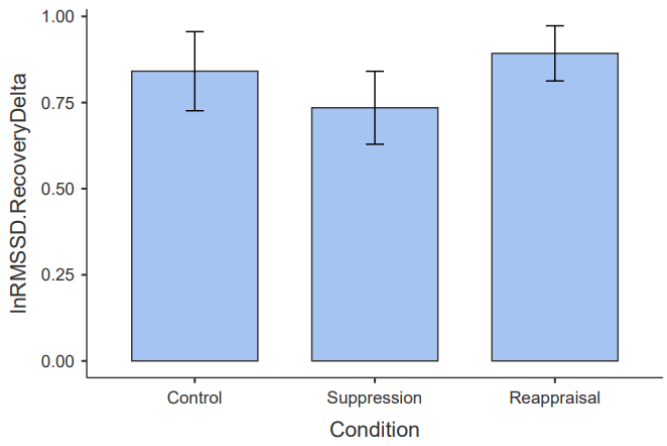

Figure 26: lnRMSSD RecoveryΔ by Condition
HRV RecoveryΔ values represented by change scores of lnRMSSD (Recovery - Start of Plank epochs) as a function of experimental condition. Error bars represent standard error of the mean.

A Welch's one-way ANOVA was performed to test for between-group differences in HRV RecoveryΔ. The results of this analysis did not yield a significant effect of group, F(2, 61.1) = 0.70, p = .500, a result that was confirmed by the Kruskal-Wallis non-parametric equivalent. Descriptively, the Reappraisal group showed the greatest magnitude increase in HRV during the Recovery following the stressor, although not significantly more than the other groups. The Reappraisal group also showed the greatest Reactivity and so had more to recover.

A hierarchical design was used to predict HRV *RecoveryΔ* using linear regression modelling. Models predicting lnRMSSD *RecoveryΔ* are described and reported here (see Appendix for models using lnHF). Two linear regression models were employed. *Model 1* predicted lnRMSSD reactivity from Baseline lnRMSSD (resting HRV) and Group (Suppression vs Control; Reappraisal vs Control); *Model 2* entered Suppression and Reappraisal use (averaged scores for modified-ERQ subscales pertaining to the Plank walk).

Model 1, predicting lnRMSSD *RecoveryΔ* from Baseline lnRMSSD and Group did not account for a significant amount of variance, R^2adj. = .007, $F(3,90) = 1.24$, p = .301. The addition of Suppression and Reappraisal scores in Model 2 did not significantly improve this model ($R^2\Delta$ = .003, $F(2,88)$ = .143, p = .867). Model 2 did not account for a significant

amount of variance in lnRMSSD *Recovery*Δ, R^2adj. = .-.01164, $F(5,88) = .786$, $p = .563$, and an increased BIC relative to Model 1 (181 to 190) indicates a worse model fit upon the addition of strategy use as additional predictors.

In sum, hierarchical modelling for both measures of HRV indicate that there is no evidence for a relationship between Resting HRV and HRV RecoveryΔ, despite a very large ($r = .91$) correlation between resting lnRMSSD and absolute values of lnRMSSD during the Recovery period.

Interim Summary

Data from Study 3 illustrate a very similar pattern of HRV change across the height-exposure paradigm as were seen in Study 2. These data replicate the initial findings, although with slightly smaller effect sizes in comparison. Further, less between-subjects variability was present relative to Study 2 (see table 12 below for descriptive statistics). I suggest that much of these two observations can be explained by the larger sample size in Study 3 — an extra 39 participants — and longer measurement Epochs (30s as opposed to 20s). It is likely that the effect sizes in Study 3 more closely reflect the true effect of height-exposure on HRV measures due to these statistical considerations. Interestingly, while HRV at height was lower than HRV at street level (as predicted), consecutive differences between epochs were only seen between the bottom and the top of the elevator, and this difference was only seen in a time-domain metric (and not a frequency-domain metric). An explanation for the different time course from study 2 is that the emotion regulation instructions given in the elevator ride may have caused anticipatory changes in autonomic arousal. Overall, the pattern of HRV Reactivity and Recovery indicates more evidence for the phenomenon described in Studies 2 and 3, whereby cardiac vagal control decreases during threat and is restored to baseline levels during restoration of resting conditions/absence of threat stimuli —

further corroborating the claim that this withdrawal of the vagal brake is inherent to the autonomic response to threatening contexts.

However, no relationship was found between Resting and either Reactivity or Recovery indexes of HRV. This null relationship is inconsistent with the Regulation hypothesis whereby higher levels of cardiac vagal control under resting conditions is associated with a more adaptive response of cardiac vagal control during emotional challenge (or indeed any relationship at all). This observation is convergent with Study 2, but divergent from that of Study 1 (the latter found results opposite to those predicted by the Regulation hypothesis, that is, people with higher resting HRV showed greater HRV decreases). This discrepancy between studies can be interpreted as either 1) evidence for a difference in the autonomic response between different threat *types*; or (2) reflecting substantial inadequacies of the HRV measurement techniques employed in Studies 2 and 3 due to constraints of the VR environment and technologies available. The short epoch lengths were able to capture the large reduction in HRV associated with emotional challenges, though the relationship between Resting HRV and HRV Reactivity may require more sensitive measures.

Consistent with the findings of Study 2, no effects of self-reported use of suppression or reappraisal were observed on indexes of HRV reactivity during the Height-Exposure. Further, in Study 3 there was little evidence for an effect of emotion regulation strategy as tested by explicit instructions given to participants: no differences were seen between the three conditions of Suppression, Reappraisal, or Control Conditions on the time course of HRV measures (as tested by primary analyses). However, there was a trend whereby participants in the Reappraisal condition showed greater HRV Reactivity when compared to those in the Suppression condition. This is an interesting pattern that is opposite to the predicted pattern (it was expected that Reappraisal would show the least reactivity). The Suppression instructions appear to have achieved their intended purpose, as those in the

Suppression condition reported using suppression to a greater degree on the modified ERQ than those in the other conditions (the equivalent is not true of the Reappraisal group). This result could be interpreted as evidence that reappraisal is associated with a higher degree of autonomic flexibility, rather than with less HRV Reactivity.

Table 13: Descriptive Statistics

Variable	*N*	Mean	SD	Min.	Max.	Skew	Kurtosis	Shapiro-Wilk *p*
Resting	98	3.567	0.675	1.51	5.40	-0.282	1.701	.003
Height	97	2.760	0.776	1.10	5.08	-0.004	0.199	.435
Recovery	99	3.594	0.684	1.62	5.47	-0.197	1.139	.042
ReactivityΔ	97	-.266	0.498	-1.62	1.83	0.253	2.797	.002
RecoveryΔ	97	.822	0.575	-1.08	2.29	-0.043	0.775	.263
Supp.Avg	97	3.500	1.294	1.00	7.00	0.116	-0.310	.387
Reapp.Avg	98	4.719	1.234	1.00	7.00	-0.988	0.856	<.001
FearΔ	98	4.306	2.240	1.00	9.00	-0.202	-0.658	.005

Table 14: Correlation Matrix

Note: Pearson's *r* reported in table. * $p < .05$, ** $p < .01$, *** $p < .001$

	Resting	Height	Recovery	ReactivityΔ	RecoveryΔ	Supp.Avg	Reapp.Avg	ΔFear
Resting	—	—	—	—	—	—	—	—
Height	.703***	—	—	—	—	—	—	—
Recovery	.908***	.696***	—	—	—	—	—	—
ReactivityΔ	.082	.569***	.096	—	—	—	—	—
RecoveryΔ	.128	-.530***	.240*	-.655***	—	—	—	—
Supp.Avg	.151	.094	.102	.030	.034	—	—	—
Reapp.Avg	.082	.097	.094	.068	.021	-.052	—	—
ΔFear	-.336***	-.181	-.265**	.065	-.109	-.246*	-.050	—

Predicting HRV Reactivity$_{\Delta}$

Table 15: Model 1 - resting lnRMSSD & Dummy-Coded Condition (Comparison) predicting lnRMSSD reactivity

Coefficient	Estimate	SE	*p*-Value
Intercept	-.2978	.2805	.291
Resting	.0323	.0761	.672
Condition:			
Supp. – Control	.0222	.1243	.859
Reapp. – Control	-.2162	.1246	.086

Table reports unstandardised coefficients. $F(3,90) = 1.74$, $p = .165$. $R^2 = .0547$, R^2adj. = .023, BIC = 149.

Table 16: Model 2 – Adding Suppression and Reappraisal during Plank-Walk

Coefficient	Estimate	SE	*p*-Value
Intercept	-.38822	.3520	.273
Resting	.02866	.0777	.713
Condition			
Supp. – Control	.02942	.1284	.819
Reap. – Control	-.21314	.1260	.094
Supp.Avg	-.00360	.0408	.930
Reapp.Avg	.02375	.0414	.567

Table reports unstandardised coefficients. $F(3,88) = 1.09$, $p = .370$. $R^2 = .0585$, R^2adj. = .00497, BIC = 158.

Model 1 – 2 comparison: $R^2\Delta = .00372$, $F(2,88) = .174$, $p = .841$.

Predicting HRV Recovery∆

Table 17: Model 1 - resting lnRMSSD & Dummy-Coded Condition (Comparison) predicting lnRMSSD Recovery∆

Coefficient	Estimate	SE	*p*-Value
Intercept	.3247	.3319	.331
Resting	.1440	.0900	.113
Condition:			
Supp - Control	-.1355	.1471	.359
Reapp – Control	.0782	.1474	.597

Table reports unstandardised coefficients. $F(3,90) = 1.238$, $p = .301$. $R^2 = .0396$, $R^2adj. = .00761$, BIC = 181.

Table 18: Model 2 – Adding Suppression and Reappraisal during Plank-Walk

Coefficient	Estimate	SE	*p*-Value
Intercept	.23047	.4166	.582
Resting	.13758	.0919	.138
Condition			
Supp. – Control	-.15068	.1520	.324
Reap. – Control	.07410	.1492	.621
Supp.Avg	.02541	.0483	.600
Reapp.Avg	.00714	.0490	.884

Table reports unstandardised coefficients. $F(5,88) = .786$, $p = .563$. $R^2 = .0427$, $R^2adj. = -.01164$, BIC = 190.

Model 1 – 2 comparison: $R^2\Delta = .00312$, $F(2,88) = .143$, $p = .867$.

General Discussion

In this thesis I investigated the patterns of parasympathetic activity that occur in response to threat in three empirical studies and tested whether between-subject factors modulate this pattern (emotion regulation, a history of self-injury, and Resting HRV). Parasympathetic activity was measured by time-domain and frequency-domain metrics of HRV to quantify cardiac vagal control. Two ecologically valid threat paradigms were used: Study 1 used social-evaluative threat (Trier Social Stress Task), whereas in study 2 and 3 participants experienced a novel virtual reality simulation that incorporated realistic height-exposure. Three research questions were addressed: (1) how does heart rate variability change during exposure to threatening contexts (i.e. what is the nature of HRV Reactivity?) (2) how is this change related to Resting HRV and (3) how is this change modulated by the use of emotion regulation strategies, or by a history of non-suicidal self-injury. In the following, I will summarise my findings pertaining to these three questions, and their relative congruence with the extant literature reviewed in the introduction.

Summary of Findings

Q1: How Does HRV Change Under Conditions of Threat?

Question 1 refers to a descriptive task: an exploration of the pattern of parasympathetic activity that accompanies the response to threat in human participants. There is a lack of empirical investigations into this question, and so the first aim in this thesis was to rigorously address this gap. Based on theory I predicted that a decrease in HRV would occur during threat.

All three studies provided evidence for a robust effect of threat on measures of HRV, such that HRV significantly decreased from baseline to threat, and was restored to at least baseline levels during a recovery period. These data support the hypothesis that cardiac vagal

control is withdrawn as part of a response to threat, helping to mobilise metabolic resources to meet environmental demands. One caveat is that there was substantial between-subject variability, such that that some participants in fact showed phasic *increases* during threat.

In Study 1, HRV decreased during social-evaluative stress, and the recovery showed a rebound effect whereby HRV increased *above* baseline levels. In Studies 2 and 3, HRV decreased between the Curb epoch, which served as an inside-VR baseline, and the Start of Plank epoch — the latter representing the maximum emotional challenge at the peak of threat exposure. Interestingly, while Studies 2 and 3 utilised a near-identical paradigm, the time course was slightly different between them. In the former case, the largest magnitude decreases in HRV occurred between the top of elevator and start of plank epochs; conversely, in Study 3, the largest magnitude decrease occurred between the bottom and top of elevator epochs. A speculative explanation for this discrepancy in time-course is that in Study 3, the instructions given to participants in the elevator ride, which comprised the emotion regulation manipulation, may have caused an anticipatory change in autonomic activity in the lead up to the elevator doors opening. Across all studies, the pattern of dynamic changes in HRV was similar for both time domain (RMSSD) and frequency-domain (high-frequency) metrics for cardiac vagal control. The consistency between measures provides converging evidence/convergent validity for the phenomenon detected in the studies, as both purportedly tap into the same underlying mechanism (cardiac vagal control). While similar patterns were observed, visual inspection of the plots depicting HRV measures in Study 2 reveals an increase in the frequency-domain measure during the two elevator epochs, while the time-domain measure remains flat across these same points. However, this difference between measures was not present in Study 3, precluding any firm conclusions based on the different patterns. Of note, there was far more between-subject variability in the frequency-domain measure than for the time-domain measure — for instance, standard deviations for HF in

Studies 2 and 3 were around twice that of RMSSD. The lower between-subject variability observed in RMSSD data is consistent with a review article summarising HRV metrics (Thayer et al., 2018), in which the authors claim that the statistical properties of RMSSD are more favourable than other commonly used metrics, and therefore recommend its use for psychology research.

Q2: Does Resting HRV predict the change in HRV During Threat?

Question 2 pertains to the relationship between two levels of HRV measurement, Resting and Reactivity. The relationship between these two variables is poorly understood, and very few studies have explicitly set out to test it. Laborde and colleagues do well to emphasize the importance of studying both Resting and Reactivity of HRV, but these studies are still among the few to actually do so. It was predicted, based on theory, that higher levels of Resting HRV (i.e. baseline values) would be associated with Reactivity values, such that those participants with greater variability under resting conditions (a trait-like measurement) should show smaller magnitude decreases in HRV under threat (a phasic measurement). Note that Reactivity was calculated as a change score, meaning that more negative values indicate more decrease upon threat-exposure, whereas positive numbers would represent an increase.

In Study 1, a significant and strong negative relationship was found between resting and Reactivity values. That is, participants with higher Resting HRV showed greater magnitude decreases during threat (r = -.43). This observation stands in marked opposition to the predicted pattern. In studies 2 and 3, no significant relationship emerged between Resting and Reactivity. However, in Study 2, the direction of the relationship was consistent with Study 1; that is, higher HRV at baseline was associated with greater decreases during threat (but did not reach significance). In Study 3 however, the (non-significant) relationship was in

the opposite direction. There are only a handful of published studies in which this question has been directly addressed.

Park et al. (2014) showed that during a target-identification task, participants with higher resting HRV exhibited phasic *increases* in HRV during trials with negative emotional faces, while those with lower resting HRV showed decreases. The present findings are in the opposite direction. The difference in the methods used between these studies and that of Park and colleagues may explain this discrepancy, as a strong emotional challenge is likely to yield different responses than a mild emotional stimulus.

Fundamentally, the question of how Resting and Reactivity levels of HRV interact is a question of what constitutes an adaptive pattern of stress reactivity. One factor that may explain the mixed findings in the present studies is the concept of an "inverted U" function which describes how the magnitude of stress reactivity relates to outcomes (Sapolsky, 2015). That is, while too little of a response (not enough vagal withdrawal) may be maladaptive, too great a response (excessive vagal withdrawal) is equally so. Support for this idea comes from observations that the relationship between resting HRV and mental health outcomes often forms a quadratic function, in which high HRV is "too much of a good thing"; individuals diagnosed with anorexia nervosa have also shown elevated Resting HRV.

Q3: Do Emotion Regulation Strategies Alter the Change in HRV During Threat?

The third question pertains to whether between-subject differences in the use of two emotion regulation strategies modulates the observed pattern of dynamic HRV during threat. This question remains largely under-studied, despite the many theories which propose a relationship between them. The predictions for this question were drawn from emotion regulation theory, and from the neurovisceral integration model. It was hypothesized that higher reported use of either expressive suppression, or cognitive reappraisal, would be

associated with less HRV reactivity. In other words, deploying emotion regulation strategies was expected to buffer vagal withdrawal, and therefore to be associated with less decrease in HRV during threat.

There was little evidence found in support for the predicted association between emotion regulation and HRV Reactivity. In all three studies, the self-reported use of neither strategy significantly predicted HRV Reactivity. This is a surprising null finding that was consistent across all studies. However, when running exploratory correlation analyses, substituting the HRV values *during* threat (as opposed to the change scores used to capture HRV Reactivity), some significant relationships emerged: in Study 2, higher self-reported suppression use was positively related to HRV during the plank-walk (r = .35; see table 2 above). However, this finding was not replicated in Study 3. In Study 1, a relationship emerged between self-reported reappraisal use and HRV during the social-evaluative stress, such that higher levels of reappraisal were associated with lower levels of HRV during the stress epoch of the study (r = - .29; see Table 2 above).

For Study 3, visual inspection of the HRV time course data suggests a qualitatively different pattern between groups, such that the Suppression group has higher HRV during the threat relative to the other conditions. Primary analyses (non-significant interaction effect in the ANIOVA) did not reveal evidence for this notion, revealing no initial support for the regulation hypothesis. So, while participants were assigned to use either suppression or reappraisal, neither of these conditions showed a significantly different time-course of HRV relative to participants in the control condition. However, when testing for differences in HRV Reactivity between the three conditions, a significant effect of group *is* found: those in the Reappraisal condition showed a greater HRV Reactivity relative to those in the Suppression condition, but not different from Control participants. And, while those assigned to the Reappraisal condition did not report using reappraisal more than those in the other

conditions, those in the Suppression condition did report using suppression more relative to the other conditions. Therefore, some evidence was found that instructed use of expressive suppression leads to smaller magnitude decreases in HRV during a fear-induction (height-exposure in VR) relative to instructed use of cognitive reappraisal.

Jentsch and Wolf (2020) use a very similar method to Study 3 in the present work, and found evidence for a difference in HRV Reactivity between participants assigned to Suppression and Reappraisal groups. In this study, the Reappraisal group showed a significantly greater magnitude decrease in HRV during a stressor relative to the Suppression group, which is consistent with my findings. Interestingly, in this study, the group difference was moderated by trait levels of reappraisal use, such that those who report greater levels of habitual reappraisal use (on the ERQ) showed greater phasic HRV Reactivity and recovery than those with lower trait levels. So, while this analysis only partially supports my predictions, it is highly consistent with this previous work. The authors of the prior study interpret the finding as follows: reappraising during stress facilitates greater cardiac vagal flexibility, which is a more adaptive response to stress.

The largely null effects of self-reported emotion regulation on HRV Reactivity is surprising, given the multitude of theories which instantiate claims for a strong link between the two constructs. In the General Introduction to this thesis, I identify six empirical studies which explicitly test the link between suppression or reappraisal and HRV during an emotional challenge, and which demonstrated an effect of at least one of the strategies on increasing HRV or reducing HRV Reactivity. In contrast to these studies, the data from the present studies finds no consistent effects. However, I used HRV Reactivity as the primary dependent variable to test this hypothesis, as opposed to absolute values of HRV during the emotional challenge. This decision is based on theories which hold HRV Reactivity to be an important construct, and due to the fact that little research has done so at present. Given that

relationships emerge in the present data when using absolute HRV values rather than change scores, this difference in approach may explain the incongruence between the literature and the present findings. Another potential explanation for the discrepancy is that the present studies used ecologically valid threat exposure paradigms, which by design elicit a relatively authentic emotional response in participants. Perhaps, it is more difficult to implement emotion regulation strategies under realistic conditions, relative to the methods used in the other studies (e.g. video clips). Finally, I did not address the question of whether the attempts at emotion regulation were actually successful, in terms of downregulating subjective emotion (i.e. beyond physiological measures of emotion). One might argue that the reason for the lack of relationship between strategy use and HRV reactivity in the present data is that an effect may only emerge among those individuals who successfully regulate, and that simply *attempting* to do so does not affect the parasympathetic nervous system. A better way to test the regulation hypothesis then may be to use moderation analyses. This could be done by testing for relationships between strategy use and change in subjective emotion ratings, using HRV Reactivity as a moderating variable. In this way it could be determined whether the effect of emotion regulation strategies depends on the degree of vagal flexibility an individual has (these analyses are beyond the scope of this thesis).

Such a method has been used in at least one study. Stange et al. (2018) induced sadness in participants via a film clip and examined the relationship between self-reported use of suppression and reappraisal (as well as distraction), and HRV Reactivity and Recovery. Findings from this study suggested that reappraisal was effective in downregulating negative affect, but only in participants with greater levels of HRV Recovery following the emotional challenge, a pattern somewhat consistent with the Reappraisal condition in Study 3 in the present work. Conversely, the use of suppression was associated with an *increase* in negative affect, but only in participants with lower levels of HRV

Reactivity during the emotional challenge, again, somewhat consistent with the Suppression group in Study 3 (who showed less HRV Reactivity). It would be interesting to explore whether the findings from Stange and colleagues (2018) replicate in the fear-induction used in the present work.

Theoretical Implications

Polyvagal Theory

Porge's Polyvagal theory holds that cardiac vagal control is a mechanism necessary for mammalian species to flexibly respond to changing metabolic demands. The theory predicts that vagal flexibility, in particular (i.e. context-appropriate HRV Reactivity) is a crucial part of the threat response. The findings in this thesis provide support for this idea. Specifically, these studies demonstrated that HRV decreases during threat and is subsequently restored. Moreover, in Study 1, Resting and Reactivity were inversely related — a surprising finding which is best explained by the Polyvagal account: individuals with higher resting cardiac vagal control are able to withdraw cardiac vagal control under conditions of metabolic demand, which in principle is an adaptive strategy for successfully interacting in the world.

Neurovisceral Integration Model

The major use of the neurovisceral account in the present work is its explanation for a link between emotion regulation and cardiac vagal control. The authors hold that observed correlations between emotion regulation, cognitive control, and psychopathology and HRV can be explained by underlying neural mechanisms in the baroreflex-central autonomic network loop. I sought to test this proposition using HRV Reactivity and two specific regulation strategies. My data overall did not support the Neurovisceral Integration model. Self-reported use of both strategies did not correlate with HRV Reactivity. However,

participants in the instructed Reappraisal condition showed more pronounced decreases in HRV compared to the instructed Suppression group, showing that they did have divergent effects on cardiac vagal control. While resting HRV may be clearly linked to some aspects of emotion regulation (e.g. the scores on global self-report questionnaires), the results of this thesis suggest that dynamic HRV may not be linked to emotion regulation in the moment. It could also be the case that there is a link, but it is more complex. Either way, the neurovisceral integration model does not explain dynamic HRV and future theoretical work could be done to expand it.

Transdiagnostic mechanistic view

An RDoC-consistent view of emotion regulation as a transdiagnostic mechanism in psychopathology is endorsed by authors such as Beauchaine, who further hold that HRV is an index of the same mechanisms. In Study 1, individuals who reported engaging in non-suicidal self-injury did not differ from control participants on measures of Resting HRV or HRV Reactivity. This null finding is surprising given the view that NSSI is driven by impaired emotion regulation ability. However, the lack of an effect should be interpreted with caution given that this proposed explanation for NSSI behaviour has been critiqued and has not been upheld in the face of more recent empirical work (including that of Robinson, 2021). The idea that individuals exhibiting signs of psychopathology should differ on HRV Reactivity is additionally an indirect approach to testing my main prediction (for question 3) that emotion regulation should impact HRV Reactivity; an approach that is predicated on the *a priori* theoretical assumption that our NSSI group is less able to use emotion regulation during threat relative to their control-group counterparts.

Gross' Process Model of Emotion Regulation

Gross and colleague's influential account of emotion regulation was used to determine the two strategies which were the focus in the present studies. Gross' view understands emotion as a process that unfolds across time, in which cognitive control strategies can be implemented to alter the trajectory and effects of an emotion. Further, the account holds that antecedent-focused strategies should have divergent consequences relative to response-focused strategies. My data revealed little evidence for an effect of the strategies on HRV. In Study 1, supplementary analyses revealed that higher self-reported use of both strategies were associated with smaller increases in negative affect (see figure 31A, in Appendix C), but these effects were not consistent across all studies; in Study 2, neither strategy was associated with change in fear, and in Study 3, higher self-reported suppression use was associated with smaller increases in fear (but not reappraisal use). An implication of this discrepancy is the possibility that the fear induced in the latter studies is too intense to be easily regulated, whereas the social-evaluation in Study 1 induced a kind of emotional experience that can be controlled more easily. So, some evidence was found that emotion regulation strategies from the Process Model are effective, though effects of the strategies on HRV were mixed.

Limitations

Here I will discuss several methodological limitations which are present in the studies in this thesis. While some novel and interesting findings are presented, surprising null findings also emerged. It is likely that certain problems in the methods used here contributed to these results. These problems fall into four broad categories: HRV methods, the question of adaptivity, the measures of emotion regulation, and the way in which emotion regulation was manipulated (in Study 3).

HRV Methods

The first category concerns possible problems in HRV data collection and processing. Firstly, I used measurement epochs which are shorter than the recording length recommended by some researchers — most suggest that 5 minutes is the minimum for reliable data that captures cardiac vagal control. However, while this is easily implemented in resting HRV measurements, it is not conducive to study designs where dynamic changes in parasympathetic activity are to be measured. The 20 and 30 second epochs in Studies 2 and 3 respectively proved to be sensitive enough to capture emotion-related changes in parasympathetic activity, replicating the pattern seen in Study 1, with longer epochs. So, regarding my attempts to address question 1 (the pattern of HRV Reactivity) these ultra-short epochs proved to be adequate. However, given the discrepancies between the findings of Study 1 and the following 2 studies, it is possible that these epoch lengths did not capture enough of the within-subject variability yield associations with individual differences (i.e. emotion regulation strategy use and Resting HRV). A second limitation regarding the HRV data is the way in which it was processed prior to analyses. I used a visual inspection process in all three studies to clean the data of artefacts. This method allowed me to ensure consistency across studies, and I was blind to experimental conditions (NSSI status in Study 1, and emotion regulation condition in Study 3). However, manual processing of this kind necessarily introduces noise associated with the possibility of human error. Further, the manual method fails to address the concern regarding what minimum or maximum R-R intervals should be included in HRV calculations. That is, physiological artefacts, known as ectopic beats, are observed in cases where a normal heart beat is followed by another beat very soon afterwards — a common phenomenon due to sinoatrial node misfiring. Another concern is that for HRV analyses, an ECG sampling rate of 256hz and above is recommended. In Studies 1 and 2, a high-quality 3-lead ECG setup was used which provided 1000hz. Conversely, in Study 3 I used an ambulatory wireless ECG setup (the Equivital belt)

which allows a sampling rate of 256hz. This sampling rate is sufficient, but leaves some to be desired, and so in future it would be better to use a higher sampling rate once technological advances are made for ambulatory systems. Finally, HRV is a variable which is known to be subject to large within-subject variability. For instance, just about any factor that is known to influence physical health has been shown to affect HRV values (e.g. smoking, coffee/caffeine, sleep duration and quality, prescription medication, non-prescription drug use, physical exercise, rumination, common illness, chronic disease, stress, birth control, time of day, eating breakfast, and so on). The within-subject design in the present studies (as opposed to those which test for correlations between Resting HRV measurement and psychological phenomena) minimalizes the impact of these concerns, at least in regard to question 1. However, questions 2 and 3 (pertaining to Resting HRV and emotion regulation) require an individual differences approach, where these factors carry more weight. Given that I did not measure and control for any of these possible influences they may have impacted the data by introducing noise.

Operationalising Adaptivity

In hindsight, it seems possible that the statistical modelling used to test question 3 (how emotion regulation alters HRV Reactivity) does not completely capture the phenomenon of interest. Given that my predictions regarding HRV Reactivity are predicated on assumptions regarding what is adaptive under conditions of threat, a major limitation is that analyses in this thesis did not account for whether attempts at emotion regulation were actually successful. It follows from the theory reviewed in the introduction that adaptivity means to successfully regulate one's emotions — not just *to try* to regulate. Therefore, the hypothesis that the use of emotion regulation strategies would alter HRV Reactivity was tested; however, a more comprehensive test of this hypothesis should take into account the success of the attempts. It seems likely that it is the *successful* downregulation of negative

emotions, through the use of the two strategies, that confers measurable changes to the autonomic nervous system. A proposed method to address this limitation in future work (using a moderation approach) is outlined above.

Emotion Regulation Measures

An inherent limitation built into the present studies is the way in which emotion regulation was measured. I used a modified version of the Emotion regulation Questionnaire (ERQ), a self-report tool published by Gross and John (2001). The original questionnaire has been validated in many empirical studies, with demonstrable qualities of reliability and validity. However, here, I was interested in the effects of using the strategies during a laboratory emotion induction, to examine their influence on HRV. Therefore, the ERQ was modified such that it referred to the participant's experience *during* the stressful component of the study procedures (i.e. during the subtraction task; during the plank-walk). Participants were tasked with thinking retroactively to their time in the study and recalling the degree to which they used the strategies. This adaptation was originally used in the Robinson (2021) study that provided data for Study 1; the intention was to capture self-reported strategy use during the Trier Social Stress task. For consistency, the same measure was used in Studies 2 and 3. The limitations of self-report, in general, are well-known, foremost the fact that people do not have infallible access into their cognitive processes. That is, even when used as a global measure, it is unclear whether the ERQ fully captures what people really do and think in their daily life. This difficulty is exacerbated using our modified version of the ERQ: we are asking people to rely on their memory of a specific instance, in which they were also experiencing an intense emotional challenge. It would be unsurprising if people's memory is impaired under such conditions — at least regarding content that is not crucial for one's goals during the emotion — in which case the modified, retrospective use of the ERQ in this way may be a limitation in that it fails to sufficiently capture what people actually did.

Manipulating Emotion Regulation

In Study 3, a randomised, controlled and blinded trial was used to test the relative effects of instructed suppression and reappraisal compared to a control condition. People were assigned to one of the three conditions and received instructions accordingly as a manipulation of emotion regulation strategy use. However, a limitation is clear here when examining whether those assigned to one or the other "active groups" actually did use the strategy more than those in the other groups. This test is made possible by the fact that we collected self-report ratings of the degree to which participants recalled using suppression or reappraisal during the stressor (using a modified ERQ). So, to the extent that the modified ERQ subscales for suppression and reappraisal use provide valid and reliable measures of strategy use, we can check whether the manipulation of strategy use as effective. Participants in the Suppression group reported using Suppression to a greater extent than those in the other two groups. However, those in the Reappraisal group did not report using reappraisal to a higher degree than those in the other two groups. Therefore, we lack clear evidence that the manipulation of reappraisal was effective. This finding is a fundamental limitation when considering the results of Study 3. It is worth noting however that participants in the Reappraisal condition were told that the backpack they were wearing was a jetpack which would save them should they fall. While this instruction taps into the mechanism of reappraisal, in that it requires one to change the way they are thinking about the same situation, it bears little resemblance to the ERQ-based questions we used to assess reappraisal following the study. That is, participants may indeed be using reappraisal, but without reporting in the ERQ that they did so, if they fail to make the logical leap between the two. The Suppression condition seems somewhat less opaque in this regard, as the instruction (to act such that nobody can tell how they feel) maps closely to the questions in the ERQ. That

aside, the finding that the Reappraisal group did not recall thinking differently with respect to their assigned strategy challenges the effectiveness of the emotion regulation manipulation.

Future Directions

The limitations described above leave clear pathways for future work. First, some analyses were simply outside the scope of the present thesis due to time constraints. It would be useful to directly operationalise successful emotion regulation — an important task if we are to link a specific pattern of HRV Reactivity to an adaptive cognitive or affective style. This may require different statistical methods but could also simply involve separately analysing subgroups of individuals. For instance, participants could be split, based on a threshold of change in fear or negative affect which indexes a certain level of effective downregulation of emotion, such that two groups are formed (effective and non-effective regulators). However, doing so begs ontological questions regarding the nature of emotion regulation, such as: are these "effective regulators" truly changing their emotions in real-time, or are they just people who *generate* less emotion to begin with[30]?

Future studies should use a similar study design to test these hypotheses but with some changes. First, some simple HRV data collection improvements can be made, such as using a higher sampling rate, longer measurement epochs where possible, and controlling for between-subject lifestyle factors within the constraints of practicality. I would also employ an automatic processing algorithm to determine which R-R intervals should be excluded, which would mean manual removal is not necessary. A threshold for R-R interval length, - for

[30] This very question is explored in a review (Gross & Barrett, 2011), where the concept of emotion regulation is explored from the perspectives of varied theoretical and ontological commitments as to the nature of an emotion. For example, the psychological constructionist view proposes a central role of emotion generation and leaves little room for the idea of regulation once an emotion has arisen, while the process view used here centres around the use of regulation strategies.

example, if an R-R interval differs by more than 25% from the preceding interval - should be used.

Other advances could be made by improvements to the manipulation of strategy use, and by investigating whether the success of emotion regulation attempts depends on the content (rather than the process itself being necessary and sufficient). It appears that the reappraisal instructions in Study 3 did not result in those participants engaging in reappraisal more than controls, at least not as measured by self-report. It may be useful to try different iterations of the same method to address this. For example, the cognitive reappraisal instructions could be changed so that participants repeat to themselves internally that they are just in a virtual world, and that what they are seeing (i.e. a vast precipice beneath them) is not real so they will be safe. This instruction seems more analogous to an authentic form of reappraisal, and is consistent with the "fictional reappraisal" subtype shown to be effective in some studies (e.g. Makowski et al., 2019). This different instruction also addresses another concern: it is possible that participants are already consciously thinking to themselves that they are in VR and cannot be harmed, as a spontaneous emotion regulation strategy (following the study, some participants indeed mentioned that they did this). Having said this, it is unlikely that participants would be using this strategy in one condition more than others, therefore it should not be considered a confound. Also, if participants were in fact doing so, it did not prove to be very effective, as all groups demonstrated large increases in subjective and physiological correlates of fear. Alternatively, another commonly used "content" of reappraisal focuses on changing one's perspective regarding the effects of stress. There are studies (e.g. Crum et al., 2013) showing the beneficial effects of engaging in positive attributions towards stress (such as "stress makes me more focused, and more effective in coping with challenges") as opposed to a negative orientation (e.g. "stress is debilitating, unpleasant and harmful to my health"). Controversy exists regarding whether the content, or

focus, of a reappraisal matters for its effectiveness (see Gross, 2015). In sum, a recommendation for future studies of this kind is to test alternate versions of reappraisal to elucidate this distinction.

Another promising domain for future research is to home in on the cognitive mechanisms underlying emotion regulation. As outlined in the introduction, emotion regulation as understood in the process model relies on cognitive control capacities which are subserved by prefrontal cortex. Reappraisal and Suppression, while operationalised in the present studies as measurable cognitive phenomena, are both undoubtedly complex constructs in their own right. Perhaps, a useful approach to investigating emotion regulation and its link to parasympathetic activity would be to first examine more tightly defined cognitive constructs such as inhibitory control, working memory, cognitive flexibility or conflict adaption. This level of specificity may be useful in teasing apart which components of cognition are measurably related to HRV, and to what degree they are influenced by emotions such as fear. An example of such a study would be to implement a between-group design, in which one group experiences the height-exposure paradigm of Studies 2 and 3 (to induce fear) and another experiences the plank at street level (control). Both complete an ecologically-valid iteration of a task such as reversal learning — that is, a task which indexes cognitive flexibility and medial PFC function — while concurrently recording HRV data. An approach using fine-grained cognitive control measures (operationalised through behaviour) would perhaps provide a better test of the present hypotheses, rather than self-reported use of emotion regulation strategies, as the latter are complex and difficult to measure phenomena.

Lastly, in the present studies analyses were limited to two commonly-used measures of HRV: RMSSD and HF, which are known to reflect vagal activity. However, there is ongoing debate as to which methods are best suited for this purpose. RMSSD is known to be highly sensitive to many different perturbations to the nervous system (Pham et al., 2021;

Karemaker, 2021), and some evidence suggests other measures such as the Porges-Bohrer method are better indexes of vagal activity (Lewis et al., 2012). Furthermore, the nonlinear domain of HRV may capture important properties of the neuro-cardiac interactions which RMSSD and HF fail to (Pham et al., 2021). For instance, measures of entropy, complexity and fractal properties may help to elucidate the nonlinear interactions which occur between the different systems involved in psychological phenomena (i.e. cognitive mechanisms, brain networks, and the autonomic nervous system). Future work should systematically investigate the different measures, and how they relate to underlying mechanisms.

Conclusions

The present studies have made progress in furthering our understanding of how the parasympathetic nervous system responds to conditions of threat, and the factors that modulate this response. Regarding Question 1, my hypothesis was supported: findings suggest that withdrawal of vagal activity is a phenomenon which facilitates emotional responding in the face of fluctuating environmental demands. In keeping with this idea, data in all three studies show that the response to threat includes a decrease in HRV, in addition to concurrent sympathetic activation. For Question 2, which pertained to the relationship between Resting and Reactivity of HRV, my hypothesis was not supported: no evidence was found that higher Resting vagal activity is related to less vagal Reactivity. In fact, data from Study 1 showed that higher Resting HRV was associated with *greater* HRV decreases during threat (i.e. more vagal withdrawal), and no relationships were found in Studies 2 and 3. This finding, though isolated to Study 1, is interpreted through the lens of "autonomic flexibility", whereby flexible withdrawal of the vagal brake is seen as adaptive during threat. For Question 3, my "Regulation" hypothesis was also not supported: no evidence was found that self-reported use of emotion regulation strategies (either expressive suppression or cognitive reappraisal) is associated with less HRV Reactivity during emotional challenge. However,

participants who were instructed to use cognitive reappraisal (Study 3) exhibited greater magnitude HRV Reactivity (larger decreases) when compared to those instructed to use expressive suppression, in direct opposition to the predicted effect. Instructed use of reappraisal — invariably considered an adaptive strategy — may therefore be associated with greater autonomic flexibility. These studies advance our understanding of dynamic changes in parasympathetic activity during emotion, and provide some proof of concept regarding the assessment of these changes in ecologically-valid research paradigms.

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Appendix A (Study 1)

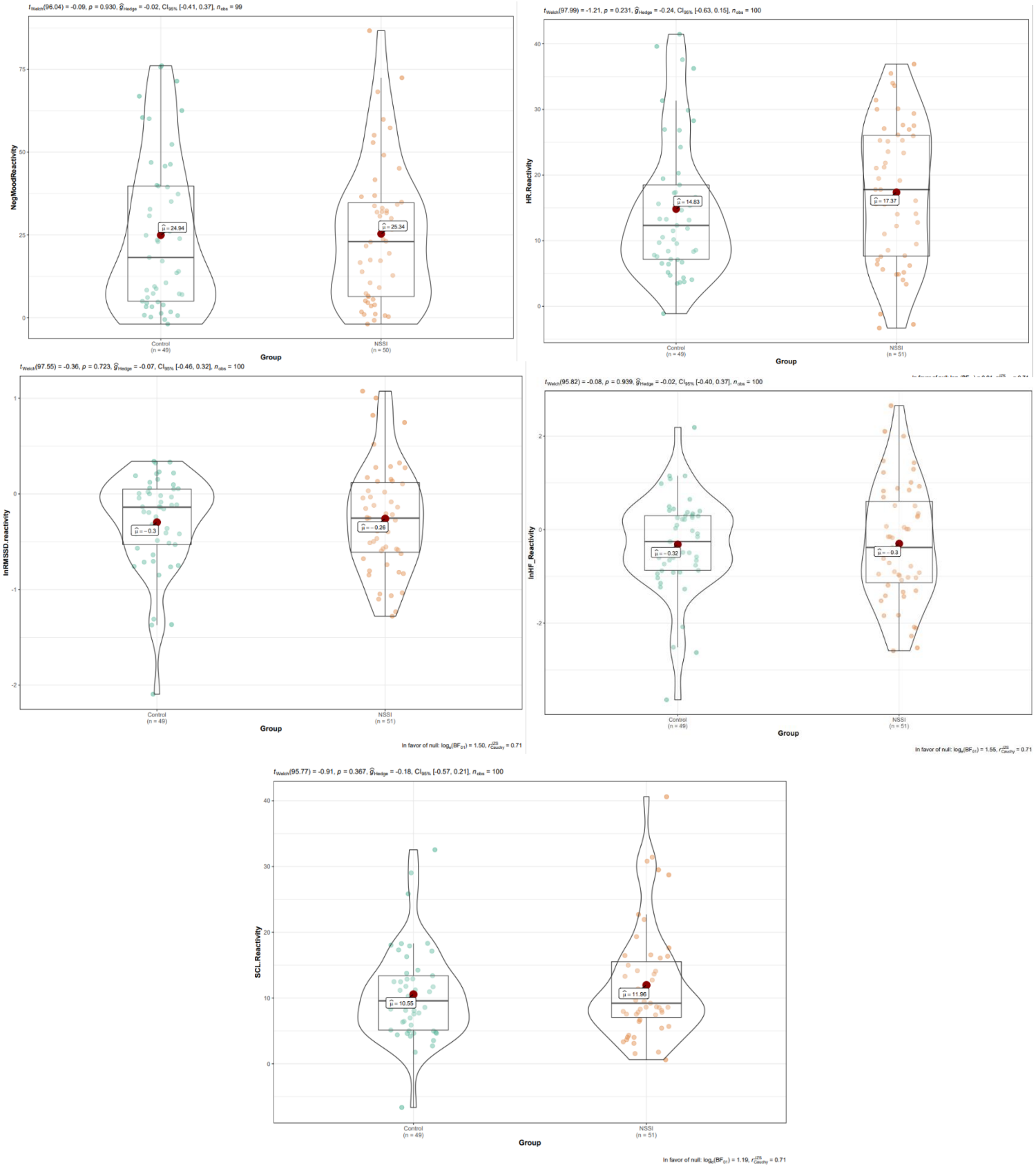

Figure 27 (A): Reactivity measures of emotion by Group
Negative affect and HR (top), lnRMSSD and lnHF (middle), and SCL (bottom) by Group

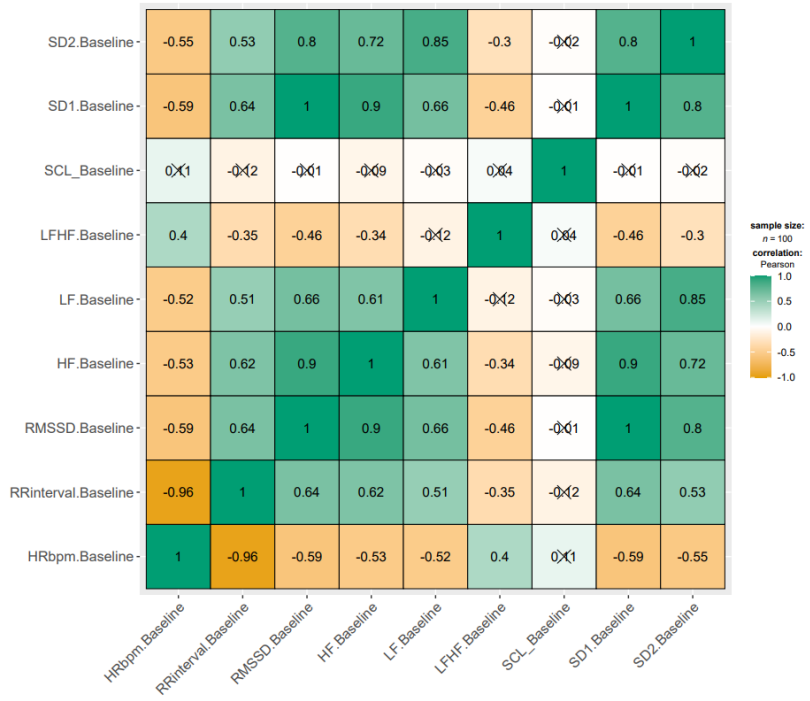

Figure 28(A): Correlations among all physiological variables at the Baseline Epoch

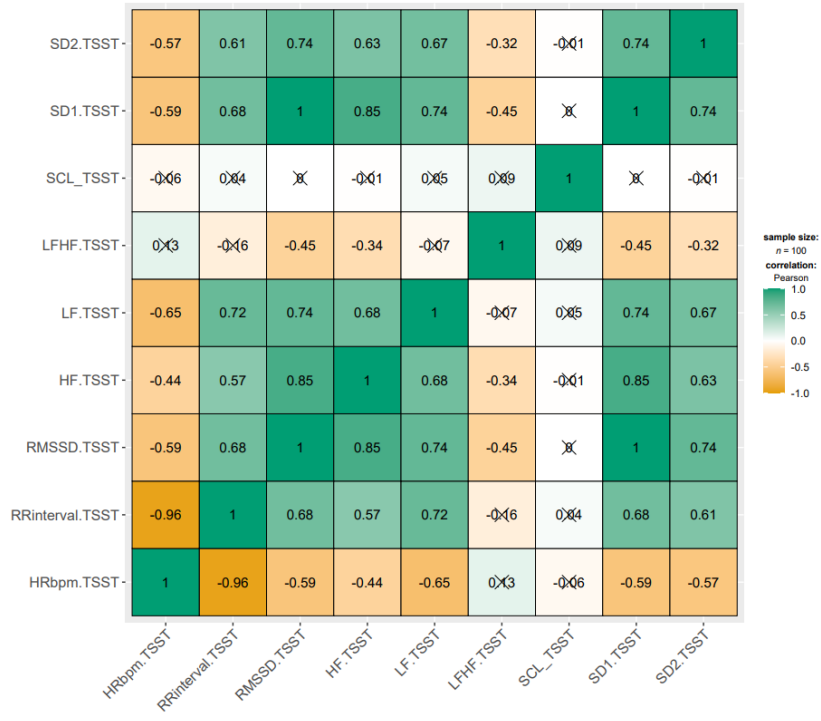

Figure 29 (A): Correlations among all physiological variables at the TSST Epoch

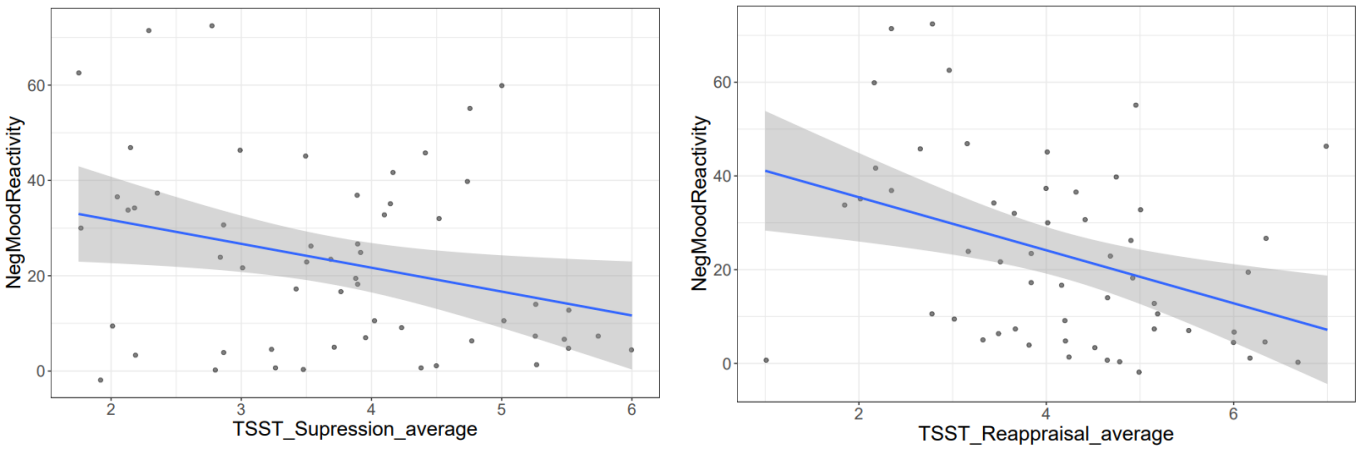

Figure 30 (A): Emotion regulation scores predicting change in negative affect
Self-reported use of both strategies predicted change in negative affect, such that higher levels of strategy use was related to smaller increases in negative affect. This relationship is stronger for reappraisal [r = -.39, p = .003] than for suppression [r = -.29, p = .027].

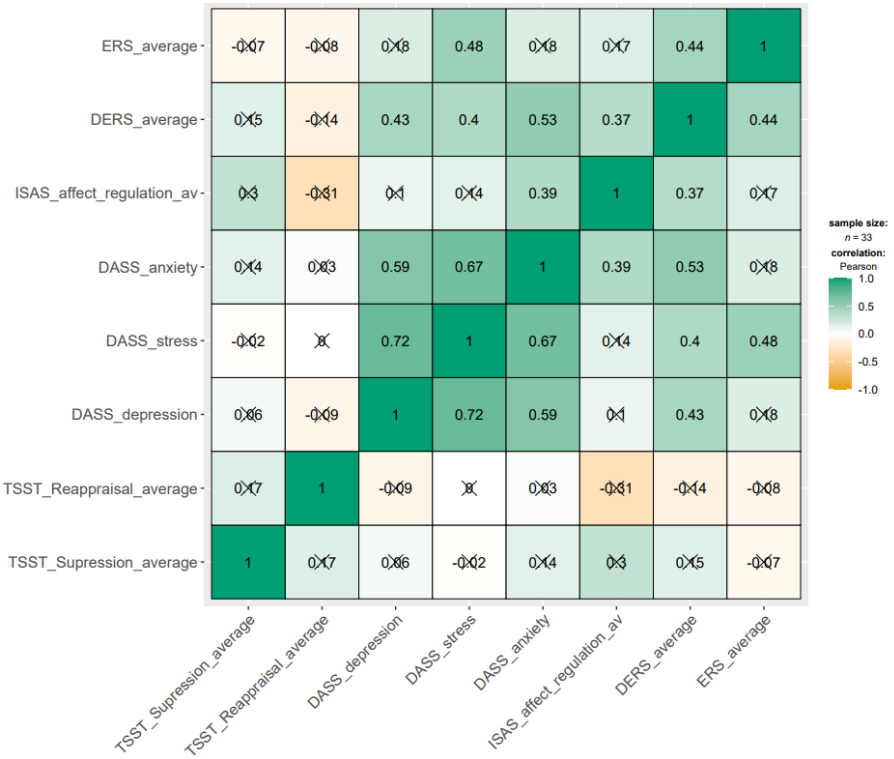

Figure 31 (A): Correlations between all questionnaire measures
The modified ERQ subscales did not correlate significantly with any other scale, suggesting a lack of external validity

Appendix B (Study 2)

Figure 32 (B): Correlations among all physiological variables at the Baseline Epoch

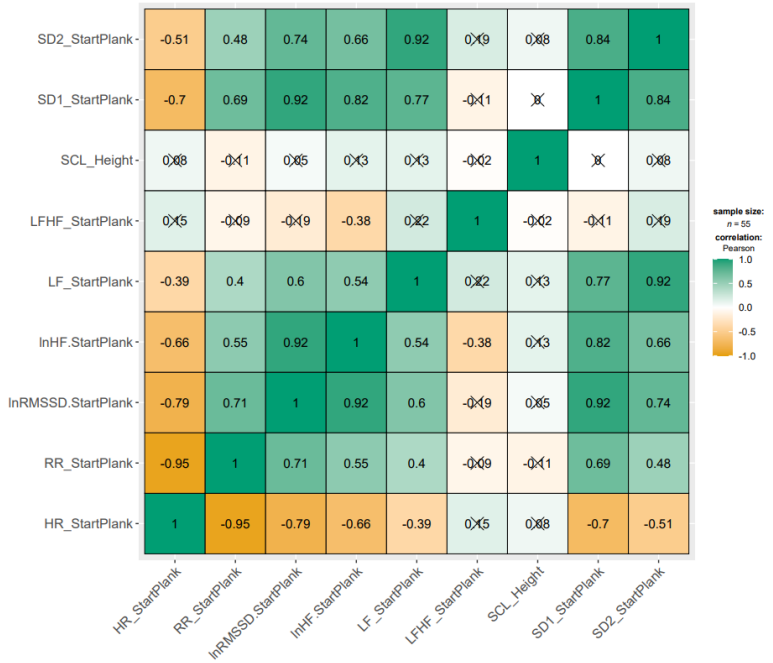

Figure 33 (B): Correlations among all physiological variables at the Start of Plank epoch

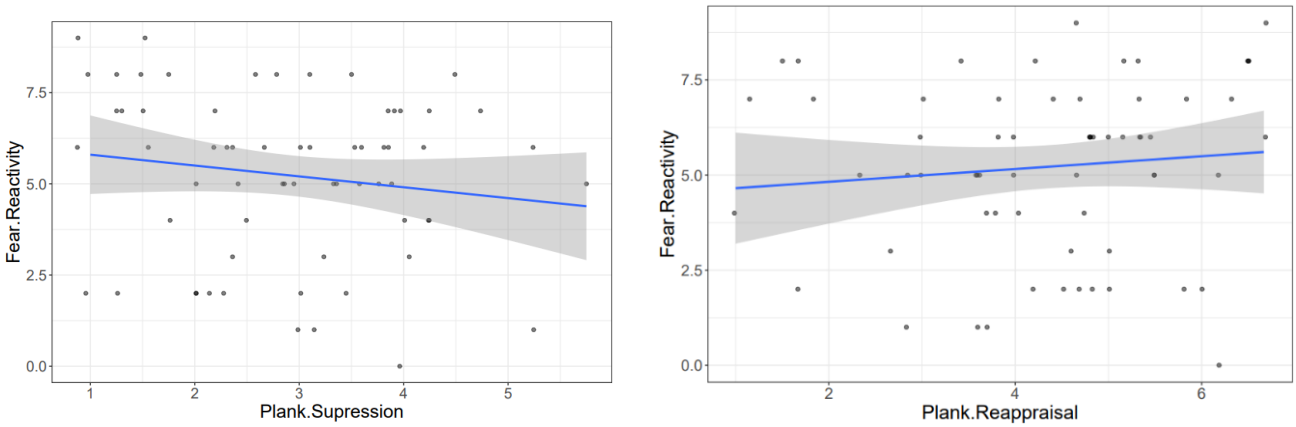

Figure 34: Emotion Regulation scores prediciting change in fear.
Neither is significant (p = .245; .853, respectively*).*

Appendix C (Study 3)

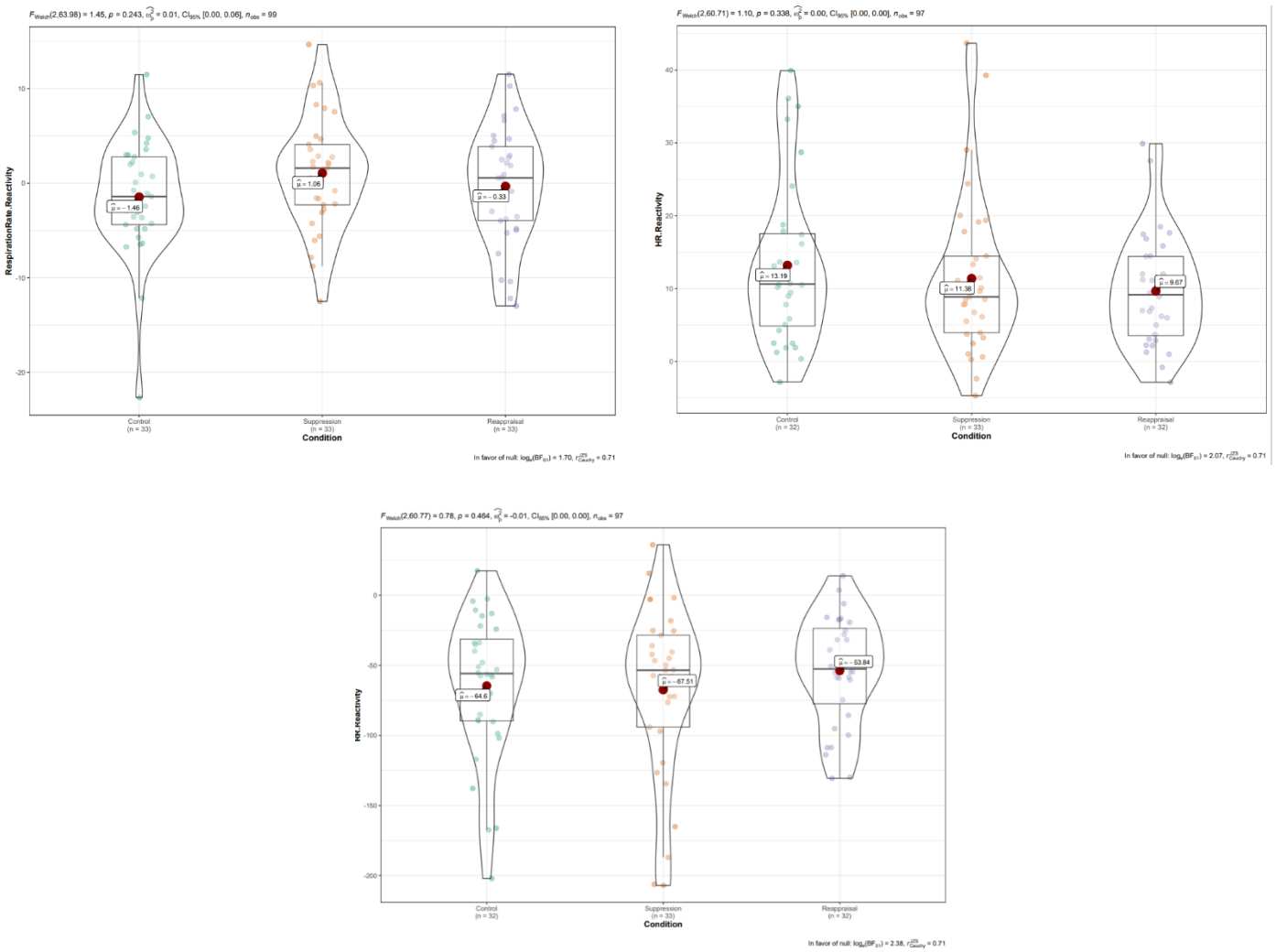

Figure 35 (C): Change in Respiration Rate (left) RR (bottom) and Heart Rate (right)
No significant difference between groups in change in either variable, ruling out that differences in HRV Reactivity can be explained by respiratory influences

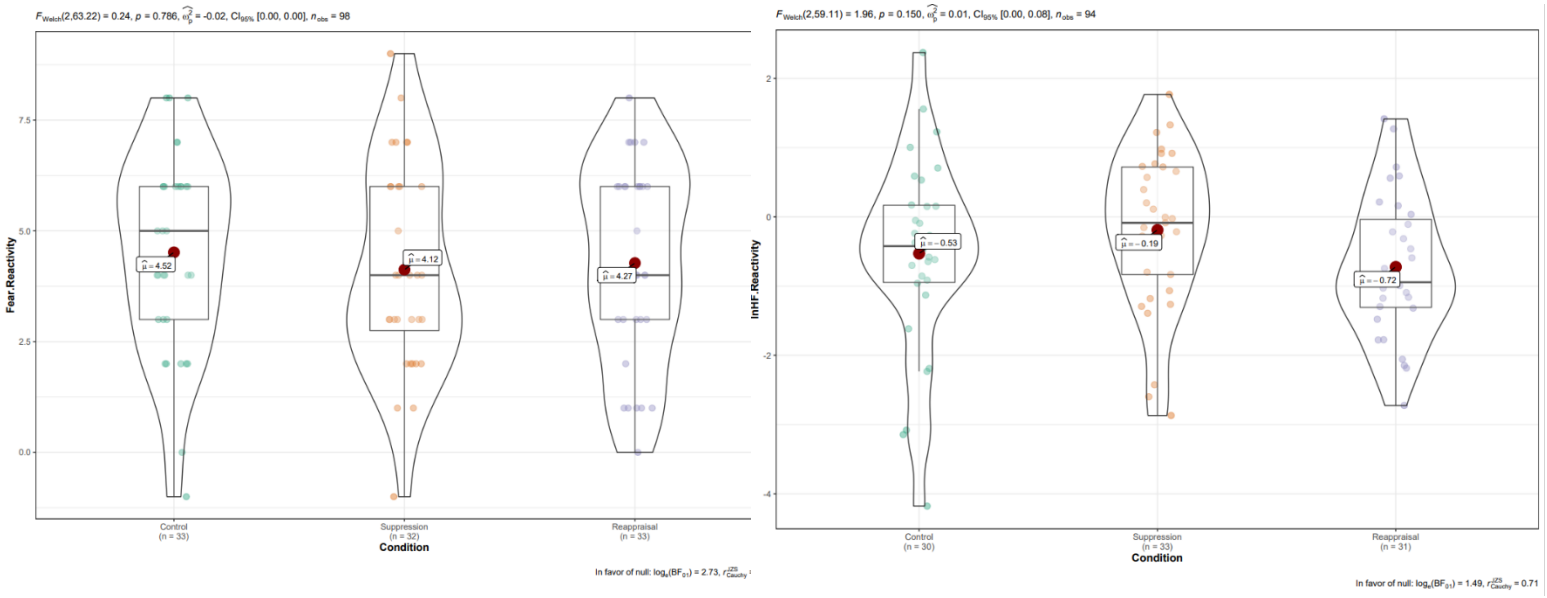

Figure 36 (C): Subjective Fear (left) and lnHF (right) reactivity scores by group

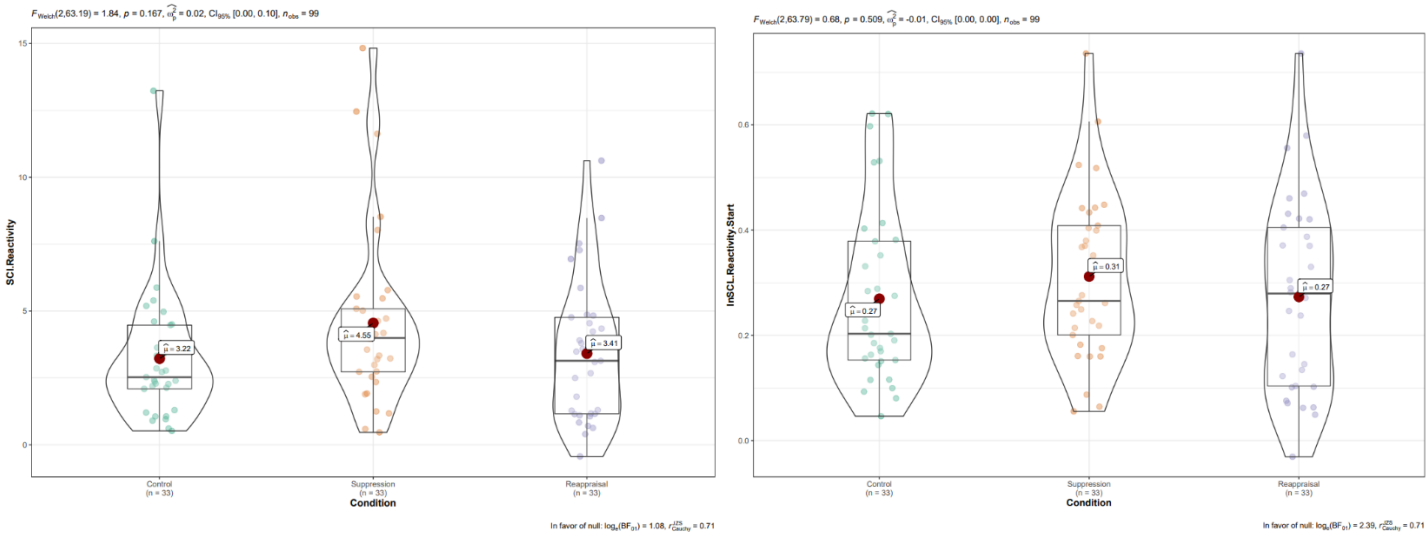

Figure 37 (C): raw skin conductance level (left) and log-transformed skin conductance (right) reactivity scores by group

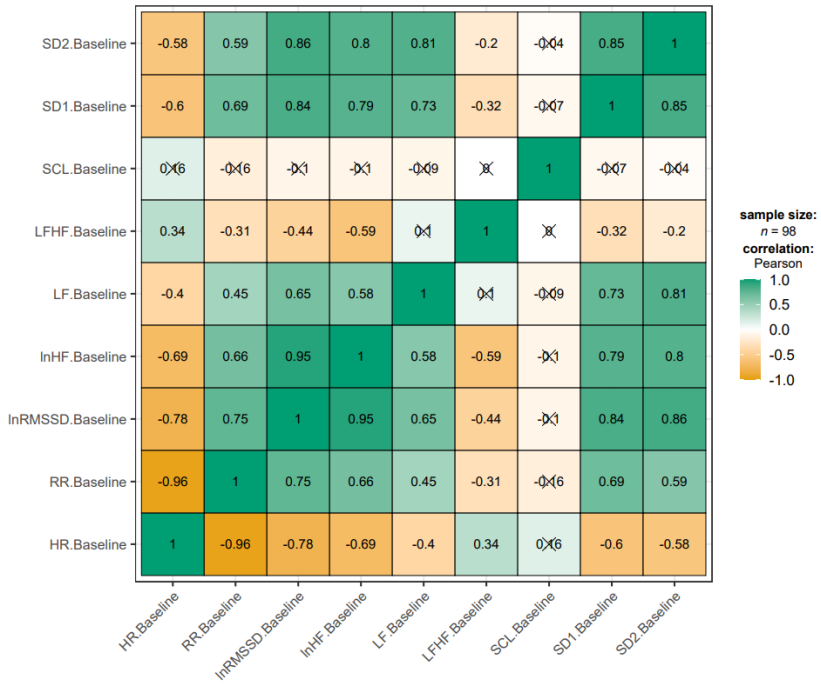

Figure 38 (C): Correlations Between all Psychophysiological Variables at the Baseline Epoch

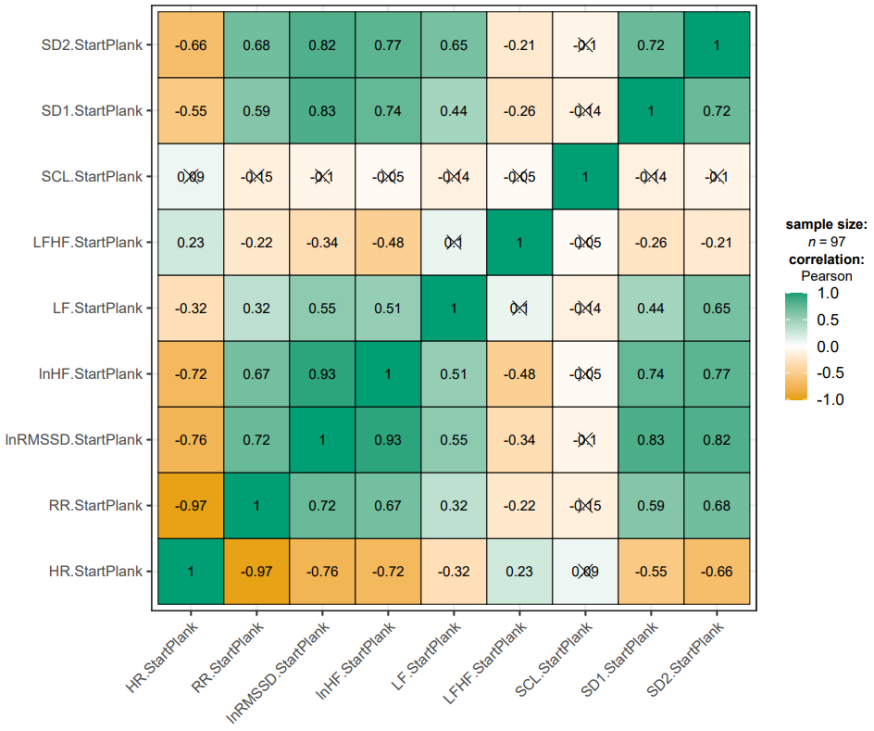

Figure 39 (C): Correlations Between all Psychophysiological Variables at the Start of Plank Epoch

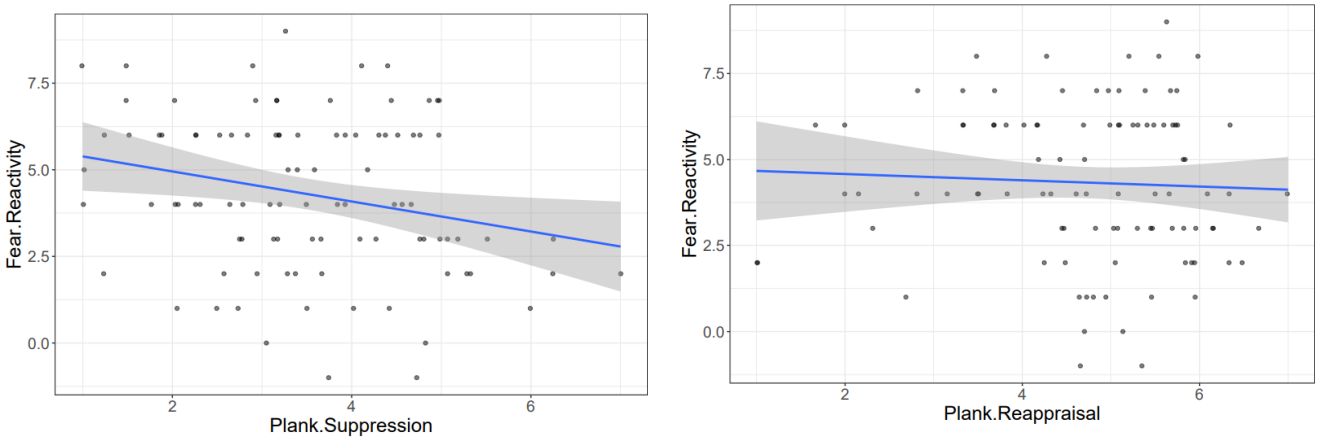

Figure 40 (C): Scatterplots, emotion regulation predicting change in fear

Self-reported use of Suppression (left) and Reappraisal (right), predicting change in fear ratings. Suppression was a significant predictor (r -.25, p = .012), Reappraisal was not (p = .658).