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The relationship between gratitude and cardiovascular responses to stress

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by

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Table of Contents

List of Tables

List of Figures

Abstract

Recent research has begun to highlight the potential for gratitude (trait, state, and interventions) to buffer against the negative effects of cardiovascular responses to stress. This thesis extends this research by examining the impact of trait gratitude on the risk of suffering acute myocardial infarction, the effects of state gratitude on cardiovascular reactivity and recovery, and the efficacy of a gratitude intervention in modulating cardiovascular reactivity. It presents three empirical studies addressing these objectives, thus providing new insights into the intersection of positive psychology and cardiovascular health. Study One's key novel finding is that there is an indirect inverse relationship between trait gratitude and the risk of acute myocardial infarction, mediated by heart rate reactivity. Study Two is the first study to suggest that state gratitude impacts both cardiovascular reactivity and recovery, further extending research on the relationship between gratitude and cardiovascular stress responses. Study Three makes use of a randomised controlled trial design, showing that a brief intervention can reduce cardiovascular stress reactivity. These studies constitute novel explorations of the longitudinal relationship between trait gratitude and risk of acute myocardial infarction, the protective relationship between state gratitude and cardiovascular recovery, and it also provides the first study to demonstrate the influence of a brief gratitude intervention on cardiovascular reactivity. These empirical analyses suggest a modest but significant relationship between gratitude and cardiovascular stress responses that is ultimately cardio-protective.

Publications and Presentations

Peer reviewed journal articles

Leavy, B., O'Connell, B. H., & O'Shea, D. (2023). Gratitude, affect balance, and stress buffering: A growth curve examination of cardiovascular responses to a laboratory stress task. *Inter- national Journal of Psychophysiology: Official Journal of the International Organization of Psychophysiology*, *183*, 103–116. https://doi.org/10.1016/j.ijpsycho.2022.11.013

Leavy, B., O'Connell, B. H., & O'Shea, D. (2023). Heart rate reactivity mediates the relationship between trait gratitude and acute myocardial infarction. *Biological Psychology, 108663*. Advance online publication. https://doi.org/10.1016/j.biopsycho.2023.108663

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Conference/Seminar Presentations and Lectures

Leavy, B. (2021, April 29). Gratitude and the cardiovascular response to stress. Presentation at the Psychology Research Day for Postgraduates & Post-Doctoral Researchers, Maynooth University, Kildare, Ireland.

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Leavy, B. (2023, May 3). Heart rate reactivity mediates the relationship between trait gratitude and heart attacks. Paper presentation at the GRASP seminar, Maynooth University, Kildare, Ireland.

Leavy, B. (2023, May 16). The results of two studies assessing the impact of gratitude on cardio- vascular health. Paper presentation at the Kemmy Business School research seminar, University of Limerick, Limerick, Ireland.

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Introduction

By the year 2030, it is a goal of the United Nations to reduce global mortality from non-communicable diseases by one third (United Nations, 2015). Globally, cardiovascular diseases are among the most common non-communicable disease and are responsible for an estimated 17.8 million deaths per year (Roth et al., 2018), with total costs estimated to increase by $1.1 trillion in the next 16 years (Cousin et al., 2021). According to the World Health Organization, this number is expected to reach 22.2 million by 2030 (Roth et al., 2018).

Emerging evidence has begun to highlight the role of non-traditional, psychological factors in the aetiology of cardiovascular diseases (Mulle & Vaccarino, 2013). Research has established the behavioural, physical and psychosocial risk factors associated with cardiovascular illness, such as physical inactivity, low social support, low socio-economic status, and obesity (Freak-Poli et al., 2021; Li et al., 2013; Li & Siegrist, 2012). Specifically, previous research has tended to focus on how negative emotions (e.g., depression, anxiety, and stress) are related to cardiovascular health (Hare et al., 2014; Kubzansky et al., 2018; Tully et al., 2016). For example, a meta-analysis of 20 studies found that anxiety was associated with increased likelihood of coronary heart disease (Roest et al., 2010), with depression linked to the development of cardiovascular diseases like hypertension (Meng et al., 2012) and myocardial infarction (Feng et al., 2019).

In line with this, decades of research have demonstrated psychological stress to be one of the major risk factors in the development of cardiovascular stress (Carroll, 2011; Steptoe & Kivimäki, 2012), with psychological stress being shown to be comparable to obesity as a risk

factor in the development of hypertension (Osborn et al., 2020). The effects of stress on the brain and body have been studied for decades (Yaribeygi et al., 2017). Stress can cause pathophysiological changes to the brain which can manifest as behavioural, cognitive, or mood disorders (Li et al., 2008). Individuals under stress are generally considered to have impaired immune systems and as a result suffer more frequently from illnesses (Khansari et al., 1990). Stress can affect appetite, either suppressing or exaggerating it (Nakamura et al., 2013), as well as disrupting the normal function of the gastrointestinal tract (Söderholm & Perdue, 2001). Stress has profound and diverse impacts, affecting brain structure and function, immune response, appetite, and gastrointestinal health.

Stress has also been associated with the onset and progression of cardiovascular illness (Carroll, 2011; Larzelere & Jones, 2008; Yaribeygi et al., 2017). A meta-analysis found that common daily stressors like loneliness and social isolation were associated with a 50% increased risk of suffering a cardiovascular disease incident (Steptoe & Kivimäki, 2012). Exposure to chronic, everyday stressors can elevate the risk of developing and dying from cardiovascular disease (B. E. Cohen et al., 2015). One study estimates that chronic stress, at both early life and adulthood, is associated with a 40–60% excess risk of coronary heart disease (Steptoe & Kivimäki, 2012) Prospective observational studies found that common daily stressors like loneliness and social isolation were associated with a 50% increased risk of incident cardiovascular disease incidents (Steptoe & Kivimäki, 2012). Similarly, the risk associated with chronic workplace stress was a 40% increase in cardiovascular disease incidence (Steptoe & Kivimäki, 2012).

Acute stress can also trigger adverse cardiovascular events (Eisenmann et al., 2016). In a study of 12,461 participants who had a first heart attack, 14.4% (N = 1,752) reported being angry or emotionally upset 1-hour period before to the onset of cardiac symptoms (Smyth et al., 2016). In the three weeks after the 2011 earthquake and tsunami in Japan there was a significant increase in patients at emergency departments with acute coronary syndrome and congestive heart failure (Nakamura et al., 2014). In Germany during the 2006 soccer world cup, there was a 2.66-fold increase in cardiac emergencies on the days when the German team was competing (Wilbert-

Lampen et al., 2008).

It has thus been suggested that we have departed from a world in which the primary cause of death is microbial in nature (Everly & Lating, 2019). In the USA, factors such as health-related behaviours and lifestyle patterns are now among the strongest predictors of these diseases (Sarafino & Smith, 2014). This has led to suggestions that the greatest threat to human health lies in human behaviour (Everly & Lating, 2019). The occurrence of stress-induced cardiovascular events underscores the significant health implications associated with psychological stress, setting the stage for further examination of how stress is associated with such negative outcomes. In this context, recent empirical research has begun to highlight how positive psychological constructs, such as positive affect, have stress buffering effects (Pressman et al., 2019). In line with this, the Model of Positive Well-Being (Boehm & Kubzansky, 2012) suggests that positive emotions both modulate cardiovascular reactivity and cardiovascular recovery, leading to long-term benefits for cardiovascular health. Concomitantly, recent research has also suggested that gratitude plays an important stress-buffering role (Wood et al., 2010). In light of accumulating evidence for the cardio-protective effects of gratitude, the Model of Positive Well-Being was adapted specifically for gratitude (Schache et al., 2019), a key prediction being that gratitude should positively impact cardiovascular health by buffering the effects of cardiovascular stress responses. These predictions have begun to be validated in empirical studies, with research demonstrating an inverse relationship between state gratitude and cardiovascular reactivity across a range of cardiovascular parameters (Gallagher et al., 2020; Ginty et al., 2020).

Aims and novelty

Despite advances in research, gaps in this picture remain. No longitudinal study has connected trait gratitude indirectly to reduced risk of cardiovascular incidents through cardiovascular reactivity. There has yet to be a study examining whether state gratitude is inversely related to both reactivity and recovery. Finally, the relationship between state gratitude and

cardiovascular reactivity has not been examined in a randomised controlled trial context. In filling these lacunae, this thesis has the following aims:

1. To examine how trait gratitude impacts heart health through the cardiovascular response to stress over time.
2. To examine the effects of state gratitude on cardiovascular reactivity and recovery and the interaction with affect.
3. To experimentally manipulate state gratitude to examine its effects on cardiovascular reactivity in a randomised controlled trial design.

In the context of positive psychology and cardiovascular health research, this thesis introduces several groundbreaking contributions that expand the current understanding of the interplay be- tween gratitude and physiological responses. Firstly, it pioneers the exploration of the connection between trait gratitude and negative cardiovascular outcomes, specifically acute myocardial infarction, through heart rate reactivity. This novel approach provides empirical support to theoretical frameworks like the Model of Psychological Well-Being (Boehm, 2021; Schache et al., 2019) and the Transdisciplinary Model of Stress (Epel et al., 2018), highlighting gratitude's potential role in cardiovascular health management. Moreover, this thesis presents the first evidence linking gratitude to long-term cardiovascular outcomes, particularly through its influence on stress reactivity. The implications of these findings are profound, suggesting that gratitude reduces cardiovascular reactivity, thereby providing a new theoretical perspective on the mechanisms through which gratitude impacts health.

Secondly, the research detailed herein presents two major and novel findings: (1) the presence of state gratitude results in both reduced reactivity and expedited recovery for systolic blood pressure, underscoring the comprehensive impact of state gratitude on the cardiovascular stress response profile; and (2) the balance of positive to negative emotions significantly interacts with state gratitude, particularly influencing the diastolic blood pressure stress response. This

interaction points to an amplification effect, extending previous research and suggesting that gratitude interacts with lower negative affect to yield stronger effects.

Thirdly, the thesis provides rigorous evidence that state gratitude effectively reduces reactivation in a randomised controlled trial study, this addresses a critical question in the field and minimises potential biases through methodological rigor and trial registration (clinicaltrials.gov Identifier: NCT05133063). Collectively, these studies underscore the significant stress-buffering functions of both state and trait gratitude, marking a substantial advancement in our understanding of gratitude's role in health psychology and stress management.

Overview

Chapters one and two examine previous literature examining stress, gratitude and cardiovascular health. Chapter one provides an in-depth exploration of the link between psychological stress and cardiovascular disease. It delves into the mechanisms through which stress contributes to cardiovascular issues, both acutely and chronically, and discusses various theoretical models of stress, such as the Transactional Model of Stress (Biggs et al., 2017). Chapter two thoroughly examines the concept of gratitude, examining its classification as an emotion. It argues for the potential of gratitude as a tool to buffer against stress, focusing on both its state and trait forms. Chapter three concludes by describing the aims and contributions of the specific empirical studies presented in this thesis.

Chapter four provides an overview of the methodology of this thesis. It describes the guiding philosophical commitments of the thesis, the quantitative approach this research takes, the experimental designs and procedures for inducing stress and assessing its biological markers.

Chapters five to seven contain the three empirical studies completed as part of this thesis. Chapter five reports the results of a study investigating the long-term indirect relationship between trait gratitude and the risk of acute myocardial infarction through cardiovascular reactivity. Employing mediation analysis, the study found that trait gratitude is indirectly linked to a reduced risk of adverse cardiovascular outcomes, specifically heart attacks, through its relationship with

heart rate reactivity. This suggests a long-term protective role of trait gratitude against cardiovascular strain from daily stress, providing a new perspective on how trait gratitude might counterbalance the potential for adverse cardiovascular outcomes associated with increased reactivity.

Chapter six reports the results of a within-subjects experimental study, aimed as assessing the relationship between state gratitude and cardiovascular reactivity and recovery. There were two key findings: first, that state gratitude resulted in both reduced reactivity and hastened recovery for systolic blood pressure, thereby improving the overall cardiovascular stress response profile; and second, that a balance of more positive to negative emotions significantly interacted with state gratitude, amplifying its effects on the diastolic blood pressure stress response. This indicates a novel amplification effect of positive emotions on state gratitude's impact on cardiovascular stress responses.

Chapter seven reports the results of a study that utilized a randomized control trial design to test the efficacy of a brief state gratitude intervention. The primary outcome revealed that the intervention successfully reduced systolic blood pressure reactivity compared to a control group, confirming that state gratitude actively reduces reactivity and is not merely a consequence of reduced reactivity.

Finally, chapter eight discusses the overall findings of this thesis. In summary, this thesis not only fills critical gaps in the existing literature but also pioneers new pathways in understanding the complex relationship between gratitude and cardiovascular health. Through a meticulous blend of theoretical exploration and empirical investigation, it elucidates how both trait and state gratitude play a pivotal role in modulating cardiovascular responses to stress. The novel insights garnered from these studies offer a more nuanced understanding of gratitude's protective mechanisms against cardiovascular diseases. This work stands as a testament to the protective potential of positive psychological constructs in health sciences, paving the way for future research and interventions aimed at enhancing cardiovascular health through psychological well-being.

Chapter 1

Psychological stress and cardiovascular disease

1.1 Theories of stress

Psychological stress is a complex phenomenon with many posited theoretical models attempting to explain its aetiology (Biggs et al., 2017; Francis et al., 2017). The most commonly used of these is the Transactional Model (Engert et al., 2019; Francis, 2018; Meijen et al., 2020; Trotman et al., 2018; Uphill et al., 2019). In transactional definitions of stress, stress occurs when a person judges that environmental stimuli are placing demands on them that exceed their ability to meet with, mitigate, or alter those demands (Epel et al., 2018). It is most commonly associated with Lazarus and Folkman (1984).

1.1.1 Lazarus and Folkman's (1984) Transactional Model of stress

Lazarus and Folkman's (1984) transactional theory of stress and coping state that individuals are constantly appraising their environment (Epel et al., 2018; Lazarus, 1999). When environmental stimuli are appraised as threatening or harmful (i.e. as stressors), the resultant

feelings of distress prompt the use of coping strategies. By coping, an individual may attempt to modify their environment, or their appraisals of the environment in either a positive or negative manner (Biggs et al., 2017). According to this view, stress is defined as a response to exposure to stimuli which are noxious and which exceed our coping capacity (Biggs et al., 2017). Thus, the intensity of a stress response is influenced by the role of the appraisal.

While the transactional model of Lazarus and Folkman offers a foundational perspective on stress, its decades-old framework may benefit from contemporary insights that reflect the evolving understanding of stress physiology. The foundational principles of stress, appraisal, and coping remain pivotal in contemporary theories of stress (Epel et al., 2018). Indeed, these principles underpin the more recent biopsychosocial model of challenge and threat (Blascovich & Mendes, 2010).

1.1.2 The biopsychosocial model of challenge and threat

The Biopsychosocial Model of Challenge and Threat (Blascovich & Mendes, 2010) describes how psychological appraisals of demands and resources influence cardiovascular responses, signifying either a challenge or a threat state in goal-oriented activities (Seery, 2013). According to the biopsychosocial model of challenge and threat, stressors are appraised on two dimensions: the demands placed upon the individual, and the resources available to meet those demands (Epel et al., 2018). The demands are appraised in terms of uncertainty, perceived danger, and the effort required to meet those demands (Blascovich & Mendes, 2010). Resources are assessed in terms of an individual's disposition, as well as the perception of any relevant knowledge, skills or support (Blascovich & Mendes, 2010). Combining these perceptions of demands and resources leads to two possible appraisals: challenge and threat (Epel et al., 2018). Challenge appraisals result from appraising high resources and low demands, whereas threat results from appraising high demands and low resources (Blascovich, 2013). Challenge and threat are characterised by different physiological profiles (Uphill et al., 2019). For example, both challenge and threat result in an elevated heart rate, but challenge results in dilated arteries and more being blood pumped, whereas

threat results in constricted arteries and less being blood pumped (Seery, 2013).

In general, a tendency towards threat appraisals with their accompanying physiological profiles is supposed to be associated with worsened health outcomes, especially in comparison with a tendency towards challenge appraisals (Blascovich & Mendes, 2010; Epel et al., 2018). For example, threat appraisals in response to acute stressors in a laboratory context are associated with increased cellular aging (O'Donovan et al., 2012). Whereas challenge appraisals are associated with in- creased performance (Trotman et al., 2019) and better decision making (Kassam et al., 2009), as well as being associated with greater cognitive processing speed in older adults (Jefferson et al., 2015). Overall threat appraisals have been associated with more maladaptive and harmful reactions to stress (Jamieson et al., 2018). However, there is a lack of research to indicate the mechanisms by which a disposition towards challenge appraisals may lead to long-term health benefits, leading to alternate models (Epel et al., 2018).

1.1.3 The Transdisciplinary Model

Where the Biopsychosocial model of challenge and threat (Blascovich, 2013; Seery, 2013) is characterised by a narrow focus on challenge and threat appraisals, the Transdisciplinary Model of Stress (Epel et al., 2018) integrates a large number of factors and stressors in the description of the stress response and its subsequent health implications (Schneiderman et al., 2005). In this model, stress is described as a set of interactive and emergent experiences that occur with respect to one's individual-level characteristics like personality and demographics, as well environmental factors like socioeconomic environment (Epel et al., 2018). For example, an individual's socio-economic context offers a framework within which experiences are interpreted and made sense of (Worthman, 2010), and this will influence whether a stressor is appraised as either threatening or challenging (Chen & Matthews, 2001).

The Transdisciplinary Model acknowledges the cumulative impact of historical and ongoing stressors, emphasizing how past experiences shape current stress perceptions and responses (McEwen, 2017). It suggests that long-term exposure to stress, whether through chronic conditions

or recurring acute events, can progressively strain an individual's adaptive capacity (Seery, 2011). This cumulative stress burden influences how new stressors are appraised and managed, potentially heightening vulnerability to future stress (Epel et al., 2018).

In this model, the acute stress response is one that unfolds momentarily in response to a stressor and involves multisystem physiological responses (Epel et al., 2018). While many aspects of an acute response are actually protective (Kagan, 2016; Uphill et al., 2019), certain profiles have been associated with more damage than protection due to a mismatch between the physiological response and the situational demands (Doan, 2021). This model suggests that the reason why stress exposure leads to long-term health outcomes is that repeated physiological reactivity and maladaptive response profiles leads to greater allostatic load, or wear and tear over time (Doan, 2021; Whittaker et al., 2021). This manifests as either exaggerated reactivity to a stressor, heightened anticipation prior to a stressor, prolonged recovery, or even blunted cardiovascular reactivity (Epel et al., 2018). These maladaptive reactivity patterns have variously been linked to worsened health outcomes, such as hypertension (Spruill, 2010), or atherosclerosis (Low et al., 2009), hence constitute important areas of continued research (Whittaker et al., 2021).

1.2 Stress and cardiovascular reactivity and recovery

While stress may be unavoidable, how we respond to it can have differential and sometimes detrimental effects on cardiovascular health (Osborne et al., 2020). Specifically, atypical cardiovascular reactions to stress (if exaggerated, prolonged, or blunted) are associated with the onset and progression of cardiovascular disease over time (Carroll et al., 2012; Phillips et al., 2011). This has been called the reactivity hypothesis (Whittaker et al., 2021). Distinct from cardiovascular adjustments during physical exertion which align with metabolic demands, reactions to psychological stress appear metabolically exaggerated (Lovallo, 2005). This observation has been further substantiated by studies in which cardiovascular activity during stress exceeded predicted levels based on oxygen consumption measures from graded exercise (Balanos

et al., 2010; Carroll et al., 2009). Such pronounced cardiovascular reactions to psychological stress, in contrast to the adaptive adjustments during physical activity, have been shown to be potentially pathophysiological (Carroll et al., 2012).

Direct evidence supporting the reactivity hypothesis stems from multiple large-scale studies indicating positive correlations between cardiovascular reactions to acute psychological stress and future blood pressure issues, hypertension, systemic atherosclerosis markers, and heart hypertrophy (Balanos et al., 2010; Carroll et al., 2001; Carroll et al., 2003, 2009; Carroll, 2011; Gianaros et al., 2002; Kapuku et al., 1999; Manuck et al., 1992; Markovitz et al., 1998; Steptoe, 2008; Treiber et al., 2003). Although effect sizes are generally modest, they align with the reactivity hypothesis's core principles. For example, a meta-analysis of 36 studies found a small yet statistically significant relationship between cardiovascular reactivity and an index of poor cardiovascular status (e.g. hypertension, clinical cardiac events, and subclinical atherosclerosis), ($r = 0.091$; Chida & Steptoe, 2010). In the same study, this finding was further confirmed by more conservative analyses of aggregated effects ($r = 0.13$) (Chida & Steptoe, 2010).

Where cardiovascular stress reactivity can be defined as the response to challenging conditions, cardiovascular stress recovery refers to the response following cessation of challenging situations (Panaite et al., 2015). Delayed cardiovascular recovery from stress is also associated with serious health problems such as hypertension and even cardiovascular death (Hocking-Schuler & O'brien, 2007; Kivimäki et al., 2006; Kivimäki & Steptoe, 2018). Indeed, cardiovascular recovery has plausibly emerged as a more useful predictor of hypertension than cardiovascular reactivity (Radstaak et al., 2011). This is because where reactivity involves the body's immediate response to stress, recovery is about how quickly the cardiovascular systems returns to baseline after stress (Panaite et al., 2015). Poor recovery indicates that the body remains in a heightened stress state for a longer duration, leading to prolonged exposure to stress hormones and increased cardiovascular strain (Qiu et al., 2017). This prolonged exposure can result in additional sustained damage to the cardiovascular system alongside reactivity (Panaite et al., 2015). Hence, inadequate recovery may be a more consistent predictor of long-term cardiovascular issues than reactivity.

An often implicit assumption in this line of research is that low physiological reactivity to psychological stressors is inherently protective or benign in nature (Carroll et al., 2003; Light & Obrist, 1983). Recent challenges to this assumption have emerged, suggesting that low reactivity may not always signal adaptive or positive health outcomes (Carroll et al., 2017; Lovallo et al., 2019; O' Riordan et al., 2022). For example, the West of Scotland Twenty-07 study (Benzeval et al., 2009). indicated that individuals from lower socioeconomic statuses or those who rated their life events as highly stressful exhibited blunted cardiovascular responses to stressors (Phillips et al., 2011; Robertson et al., 2014). Similarly, blunted reactivity has been found to characterise smokers (Ashare et al., 2012), individuals with high adiposity (Phillips, 2011), and alcoholics (Lovallo et al., 2019). Such findings introduce complexity into our understanding of the relationship between cardiovascular reactivity and health, suggesting that both exaggerated and blunted responses to stress may hold distinct health implications.

1.2.1 How are reactivity and recovery associated with worsened outcomes?

In accordance with the Transdisciplinary Model of Stress, maladaptive responses to acute stress that activate and then very quickly shut off do not harm the body (Epel et al., 2018). Moreover, as previously noted, some physiological responses to stress (i.e. challenge appraisals) are beneficial (Panaite et al., 2015; Uphill et al., 2019). However, if the response is sustained, exaggerated, or is delayed in returning to baseline, it can harm the body (Epel et al., 2018). Fundamentally, this has been associated with wear-and-tear in the body (Whittaker et al., 2021).

Allostatic load is often used to understand this (Whittaker et al., 2021). Allostatic load describes the physiological adaptations the body makes in response to environmental stressors (Juster et al., 2010; McEwen & Stellar, 1993). The Transdisciplinary Model of Stress defines allostatic load as the cumulative strain on various bodily systems due to prolonged or intense stress (Epel et al., 2018). This strain pushes these systems to a state of persistent overactivity, underactivity, or

erratic activity, even after the initial stress has subsided (Epel et al., 2018). The ongoing imbalance and resulting stress response can cause long-term damage to the body (Doan, 2021).

Different patterns of maladaptive response patterns have been proposed, including repeated physiological stress responses (e.g. through social or economic adversity (Geronimus et al., 2006)), lack of adaptation to repeated stress (e.g. through lowered resilience (Cameron & Schoenfeld, 2018)), inadequate responses to stress (e.g. through motivational disengagement (Hase et al., 2020)), and a prolonged stress response (McEwen, 2000). Over time, such continual physiological adaptations might predispose individuals to several chronic diseases (Juster et al., 2010; McEwen, 1998; McEwen & Stellar, 1993). For example, a recent meta-analysis of eight studies concluded that high allostatic load was associated with a 31% increased risk cardiovascular disease mortality (Parker et al., 2022).

The Transdisciplinary Model of Stress also suggests that the interaction between chronic and acute stressors may also be useful in understanding the link between stress and health outcomes (Epel et al., 2018). Acute stress is an intense, short-term experience (Eisenmann et al., 2016). Chronic stressors are present over longer periods of time (Spruill, 2010). Chronic stressors predict increased acute stress (Hammen et al., 2009), as well as increased threat appraisals of acute stressors in laboratory research settings (O'Donovan et al., 2012). This suggests that chronic stress increases the frequency of acute stress and maladaptive responses to them (Rohleder, 2019). For example, financial strain is a chronic stressor associated with increased perceived stress (Park et al., 2017), blunted responses to acute stressors (Steptoe et al., 2020), and increases in the odds of suffering myocardial infarction (Moran et al., 2019). This suggests that the context of a person's life shapes the frequency of daily stressors they experience and their reactions to them, meaning that moment-to-moment responses to acute stress are important in understanding the relationship between stress and health (Epel et al., 2018).

The mechanism associating reactivity and recovery with the development of cardiovascular dis- ease is that small, persistent elevations in blood pressure in response to stress are thought to place additional strain on the heart and blood vessels over time (Epel et al., 2018; Whittaker et al.,

2021). Levine (2022) outlines a number of pathways by which both acute and chronic stress can lead to conditions from hypertension and cardiac arrythmia to acute myocardial infarction (heart attacks).

According to Levine (2022), a key process is atherogenesis, which refers to the pathophysiological process of atherosclerotic plaque development (Cichoń et al., 2017), leading to atherosclerosis. This is a disease that is characterized by the build-up of lipids and fibrous elements in the large arteries (Lusis, 2000). Chronic stress has been linked to atherosclerosis through the activation of the hypothalamic-pituitary-adrenal axis, greater sympathetic-adrenal-medullary activity, and greater peripheral sympathetic nervous system activity (Levine, 2022), leading to build-ups of vulnerable arterial plaque. Acute stress can then underpin the rupture of this plaque, leading to acute myocardial ischemia and infarction, through processes such as endothelial dysfunction, platelet activation and coronary vasoconstriction activity (Levine, 2022; Zupancic, 2009).

Cardiovascular reactivity has been found to be associated with increased atherosclerosis (Gianaros et al., 2002; Roemmich et al., 2011), as well as increased vascular resistance, hardening of the vessel walls, and higher inflammatory reactions which are associated the progression hypertension and atherosclerosis (Lovallo, 2005; Whittaker et al., 2021). Similarly, delayed cardiovascular recovery has been associated with hypertension (Steptoe & Marmot, 2005) and atherosclerosis (Jae et al., 2008). Other regulatory mechanisms may also be implicated in this process, such as greater or altered beta-adrenergic receptor density or sensitivity (Kelsey et al., 2010) increased left ventricular mass or wall density (Taylor et al., 2003), and even altered neurophysiological activity, for example in the amygdala (Trotman et al., 2019) which has been implicated in arterial inflammation (Tawakol et al., 2017).

Thus, the combination of the Transdisciplinary Model of Stress (Epel et al., 2018) and allostatic model (Guidi et al., 2020) may suggest that stress exposure impacts cardiovascular health by a process of cumulative strain, which may be reflected in heightened or blunted cardiovascular reactivity to acute stress and delayed recovery from acute stress (Epel et al., 2018; Low et al.,

2009). However, a recent review of the reactivity literature concluded that the exact causal mechanisms by which reactivity affects arterial wall biology or the structure of the heart remain to be elucidated (Whittaker et al., 2021). It is also worth mentioning that examining cumulative lifetime stressor exposure is a difficult, time-consuming process, leading to the use of retrospective measures of cumulative stress experiences, the validity of which are questionable (Newbury et al., 2018). Nonetheless, much evidence points towards laboratory-assessed reactivity and recovery as a relatively stable trait (Rutledge et al., 2000, 2001), indexing "wear and tear" on the cardiovascular system (Whittaker et al., 2021), and representing adaptive or maladaptive stress coping (Cavanagh & Obasi, 2021; Howard et al., 2017; Mrug et al., 2023), making reactivity and recovery important endpoint sin the study of stress and cardiovascular disease (Zanstra & Johnston, 2011).

1.2.2 Psychosocial predictors of reactivity and recovery

The Transdisciplinary Model of Stress suggests that repeated exposure to acute stressors leads to worsened health outcomes through wear-and-tear (Epel et al., 2018). This prediction is validated by a range of empirical studies (Eisenmann et al., 2016; Garfin et al., 2018). However, there is tremendous variability in how vulnerable one is to stress (Kagan, 2016), with a range of variables co-varying with or moderating the stress response (Pidgeon et al., 2014). For example, blood pressure reactivity increases with age (Brindle et al., 2014), whereas heart rate reactivity and recovery tend to decrease (Pearman et al., 2021; Shcheslavskaya et al., 2010). These relationships are likely due to age-related declines in sympathetic nervous system responsiveness (Uchino et al., 2010).

Similarly, there is evidence that psychological factors impact reactivity and recovery (Whittaker et al., 2021). Adverse childhood experiences have been associated with increases in blood pressure in laboratory stress tasks (Dempster et al., 2023). Personality type has received a lot of attention (O'Riordan, Gallagher, et al., 2023), with type D personality associated with lowered reactivity (O'Riordan, Howard, et al., 2023). Research has also focused on the more

negative dimensions of the big five, with reactivity and recovery being associated with neuroticism (Hutchinson & Ruiz, 2011; Jonassaint et al., 2009) and low openness (Soye & O'Súilleabháin, 2019). Though, a more recent meta-analysis concluded that trait neuroticism has no relationship with reactivity (Ahmad et al., 2021). However, while socioeconomic status does not predict an exaggerated cardiovascular response to stress, it is inversely associated with delayed recovery (Boylan et al., 2018). These results highlight the importance of considering a multitude of biopsychosocial factors when assessing the impact of stress on cardiovascular health outcomes.

Psychological buffers of the impact of stress on cardiovascular reactivity and recovery from stress

In the Transdisciplinary Model of Stress, protective factors are acknowledged as crucial elements that modulate an individual's resilience to stress (Epel et al., 2018). These factors, typically characterized as malleable social, psychological, and behavioural traits, play a significant role in enhancing an individual's ability to withstand or recover from stress. Examples include supportive family structures and engaging in a physically active lifestyle (Sharma & Singh, 2019; Tsatsoulis & Fountoulakis, 2006). The theoretical rationale behind these protective factors lies in their capacity to buffer the adverse effects of stress, thereby promoting healthier stress response patterns and reducing the risk of stress-related health issues (Epel et al., 2018).

A number of studies show that positive constructs such as positive affect (Pressman et al., 2019) may also play this protective role, or buffering role, with respect to stress reactions (Parra-Gaete & Hermosa-Bosano, 2023). Useful evidence comes from laboratory studies, where stress and positive affect are manipulated, support a buffering effect for positive affect (Fredrickson & Levenson, 1998; Pressman et al., 2019) and optimism (Parra-Gaete & Hermosa-Bosano, 2023). Similarly, personality factors such as high conscientiousness has been found to moderate the effects of variables such as life stress on cardiovascular reactivity (Gallagher et al., 2018).

The potential for the buffering effect of positive constructs is underscored by a recent meta-

analysis of 347 studies which found moderate effects of positive psychology interventions on stress (Carr et al., 2021). In this context, gratitude interventions were one of only two interventions (the other being humour) to significantly impact stress (Carr et al., 2021). Yet, despite much robust work on psychosocial stress moderators on reactivity and the growth in the literature on the benefits of gratitude for health (e.g. Boggiss et al., 2020), scholars have only begun to explore how gratitude, through positively appraising daily life, physiologically influences cardiovascular reactions to stress (Gallagher et al., 2020; Ginty et al., 2020).

1.3 Conclusion

In sum, this chapter covered the relationship between stress and cardiovascular illness. It demonstrated that stress, chronic and acute, is associated with negative outcomes. Commensurate with the Transdisciplinary Model of Stress, it suggested a primary mechanism by which stress negatively impacts the cardiovascular system is through wear and tear due to maladaptive stress responses. It was suggested that exaggerated or blunted reactions to stress and/or delayed recovery from stress were key, cardiotoxic response profiles. It thus characterized cardiovascular reactivity and recovery and finally it reviewed psychosocial buffers of these responses. In the next section, I will focus on gratitude as a specific psychosocial buffer of stress.

Chapter 2

Why target gratitude?

Prima facie, gratitude is a plausible candidate as a moderator of the effects of stress on health. It predicts lowered frequencies of acute stress (Lee et al., 2021), overall increases in well-being (Carr et al., 2021), and better health outcomes (Boggiss et al., 2020). However, the nature of gratitude is multifaceted; it has been considered as an emotion, habit, virtue, and disposition, leading to significant conceptual variations in academic discourse (Emmons et al., 2003; Emmons, 2004; Emmons & Crumpler, 2000; Froh et al., 2011). In light of this, the following sections will deal with what gratitude is, whether it is an emotion, and specifically a positive emotion, what the value of gratitude is, does gratitude buffer the effects of stress, and how it might buffer the effects of stress.

2.1 What is gratitude?

Gratitude has been classified into a broad number of categories (Emmons, 2019). It has been viewed as a virtue, an emotion a habit, a coping response as well as an attitude (Emmons, 2004, 2016; McCullough et al., 2004). This has led to substantial heterogeneity about the nature of gratitude (Wood et al., 2010). Useful evidence regarding the nature of gratitude comes from prototype studies (McCullough, 2002; Morgan et al., 2014). Prototype studies identify the

meanings and descriptions of concepts by asking individuals what properties they associate with a given concept (Morgan et al., 2014). In one such study, the results distinguished between two forms of gratitude: gratitude felt specifically in response to a received benefit from a specific benefactor, em- phasising its interpersonal nature (McCullough, 2002), and a generalised form of gratitude which an individual experiences as a result of awareness of what is valuable and meaningful in their life (Lambert et al., 2009). These correspond to state and trait gratitude (Wood et al., 2008b).

This division of gratitude into state and trait forms is based on Rosenberg's (1998) description of states and traits as hierarchical levels of analysis. Traits are predispositions towards certain kinds of emotional responses (Rosenberg, 1998), and thus are characterised by the frequency with which emotions are experienced in daily life (Wood et al., 2008a). At the state level, emotions involve the experience of temporary affects, which may be associated with particular thought or action tendencies (Rosenberg, 1998). Generally, gratitude has been studied almost exclusively at the levels of states and traits (Wood et al., 2008a; Youssef-Morgan et al., 2022).

As a state, gratitude refers to momentary feelings of appreciation for the good one has in their life (Wood et al., 2008a). Gratitude has been characterized by daily fluctuations of thankfulness (Spence et al., 2014). This variability supports the concept of gratitude as a state (Rosenberg, 1998). Moreover, state gratitude is associated with specific situational appraisals, and is not a permanent disposition, differentiating it from trait gratitude (Wood et al., 2008b; Youssef-Morgan et al., 2022).

As a trait gratitude refers to the predisposition to appreciate the good one has in their life (Wood et al., 2010). This stability is evidenced by longitudinal studies that demonstrate a stable tendency to recognize and respond with thankfulness across varied life circumstances (Wood et al., 2008b). This disposition is linked to higher well-being and positive social outcomes (Chopik et al., 2019; Portocarrero et al., 2022), suggesting a durable quality rather than a transient state. Similarly, individuals with a grateful disposition exhibit a frequent recognition of and appreciation for assistance and support (Wood et al., 2010).

According to Wood et al. (2008)'s social-cognitive model, trait and state gratitude are connected by a process whereby trait gratitude influences the appraisal of beneficial events, leading to state gratitude (Wood et al., 2008a). Individuals with higher levels of trait gratitude tend to make more positive appraisals of help received, which mediates their experience of state gratitude (Wood et al., 2008a). These appraisals include percep- tions of the value of the help, the perceived cost to the benefactor, and the benefactor's altruistic intent. This highlights that trait gratitude consistently enhances the frequency and intensity of state gratitude experiences (Wood et al., 2008b).

Studying gratitude in both its state and trait forms offers a comprehensive perspective on its role in human psychology and well-being. State gratitude provides insights into immediate emotional responses and their short-term benefits, such as increasing pro-social helping behaviours (Ma et al., 2017). In contrast, trait gratitude, a stable disposition towards appreciating life's positives, delves into long-term patterns and their sustained benefits, such as enhanced life satisfaction (Kong et al., 2020) and improved physical health (Hill et al., 2013).

While state and trait gratitude describe different dimensions of how we experience and express gratitude, their emotional underpinnings segue into a broader exploration: Is gratitude itself a distinct emotion? This leads me to consider gratitude within the wider landscape of emotional theories. In this context both states and traits have been implicated in broader discussion of what constitutes an emotion (Naar, 2022; Rosenberg, 1998). For example, while traits have been suggested to govern the threshold for the activation of emotions (Kaspar & König, 2012), they are often distinguished from the emotion itself (Ekman, 1992, p. 174). Nonetheless, the distinction between the two has often been held to be fuzzy (Lance et al., 2021), with Hertzog & Nesselroade (1987) often quoted as saying "Generally it is certainly the case that most psychological attributes will neither be, strictly speaking, traits or states. That is, attributes can have both trait and state components" (p. 95). Therefore, in exploring gratitude as a distinct emotion, it is practical to consider how its stable, trait-like characteristics interplay with its more fleeting, state-like aspects. This holistic approach could provide a more nuanced and complete picture of gratitude's place in

the spectrum of human emotions.

2.1.1 Is gratitude a distinct emotion?

Is gratitude a distinct emotion? As noted above, gratitude has also been very commonly regarded as an emotion (Wood et al., 2010). Emotions have historically been the subject of various interpretations (Harmon-Jones et al., 2017). Predominantly, two perspectives have emerged: (1) the categorical and (2) the dimensional views (Keltner & Lerner, 2010). The categorical perspective, rooted in the work of researchers like Ekman (1992) and Izard (2007), posits that there are dis- tinct, primary emotions, each with its unique physiological and behavioural signature (Consedine & Moskowitz, 2007; Ekman, 1992). It suggests that each primary emotion is universally recognizable and has an evolutionary basis (Ekman, 1992; Izard, 2007). This perspective implies a finite set of emotions, each with specific, cross-culturally consistent expressions and physiological patterns (Consedine & Moskowitz, 2007).

The dimensional view, however, conceptualizes emotions more fluidly, without clear boundaries, where emotions are characterized by their position on continuous scales, such as pleasantness-unpleasantness and activation-deactivation (Barrett, 2006; Russell, 1980). This model advocates for a more nuanced understanding that allows for a broader and more individualized interpretation of emotional experiences (Harmon-Jones et al., 2017; Posner et al., 2005). Debate continues between these two positions (Lindquist et al., 2013), with some attempting to reconcile the positions (e.g. Harmon-Jones et al., 2017; Shiota et al., 2017). For example, Harmon-Jones et al. (2017) argue that both dimensional models and discrete models have advantages. Where dimensional models highlight how emotions vary along continua like arousal and valence, discrete models emphasize the specific adaptive functions of different emotions. Harmon-Jones et al. (2017) present evidence demonstrating that affective valence within discrete emotions can vary depending on individual factors and situational contexts. Thus, they suggest that integrating both views leads to a more nuanced appreciation of emotional complexity and the

varying roles emotions play in our lives (Harmon-Jones et al., 2017). Taken together, this suggests that we can use criteria from both to evaluate whether gratitude can be categorised as a distinct emotion.

According to categorical emotion theorists (Izard, 2007), there are several primary, distinct emotions that have unique characteristics, including specific antecedents, physiological responses, and specific facial or behavioural expressions (Ekman, 1992; Izard, 2007). Through this lens, gratitude emerges as its own unique emotion. It arises from specific antecedents (e.g., from the receipt of monetary gifts; Karns et al. (2017)), primarily when an individual perceives a benefit they've received as intentional and not due to their own actions (Emmons, 2019; McCullough, 2002). Physiologically, experiences of gratitude have been linked to specific brain activation patterns, particularly in regions associated with social bonding and reward (Zahn et al., 2009). Gratitude consistently activates the anterior cingulate cortex and the medial prefrontal cortex (Fox et al., 2015). In prototype studies where individuals are asked what gratitude is, gratitude has also consistently been associated with having a positive 'feeling' (Lambert et al., 2009; Morgan et al., 2014). Moreover, gratitude's evolutionary significance is underscored by its role in fostering cooperation and strengthening social ties, ensuring the formation of beneficial alliances (Algoe, 2012; Algoe et al., 2020). The particular triggers, physiological underpinnings, and adaptive benefits of gratitude set it apart from other emotions, supporting its categorization as a distinct emotion, in line with previous research (Fredrickson, 2013). This, therefore, suggests that gratitude is a distinct emotion.

From the dimensional emotion perspective, emotions are better understood as points in a continuous space or plane, often defined by axes like valence (from pleasant to unpleasant) and arousal (from activated to deactivated) (Barrett, 2006; Condon et al., 2014; Posner et al., 2005). In this framework, gratitude can be situated as a positive-valence (Gulliford & Morgan, 2018), moderately active emotion (Kraiss et al., 2023), characterized by feelings of warmth and appreciation (Lambert et al., 2009), placing it firmly on the positive side of the valence axis. At the same time, the motivational aspect of gratitude, which often leads to reciprocation, suggests a level

of activation or action tendency (Fredrickson, 2004a). Although gratitude may share this dimensional space with other positive emotions, such as joy or relief, its unique triggers (e.g., recognising a freely given benefit from another (Wood et al., 2010) and subsequent behaviours (like acts of kindness or reciprocity) distinguish it within the dimensional model (Algoe et al., 2008). Thus, gratitude maintains its individuality even within a dimensional understanding of emotions.

Another proposal for what makes gratitude particularly distinct comes from the Find, Remind, and Bind theory (Algoe, 2012). This is a functional theory of emotion which describes gratitude as an emotional response to a benefit received from a benefactor (Algoe, 2012). In a nutshell, this theory posits that gratitude serves the evolutionary functioning of finding a high-quality partner and strengthening the relationship with them (Algoe, 2012; Algoe et al., 2020). Thus, gratitude "finds new or reminds of a known good relationship partner, and helps to bind recipient and benefactor closer together" (Algoe, 2012, p. 457). Gratitude does so by biasing cognitions around benefactors positively (Algoe & Haidt, 2009), and motivating individuals to engage in behaviours to promote interpersonal bonds (Algoe, 2012, p. 457), such as spending more time with their partner (Algoe & Haidt, 2009). Similarly, gratitude expressions typically signal to the benefactor that the recipient is responsive to them (Algoe & Zhaoyang, 2016). Altogether, this suggests that gratitude should lead to higher social support and higher relationship quality (Algoe, 2012; Wood et al., 2010). This is consistent with studies showing that gratitude expressions are associated with greater relationship commitment (Park et al., 2019) and that trait gratitude is associated with increased perceptions of social support (Kong et al., 2015). Thus, this suggests that by criteria of multiple theories of emotion, gratitude can be conceptualized as a distinct emotion.

2.1.2 Is gratitude a positive emotion?

Within this discussion of the exact nature of gratitude, it is prudent to also consider if gratitude

is a positive emotion, as it is typically regarded as so (Roth & Laireiter, 2021). However, the precise meaning of this is somewhat controversial (Gulliford & Morgan, 2018). On one account, what counts as a positive emotion are any emotions with a positive valence or "phenomenology" (Cohen, 2006; Fredrickson, 2013; Navarro & Tudge, 2020), meaning it "feels good" to experience them. However, according to Fredrickson & Cohn (2008), what distinguishes a positive emotion from positive affect and other positive states is that positive emotions have specific appraisal antecedents – judgments people make about their environment (Fredrickson & Cohn, 2008). Thus, a positive emotion is defined as a pleasant affective state that arises in response to stimuli or events that are appraised as beneficial or fulfilling (Fredrickson & Levenson, 1998; Ryff & Singer, 1998). Positive emotions, such as joy, pride, or gratitude, are differentiated from other positive states by their connection to specific cognitive appraisals (Csikszentmihalyi & Csikszentmihalyi, 2006; Fredrickson & Cohn, 2008). Gratitude, as a positive emotion, emerges from the recognition and appreciation of a beneficial act or circumstance (Tsang et al., 2021).

Positive emotions are also distinguished by their adaptive functions in motivating behaviours that foster growth and well-being (Fredrickson & Cohn, 2008). For example, trait and state gratitude have been shown to be associated with increased well-being (Sansone & Sansone, 2010), increased performance at work (Cortini et al., 2019), facilitated goal contagion (Jia et al., 2014), and an increased likelihood to engage in prosocial, helping behaviours (Bartlett & DeSteno, 2006). This demonstrates an association between gratitude and increased engagement with rewards and opportunities in an individual's environment. Thus, gratitude is a positive emotion on the basis of Fredrickson and Cohn (2008) as it feels good, arises from specific antecedent appraisals, and prompts specific action tendencies.

2.2 Why study gratitude?

Having established gratitude as a distinct positive emotion, both within the frameworks of categorical and dimensional emotion theories, the question naturally arises: Why is it important

to study gratitude in-depth? The following sections suggest a number of reasons to target gratitude in the context of physical and psychological health. It will be suggested that gratitude has beneficial relationships with health and well-being, is amenable to being manipulated or changed, and thus plays an important health-protective role.

2.2.1 Gratitude and health and well-being

Research suggests an important overall link between both state and trait gratitude and health (Boggiss et al., 2020). In a cross-sectional study of 962 participants, Hill et al. (2013) found that trait gratitude positively correlated with physical health (r = .16), psychological health (r = .29), propensity for health activities (r = .32) and willingness to seek help for health concerns (r = .22). Similarly, in a cross-sectional study of 401 participants, trait gratitude was found to predict greater sleep quality and sleep duration (Wood et al., 2009). A cohort study of 164 participants found that trait gratitude was associated with lower levels of endothelial dysfunction (Celano et al., 2017). A recent review of 13 studies concluded that trait and state gratitude have positive associations with cardiovascular health (Cousin et al., 2021), with state gratitude being inversely associated with cardiovascular reactivity (Gallagher et al., 2020). Evidence from studies utilizing gratitude interventions also suggest an important relationship between gratitude and cardiovascular health. A randomised controlled with a sample of 119 women found that individuals in a gratitude intervention group had lowered ambulatory diastolic blood pressure (Jackowska et al., 2016). A longitudinal study of 70 cardiac patients found that individuals who engaged in gratitude journaling interventions had reduced inflammatory biomarker index scores, and increased parasympathetic heart rate variability responses throughout journaling tasks (Redwine et al., 2016). These studies collectively provide compelling evidence for the importance of studying gratitude, particularly in relation to cardiovascular health.

Wood et al. (2010) suggest that, generally, gratitude's usefulness comes from (1) its explanatory power with respect to well-being, and (2) that it is possible to create effective gratitude

interventions for well-being (e.g. Froh et al. (2009)) In a study of 31,206 participants, trait gratitude remained a reliable predictor of well-being across age groups (Chopik et al., 2019). Trait gratitude is a consistent predictor of facets of subjective well-being such as life-satisfaction and meaning (Peterson et al., 2007). These findings are further supported by a comprehensive meta-analysis encompassing 30 experimental studies, which revealed that interventions focused on gratitude significantly enhance subjective well-being in comparison to control groups (Davis et al., 2016). In a context where subjective well-being is a useful predictor of cardiovascular health (Boehm, 2021) due to its capacity to buffer stress (Brummett et al., 2009), the connection between gratitude and well-being becomes particularly valuable.

According to the Find, Remind, and Bind theory of gratitude (Algoe, 2012), trait gratitude serves a unique social function. This is relevant for health as social support is a well-known predictor of health outcomes (Reblin & Uchino, 2008). For example, a meta-analysis of 128 articles found that increased loneliness was associated with worse cardiovascular and mental health outcomes (Courtin & Knapp, 2017), while a more recent meta-analysis of 41 studies found that increased social support was linked to lowered inflammation, a key predictor of cardiovascular disease progression (Uchino et al., 2018). Longitudinal studies suggest that better relationship quality between heart failure patients and their caregivers is associated with lowered mortality likelihood (Hooker et al., 2015). A meta-analytic review of 126 empirical articles found that greater marital quality was related to better health outcomes, and that this effect was mediated by cardiovascular reactivity (Robles et al., 2014). In a cross-sectional study, O'Connell & Killeen-Byrt (2018) found that trait gratitude can enhance physical health by reducing feelings of loneliness and perceived stress, suggesting that psychosocial well-being is a key pathway through which gratitude exerts its beneficial effects. Thus, trait gratitude may promote health through its unique functioning of building and maintaining high quality social relationships (Wood et al., 2010).

Wood et al. (2010) suggest that if trait gratitude is conceptualised broadly as a life orientation that it should be strongly related to well-being. Indeed, gratitude does seem to bias individuals to

view events positively rather than negatively (Watkins, 2014). This view has been echoed by other authors (e.g. Alkozei et al., 2018). However, this claim that gratitude has a strong relationship with well-being is not well-supported in meta-analytical studies (Carr et al., 2023). In a more recent analysis of 38 studies found that gratitude interventions had modest effects on outcomes such as well-being (Dickens, 2017). Similarly, in a meta-analysis of 347 studies examining the effects of positive psychological interventions, gratitude interventions were found to have relatively modest effects on well-being (Hedges's g = .30) (Carr et al., 2021). However, in the context of stress, meta-analytic studies suggest that gratitude (assessed as both a state and trait) has a somewhat unique relationship with stress, with Carr et al. (2021) finding that gratitude interventions had a modest effect on stress reduction.

Gratitude is amenable to change

An important and useful feature of gratitude is that it is there are interventions which can modulate state gratitude (Komase et al., 2021). These gratitude interventions are activities which are de- signed to cultivate experiences of appreciation and thankfulness (Boggiss et al., 2020). Gratitude interventions aim to shift individuals' focus from negative aspects to positive aspects of their lives, which foster a sense of gratitude and well-being (Cregg & Cheavens, 2021; Kirca et al., 2023). Examples include gratitude journaling, gratitude letters, gratitude lists, and mindfulness-based practices (Davis et al., 2016). These have the advantages of being low-cost, simple to administer, and are generally well-received by participants (Boggiss et al., 2020; Wood et al., 2010).

Several studies suggest that gratitude interventions increase state gratitude (Davis et al., 2016). For example, a randomised controlled found that gratitude lists had a significant effect on state gratitude (Otsuka, 2012). Similarly, an intervention study of elementary school children found that writing gratitude lists had a significant effect on state gratitude (Froh et al., 2014). A recent meta-analysis of 38 studies found that gratitude interventions had modest effects on both state and trait gratitude (Dickens, 2017). Previous studies have also shown that trait gratitude can

be successfully manipulated through gratitude interventions (Cregg & Cheavens, 2021; Davis et al., 2016; Kirca et al., 2023). This is also supported by a meta-analytic study of 26 studies (Davis et al., 2016). However, as traits are typically quite stable (Bleidorn et al., 2021), it is likely that the measurement of trait gratitude in these studies may not be sensitive enough to the state and trait conceptualizations of gratitude (Toepfer et al., 2012).

Randomised controlled trial studies of gratitude have found effects for a variety of outcomes such as well-being and depression Kirca et al. (2023). They typically involve asking participants to write gratitude lists or other gratitude activities (Komase et al., 2021). Meta-analytic evidence is very promising with studies showing that increasing trait gratitude is associated with reductions in perceived stress and depressive symptoms (Cheng et al., 2015; Fekete & Deichert, 2022). However, such randomised controlled trial designs have not been utilised to assess the impact of gratitude on cardiovascular stress responses, a lacuna this thesis addresses.

In sum, these studies suggest that gratitude is manipulable. Gratitude interventions, such as gratitude journaling and writing gratitude lists, are effective in enhancing both state and trait gratitude.

Overall

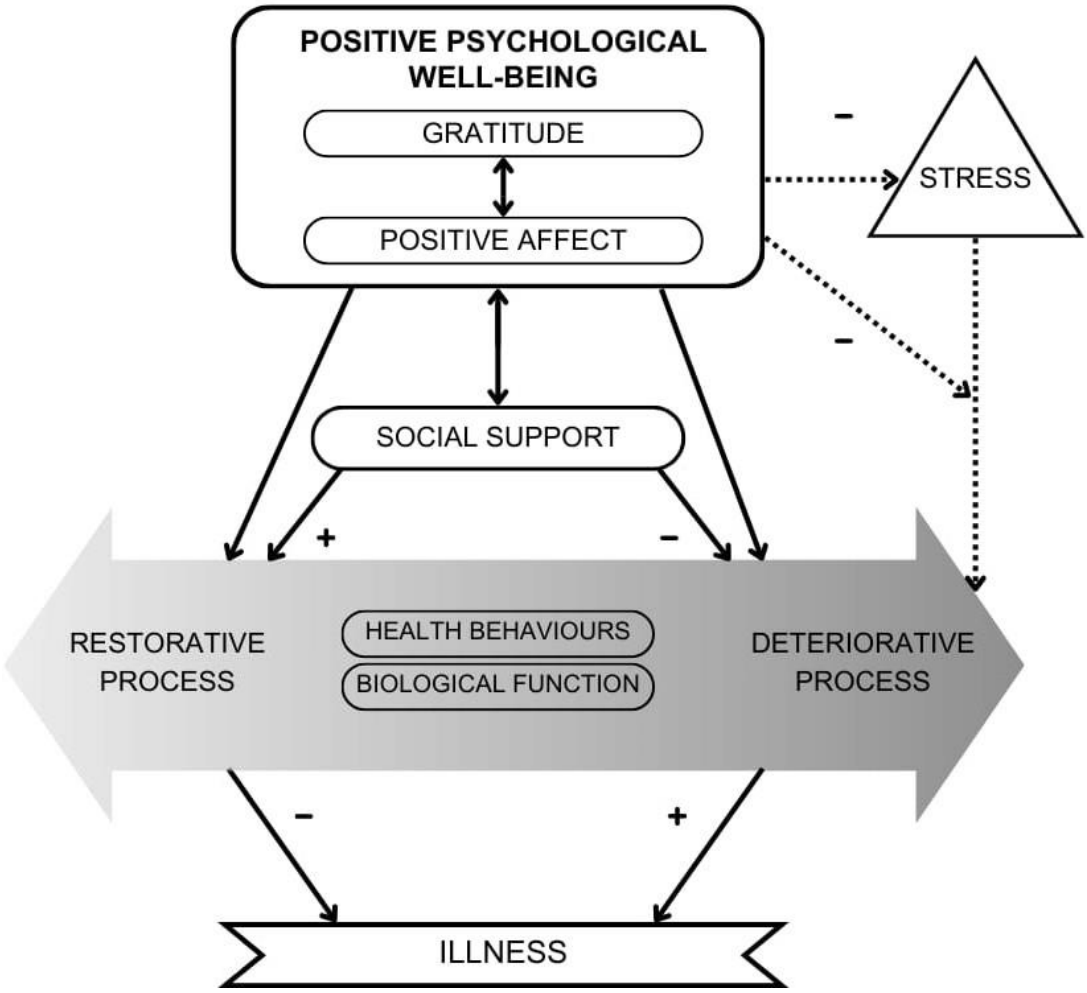
The empirical investigation of gratitude reveals its substantial impact on social, psychological, and physiological domains, reaffirming its value as a subject of scientific study. The evidence suggests that gratitude is a useful construct that can influence well-being and health outcomes in a multitude of different ways, and can also be manipulated. As we shall see below, there is also evidence that gratitude buffers the deleterious impacts of stress.

2.3 Does gratitude buffer stress?

The Transdisciplinary Model of Stress (Epel et al., 2018) suggests that certain factors like social support (e.g. Wang et al., 2014) may play a protective or buffering role with respect to the deleterious impacts of stress. This is consistent with the Model of Positive Psychological Well-Being (Boehm & Kubzansky, 2012). The model predicts that facets of psychological well-being, like optimism, are prospectively associated with better cardiovascular health metrics and outcomes (Kubzansky et al., 2018). This relationship is mediated through both enhancing and diminishing processes affecting health behaviours, such as dietary choices and exercise, as well as biological functions, including inflammation and heart rate regulation (Boehm & Kubzansky, 2012; Kubzansky et al., 2018). The idea is that positive psychological well-being leads to healthier lifestyle choices and improved biological functioning, which collectively reduce the risk of cardiovascular diseases. Recent updates to this model emphasise that stress buffering is an important pathway by which positive emotions promote cardiovascular health (Boehm, 2021). This suggests that positive emotions both modulate cardiovascular reactivity and cardiovascular recovery (Pressman et al., 2019; Schache et al., 2019).

The Model of Positive Psychological Well-Being has been adapted (see Figure 2.1) by Schache et al. (2019) to specifically detail pathways by which gratitude impacts health. In particular, this model suggests that gratitude might buffer the effects of stress, reducing the deteriorative biological processes associated with it (Schache et al., 2019). Extant evidence indicates that gratitude plays a stress-buffering function (Wang & Song, 2023), with studies indicating that trait and state gratitude significantly influences stress evaluation (Meyer & Stutts, 2023; Wood et al., 2007; Wood et al., 2010) and related cardiovascular effects such as endothelial dysfunction (Cousin et al., 2021). Evidence for these effects for gratitude can be considered in gratitude's trait, state and intervention forms.

Figure 2.1: Pathway between gratitude and illness in the model of psychological well-being (adapted from Schache et al., 2019, p. 6)



2.3.1 Trait gratitude and stress buffering

Trait gratitude aids in viewing stressful situations as challenges instead of threats (Enko et al., 2021) and in recognizing more coping resources (Wood et al., 2010). For example, trait gratitude has been found to moderate the relationship between daily hassles and life satisfaction (Tachon et al., 2021). Empirical evidence suggests that trait gratitude also seems to buffer against the effects of adverse life events (Duprey et al., 2018). For example, a study of 854 Chinese adolescents found that trait gratitude buffered the relationship between stressful life events and

non-suicidal self-injury (Wei et al., 2022). Trait gratitude was also found to be a protective factor for Israeli adolescents who witnessed missile-attacks, with gratitude being found to reduce post-traumatic stress disorder symptoms (Israel-Cohen et al., 2015). Meta-analytic evidence also suggests that trait gratitude has an inverse, medium-sized effect on PTSD symptoms (Richardson & Gallagher, 2020).

2.3.2 State gratitude and stress buffering

A recent review of 19 studies involving 2951 participants suggests that state gratitude has the potential to improve biomarkers of cardiovascular disease, with the regulation of inflammation markers and the hypothalamic–pituitary–adrenal axis being key mechanisms underpinning this relationship (Wang & Song, 2023). State gratitude's association with acute stress responses has been demonstrated in laboratory contexts. S. Gallagher et al. (2020) found that state gratitude had an inverse relationship with systolic blood pressure reactivity in a sample of 86 participants. Similarly, Ginty et al. (2020) found that state gratitude was inversely associated with cardiovascular reactivity across a number of parameters. Thus, state gratitude has been associated with a reduction in inflammatory biomarkers (Moieni et al., 2019; Redwine et al., 2016) which are typically associated with higher allostatic load (Rodriquez et al., 2019). Taken together, this suggests that gratitude is likely playing a stress-buffering and leading to healthier response profiles.

2.3.3 Gratitude interventions and stress buffering

A substantial body of evidence supports the potential for gratitude interventions on trait gratitude to decrease negative feelings and thus buffer against their negative health outcomes (O'Connell & Killeen-Byrt, 2018; Wolfe, 2022; Y. Yang et al., 2018). Gratitude interventions increasing trait gratitude have also been associated with reductions in amygdala reactivity in a

randomised con- trolled (Hazlett et al., 2021), and reductions in cortisol levels in pregnant women (Matvienko-Sikar & Dockray, 2017). Similarly, a systematic review of 19 gratitude interventions studies concluded that gratitude may have a positive effect on biomarkers of cardiovascuvlar disease risk, with stress reduction being a noted critical pathway (Wang & Song, 2023). Thus, evidence suggests that state gratitude, trait gratitude, and gratitude interventions play important stress buffering roles, as predicted by Schache et al. (2019) and Boehm (2021).

2.3.4 Gratitude and affect

The Positive Activity Model (Lyubomirsky & Layous, 2013) suggests that there are important conditions underpinning the success of positive psychological interventions. For example, intervention-related features such as dosage and variety are important components of a positive psychological intervention which determine effectiveness (Parks et al., 2012). Similarly, there are people-related features such as motivation and baseline affective state (Lyubomirsky & Layous, 2013). Indeed, there are suggestions that individuals low in positive affect benefit most from gratitude interventions (Alkozei et al., 2018). This is informed by the resistance hypothesis, which suggests that individuals already predisposed to see the world positively do not benefit from additional positive experience (Rash et al., 2011). Prior research has found evidence for this in a gratitude intervention context on positive affect (Froh et al., 2009), the interaction between the balance of positive to negative emotions and gratitude in the context of cardiovascular stress buffering has not been examined.

2.3.5 Mechanisms

In sum, studies suggest trait and state gratitude provide a buffer against negative health outcomes. Additionally, gratitude is associated with reductions in biomarkers related to stress and inflammation, suggesting a possible mechanism for its beneficial health effects. A number of

mechanisms have been proposed. The Model of Positive Psychological Well-Being (Boehm & Kubansky, 2012) suggests that gratitude, along with other positive emotions, may improve health outcomes by moderating the effects of stress. This can be through increasing positive coping styles, through re- framing negative events positively, or through increasing access to resources such as creativity and social support (Alkozei et al., 2018; Kubzansky et al., 2018; Schache et al., 2019). Trait and state gratitude have been suggested to inculcate positive coping strategies (Wood et al., 2010), and state gratitude has been found to bias cognition to see events more positively and less negatively (Alkozei et al., 2018), facilitating positive re-framing. Through these mechanisms, gratitude may reduce the frequency and intensity of daily stressors (Tachon et al., 2021). These stress buffering effects associated with gratitude may have the useful effects of reducing inflammation and sympatho adrenomedullary system activity (Wang & Song, 2023). Both inflammation and sympatho adrenomedullary system activity have been linked to cardiovascular disease (Levine, 2022). Gratitude may reduce inflammation by favouring decreased amygdala activity (Hazlett et al., 2021), a key component in triggering inflammation (Irwin & Cole, 2011). Similarly, gratitude has been associated with increased MU-Opioid signalling (Henning et al., 2017). MU-opioid receptors have been thought to be involved in stress-relief and restoration (Nakamoto & Tokuyama, 2023). Thus, practicing gratitude can alleviate strain on the body and positively influence cardiovascular health (Henning et al., 2017). Wang et al., (2023) provide a review of the potential underlying neurological mechanisms determining how gratitude might result in physiological changes that are cardio-protective.

2.4 Conclusion

Based on the comprehensive exploration of gratitude throughout the chapter, it can be concluded that gratitude plays a significant and multifaceted role in enhancing cardiovascular health. The evidence presented underscores gratitude's utility as both a state and trait, contributing

to positive cardiovascular health outcomes through buffering against the deleterious effects stress. Its distinctiveness as a positive emotion, alongside its amenability to interventions, suggests that fostering gratitude can be a valuable component of psychological and physical health strategies. Altogether this suggests that gratitude is a valuable candidate for further exploration of its stress buffering potential.

Chapter 3

Aims, overview and summary of empirical studies

3.1 Aims

In accordance with theoretical frameworks and empirical evidence we would thus expect gratitude to act as a psychosocial buffer of the negative impacts of psychological stress on cardiovascular health. Epel et al. (2018) provide a coherent framework for understanding how stress responses can, over time, accumulate to adversely impact cardiovascular health. Similarly, the Model of Positive Psychological Well-Being (Schache et al., 2019) suggests that within this framework, gratitude may act as a critical psychosocial buffer. Prior work by Gallagher et al. (2020) and Ginty et al. (2020) provide empirical validation for these models. Together, these suggest that gratitude is a likely buffer of cardiovascular stress.

However, critical gaps in our understanding of how gratitude – as a trait, state or through intentional practices – impacts overall health and well-being highlight the imperative for targeted scientific inquiry in this domain. It has not been demonstrated whether gratitude's relationship to stress reactivity leads to a lowered risk of worsened cardiovascular health outcomes. While

gratitude has been associated with lowered reactivity (Gallagher et al., 2020) and better biomarkers of cardiovascular health like lower prognostic inflammation (Cousin et al., 2021), studies have yet to link it specifically to cardiovascular events, like acute myocardial infarction, through its impact on reactivity. Evidence connecting trait gratitude to adverse cardiovascular outcomes – like risk of acute myocardial infarctions – would be useful in validating the model of positive psychological well-being (Schache et al., 2019), which suggests a long-term relationship between gratitude and cardiovascular health outcomes through the stress-buffering function of gratitude.

Furthermore, no research has examined the association between state gratitude and both the cardiovascular reaction to stress and recovery from stress (Gallagher et al., 2020). Both exaggerated reactions to stress and delayed recovery lead to worsened cardiovascular outcomes (Panaite et al., 2015; Whittaker et al., 2021). As a moderator of the effects of stress, it would be expected that gratitude both reduce the reaction to stress (Brummett et al., 2009) and hasten recovery (Papousek et al., 2010). This is important as it further validates prior theoretical models such as the Model of Positive Well-Being (Boehm & Kubzansky, 2012), while also providing critical evidence around the importance of positive psychology in promoting cardiovascular health (Labarthe et al., 2016).

The relationship between affect and gratitude in this context has not been explored. Understanding the conditions under which gratitude may impact the cardiovascular response to stress is important in developing effective gratitude interventions (O'Connell et al., 2017). In this context, positive affect might act as a moderator for the association between gratitude and well-being (Rash et al., 2011). Gratitude may buffer the impact of stress by increasing positive affect (Wood et al., 2010) which dampens the effects of negative arousal (Dignath et al., 2020; Steenbergen et al., 2015). However, an alternate position is that individuals already experiencing higher levels of positive affect may not benefit as strongly from gratitude (Klibert et al., 2019). This is informed by the resistance hypothesis (McCullough et al., 2004), which suggests that individuals who already experience a high amount of positive emotions are unlikely to be affected by additional positive experiences such as experiencing gratefulness (Rash et al., 2011). As such, gratitude may inter-

act with the balance of positive to negative affect in how it buffers stress, with individuals who experience more negative than positive affect benefiting more from gratitude (Froh et al., 2009). Understanding this is important in understanding who may potentially benefit most from a gratitude intervention (O'Connell et al., 2017), while also further validating the positive activity model (Lyubomirsky & Layous, 2013).

Finally, there has yet to be a randomised-controlled trial study exploring how gratitude interventions can impact cardiovascular reactivity. Although there has been previous cross-sectional research on the subject, these studies are limited in their capacity to demonstrate causal relation- ships between gratitude and cardiovascular reactivity (e.g. Ginty et al., 2020). In this context, a lab-based experimental design which integrates the principles of randomised control trials will offer a robust framework in which to make causal inferences with minimal confounding and bias (Spieth et al., 2016). Importantly, without a randomised controlled trial, it could be argued that the association between state gratitude and cardiovascular reactivity might be a case of reverse causality, where reactivity causes state gratitude (Antonakis et al., 2010; Sattar & Preiss, 2017). Thus, a randomised controlled trial is crucial to provide more robust insights into the association between state gratitude and cardiovascular reactivity, constituting good scientific practice.

This research has the following aims:

1. Assess the impact of trait gratitude on the risk of suffering acute myocardial infarction indirectly through cardiovascular reactivity.
2. Assess the impact of state gratitude on cardiovascular reactivity and recovery.
3. Assess the interaction between affect balance and state gratitude on cardiovascular reactivity and recovery.
4. Conduct a randomised-control trial study to examine the effectiveness of gratitude interventions in modulating cardiovascular reactivity and recovery in response to stress.

Thus, this thesis will offer key insights into (1) the long-term relationship between gratitude, cardiovascular reactivity and cardiovascular health, (2) the relationship between state gratitude and reactivity and recovery, (3) the interaction between gratitude and the balance of positive to

negative emotions in buffering the cardiovascular effects of stress, and (4) experimentally manipulating state gratitude and then exposing participants to acute stressors in a randomised-control trial design.

3.2 Overview and summary of empirical studies

Study one examines the long-term, indirect relationship between trait gratitude and the risk of acute myocardial infarction through cardiovascular reactivity using a within-subjects, correlational design. As previously suggested by Epel et al., (2018) and Whittaker et al. (2021) stress reactivity may impact cardiovascular health through cumulative strain in response to acute stressors. If gratitude acts as a buffer against these long-term stressors, we should expect to see improved cardiovascular outcomes through the association with cardiovascular reactivity. However, Gallagher et al. (2021) found that trait gratitude was associated with increases in cardiovascular reactivity (i.e. systolic and diastolic blood pressure). This finding implies that over time trait gratitude may be associated with negative cardiovascular out- comes through exaggerated reactivity (Whittaker et al., 2021). Study one explores this by making use of secondary data analysis of the Mid-Life in the United States data set over 6.7 years (Feingold et al., 2019). Study one thus accomplished two novel things: (1) it demonstrated that trait gratitude is indirectly associated with a lowered risk of suffering an adverse cardiovascular outcome through its relationship reactivity, and (2) this indirect reduction in risk occurred despite increases in reactivity. This is novel because it challenges conventional understanding of how trait gratitude influences long-term health outcomes. Previous research as suggested that increased cardiovascular reactivity to stress is considered a risk factor for acute myocardial infarction due to the strain associated with cardiovascular reactivity on the cardiovascular system (Carroll et al., 2012). However, these findings suggest that the nature of this relationship is more complex, with trait gratitude offering a protective buffer that mitigates the long-term adverse effects of heightened cardiovascular reactivity. Indeed, this supports more novel evidence demonstrating that both exaggerated and

blunted reactivity lead to adverse cardiovascular outcomes (Whittaker et al., 2021).

Study Two is a within-subjects correlational study. While Study One focuses on the long-term, indirect relationship between trait gratitude and cardiovascular health through cardiovascular reactivity, Study Two extends the examination of gratitude from a trait perspective to a state perspective in a laboratory context. Study Two examined the impact of state gratitude on both cardiovascular reactivity and recovery. To do so, it made use of multi-level growth-curve analysis in order to ex- amine the non-linear trajectory of the stress-response (Curran et al., 2010). Study two also aimed to assess whether there was an interaction between state gratitude and the balance of positive to negative emotions (Dignath et al., 2020; Steenbergen et al., 2015). This study resulted in two novel findings: (1) state gratitude resulted in both lowered reactivity and hastened recovery for systolic blood pressure, demonstrating that state gratitude was improving the overall cardiovascular stress response profile, and (2) that the balance of positive to negative emotions interacted significantly with state gratitude whereby more positive emotion amplified to effects of state gratitude on the diastolic blood pressure stress response.

Where Study One analysed the long-term, indirect role of trait gratitude in mitigating cardio- vascular risks, Study Two complements this by demonstrating the acute, beneficial effects of state gratitude on cardiovascular reactivity and recovery, offering insight into the immediate physiological mechanisms at play. The results of Study Two also have implications for the positive activity model (Lyubomirsky & Layous, 2013). Where this model (Lyubomirsky & Layous, 2013) and prior research suggests that those who experience higher levels of positive affect will benefit less from gratitude (Froh et al., 2009), the results of Study Two suggest an amplification effect whereby a greater balance of positive to negative emotions resulted in greater stress buffering associations with state gratitude. Together, Study One and Study Two form a mutually beneficial understanding of gratitude's influence on cardiovascular health, spanning from transient states to enduring traits, and underscores the potential for integrating gratitude into health interventions.

Study Three utilised a randomised controlled trial design to explore how a brief gratitude intervention impacts cardiovascular reactivity. Study Three builds on the insights provided by

Studies One and Two by utilizing a rigorous experimental approach to directly test the causal relation- ship between state gratitude and cardiovascular reactivity. This progression from observational and within-subjects methodologies to a randomised controlled trial design marks a critical step in understanding the dynamics of gratitude and its effects on physical health. While Studies One and Two established associations and immediate effects of gratitude on cardiovascular health, Study three strengthens the evidence base by demonstrating a causal link between state gratitude and reduced cardiovascular reactivity through an RCT, the gold standard for causal inference in clinical research (Hariton & Locascio, 2018). In the absence of experimental manipulation, it is possible that reactivity is an increasing state gratitude and not the other way around (Sattar & Preiss, 2017). Unlike the secondary data analysis and within-subjects design of the earlier studies, Study Three's experimental manipulation of gratitude allows for a clearer determination of how inducing feelings of gratitude can directly impact physiological stress responses. The major novel finding of this study confirms that state gratitude is indeed reducing reactivity. The value lies in the clear demonstration of a causal relationship, providing a strong foundation for designing gratitude- based interventions aimed at cardiovascular health promotion and stress reduction (Labarthe et al., 2016). Furthermore, to minimise publication bias and selective reporting, this trial was registered on clinicaltrials.gov (Identifier: NCT05133063).

Employing multiple methodological frameworks strengthens the foundation and breadth of re- search findings in this thesis. Observational studies serve as the foundation, offering initial insights and justifications for more nuanced experimental inquiries (Boyko, 2013). Within-subjects experimental designs permit the examination of effects within the same individuals over time (Easterby-Smith et al., 2021), thus controlling for inter-individual variability and allowing associations to be demonstrated between state gratitude and both cardiovascular reactivity and recovery. Similarly, within-subjects experiments also offer a practical advantage of offering a boost to statistical power (Charness et al., 2012). Randomised controlled trials, regarded as the gold standard for causal research (Hariton & Locascio, 2018), extend this foundation by testing hypotheses under rigorously controlled conditions, thereby minimizing biases and augmenting the

generalizability and reliability of research outcomes. This combination of methods not only works harmoniously, each building on the results of the previous, but also permit this research to address its key aims effectively and efficiently.

Taken together, these studies suggest that both state and trait gratitude have important stress-buffering functions. By examining both trait gratitude (an enduring aspect of personality) and state gratitude (a temporary emotional state), along with the effects of a gratitude intervention, the research encompasses the broad spectrum of how gratitude can be experienced and cultivated (Wood et al., 2010). This comprehensive approach ensures a deeper understanding of gratitude's impacts on health. Similarly, employing observational, within-subjects experimental, and randomised controlled trial designs allow this thesis to address different research questions, from exploring the long-term associations between trait gratitude and cardiovascular health to exploring mechanisms and testing causality. Thus, in sum, the results of the three studies presented in this thesis provide the following novel evidence:

1. Evidence that there is an indirect, longitudinal relationship between trait gratitude and risk of acute myocardial infarction through heart rate reactivity over 6.7 years.
2. Evidence that state gratitude is associated with lowered reactivity and hastened recovery, and this relationship is moderated by a higher ratio of positive to negative emotions.
3. Evidence demonstrating the effectiveness of a brief gratitude intervention at reducing cardiovascular reactivity.

In summary, these studies shed light on how gratitude, both as a lasting trait and a state, plays a significant role in cardiovascular health. This research highlights the powerful ways gratitude can influence our cardiovascular health and offers promising pathways for improving heart health through gratitude practices.

Chapter 4

Epistemology and methodological approach

4.1 Introduction

Chang (2022) notes that much of what is valued in science is knowing *how* to do something. This is put well by philosopher Gilbert Ryle (1946, p. 15) who writes: “The advance of knowledge does not consist only in the accumulation of discovered truths, but also and chiefly in the cumulative mastery of methods”. In this vein, Chang (2022) proposes that knowledge is built out of the skilful and harmonious fitting together of activities to achieve research goals. Cortina (2020) suggests that it is good methodological practice for a researcher to state the ontological and epistemological assumptions of their research. Ontology is about the nature of reality and epistemology concerns the nature of knowledge (Easterby-Smith et al., 2021). As such, in the sections below, I will outline the realist and positivist frameworks within which this thesis operates.

4.2 Scientific realism and its alternates

This research adopted a scientific realist account of ontology and epistemology. This means that ontologically, scientific theories or observations regarding the social world accurately correspond to existing, mind-independent phenomena (Devitt, 2008), and epistemologically that the social world can be measured using objective methods (Easterby-Smith et al., 2021). In research methods parlance, this research adopts a realist ontology and positivist epistemology (Easterby-Smith et al., 2021).

According to a scientific realist account, measuring a physical or mental attribute, entails understanding that attribute to be a true reality, independent from the mind of the observer (Guyon et al., 2018; Zachar, 2010). Therefore, in the case of this research, heart rate, blood pressure, stress, and gratitude are all regarded as really existing entities that are available for us to study, and can be done so quantitatively. This is commensurate with the quantitative imperative, which suggests that to study something scientifically means to measure it numerically (Michell, 2003).

Ontological alternatives to this view include positions such as relativism or nominalism (Easterby-Smith et al., 2021). These are views that deny that there is an objective reality with which scientific theory corresponds (e.g. Rorty, 1998). Epistemological alternatives include social constructivism (Easterby-Smith et al., 2021). This is the views that science is a social praxis and thus engages in a process of socially constructing scientific facts (Zyphur & Pierides, 2020). Methodologically, these views typically correspond with qualitative methods (Easterby-Smith et al., 2021).

A prominent philosophical argument in favour of scientific realism over other views is the No Miracles argument (Bonilla, 2019; Putnam, 1983). The essence of the No Miracles argument is that the success of science would be a "miracle" if scientific theories were not at least approximately true descriptions of the world (Devitt, 2008). Thus, scientific theories are not simply useful tools, but are actually accurate descriptions of an external, independent world.

In contrast, the Meta-Induction or Pessimistic Induction Argument is often used in response

to the No Miracles Argument (Devitt, 2008). This suggests that successful scientific theories of the past have been found to be false, and as such we have no reason to believe our current theories will not be demonstrated to be false in the future (Park, 2011). This undermines the no miracle argument's premise that scientific theories are accurately describing reality (Park, 2011).

According to Mizrahi (2013), the pessimistic induction argument can be criticised for the following reasons: Firstly, it incorrectly assumes that the difference in content between past and current theories implies a difference in truth value (Mizrahi, 2013). Secondly, it relies on an un- representative sample of past theories, selectively focusing on those that were eventually falsified (Mizrahi, 2013). Thirdly, Lastly, it overlooks the fact that successful theories, even if later proven false, contribute valuable insights and advancements, which are foundational for subsequent theories (Mizrahi, 2013).

In this context, the No Miracles argument is a persuasive argument for scientific realism. To repeat, this argument suggests that the success of science would be a "miracle" if scientific theories were not at least approximately true descriptions of the world. It asks that if we assume that scientific theories do not accurately refer to existing entities, what explains the success of scientific theories? Van Frassen (1980) might suggest that scientific success is not about truth but only about prediction and control (or empirical adequacy), but as Borsboom (2005) notes, this leaves us completely in the dark about why our predictive machinery works. Given that there are persuasive reasons not to reject the claim that past scientific theories have been successful, I think it is a suitable position for this research.

4.3 Methodological consequences of scientific realism

The methodological consequences of the scientific realism approach for this thesis are that – due to the positivist epistemology – it emphasises quantitative measurement and experimental manipulation (Easterby-Smith et al., 2021). It relies heavily on objectivity, attempting to reduce the impact of subjective experience through its use of objective measures, standardised research

protocols, and statistical inference (Park et al., 2020). Its goal, then, is to isolate and control all the key influential factors involved in this research so that in the case of this research only the key variables of blood pressure, stress, and gratitude are studied (Park et al., 2020).

In adopting a realist ontology and positivist epistemology, this research limits its scope to observable and measurable phenomena, potentially overlooking the subjective and interpretative aspects of human experience (Easterby-Smith et al., 2021). While this approach ensures rigorous, quantifiable data, it may not fully capture the complex, nuanced nature of individual perceptions and experiences related to gratitude and stress (Easterby-Smith et al., 2021). Furthermore, the reliance on surveys and standardized experimental protocols could limit understanding of these phenomena in naturalistic settings, as laboratory conditions may not fully replicate real-world environments (Zanstra & Johnston, 2011). The methodological choices, while robust, suggest a need for cautious interpretation of the findings within these constraints.

The employment of quantitative methods, underpins the thesis's scientific objectivity. Quantitative methods offer a useful advantage of objectivity under a positivist paradigm (Easterby-Smith et al., 2021). In this context, a similar advantage is the quantification of the mind-body relationship. By taking the intangible mental properties as localised in the human body, natural science can bring the "mental" side inherited from Descarte's substance dualism (Thibaut, 2018) into the realm of real-spatial causality (Hemmen, 2021). Thus, we can also bring psychological properties such as emotions into contact with the domain of calculability, prediction and control (Floris-Cohen, 2016). This aligns very well with Karl Popper's epistemic principle of falsifiability. Karl Popper (1963) famously argued that scientific theories should be testable and refutable, arguing that for a theory to be considered scientific, it must be susceptible to being proven false. For instance, after observing 1,000 white swans and deducing that all swans are white, the presence of a single black swan would invalidate this theory. This is called falsification. Wilkinson (2013) suggests that this Popperian perspective underpins Null Hypothesis Significance Testing.

In statistical hypothesis testing, a hypothesis (H) is an assertion about the distribution of a particular variable, encompassing both its mean value (μ) and variance (Masson, 2011). There are

two primary hypotheses: the null hypothesis (H_0) and the alternative hypothesis (H_1). Both H_0 and H_1 generally revolve around potential values of μ (Szucs & Ioannidis, 2017). Specifically, the mean for H_0 is represented as μ_0, which is a predefined value, often based on prior knowledge and often set to 0. For instance, in testing the impact of a variable on treatment responses, μ_0 might represent the typical response to that treatment. In essence, the Null Hypothesis suggests no deviation or change. This stands in contrast to the alternative hypothesis, which indicates a deviation, sometimes even specifying the direction of this change, from the Null Hypothesis (Anderson et al., 2000).

Null hypothesis significance testing provides a method to evaluate both the null and the alternative hypotheses. The process assesses the likelihood (P) of observing the given data under the presumption that H_0 holds true, often expressed as P(Data|H_0) (Branch, 2014); J. Cohen (1994)]. In this context, a p-value represents the chance of encountering the observed data if H_0 were accurate. A smaller p-value suggests that the observed data is less likely under H_0. Commonly, a p-value less than .05 is deemed statistically significant, leading to the rejection, or falsification, of H_0 and acceptance of H_1 (Szucs & Ioannidis, 2017). Confidence intervals provide a convenient method of summarizing the results of hypothesis testing for many effects (Greenland et al., 2016). Null hypothesis significance testing aligns with the Popperian falsification framework, granting researchers a deductive lens through which to interpret their findings (Wilkinson, 2013). As such, this is the approach adopted by this thesis.

However, there are significant criticisms of null hypothesis significance testing (Szucs & Ioannidis, 2017). Null hypothesis significance testing is inversely affected by sample size, where larger samples may detect trivial effects as statistically significant, undermining the practical relevance of findings (Cohen, 1994). Moreover, its sensitivity to sample size imposes a dichotomy of significance that may not accurately reflect the underlying effect magnitude or its uncertainty, promoting misleading conclusions (Ioannidis, 2005). Despite these criticisms, null hypothesis significance testing remains the predominant approach (Szucs & Ioannidis, 2017). To address this, this research will report effect sizes and confidence intervals to avoid the sample size related

limitations of this approach (Thompson, 2002).

In sum, this research adopts a scientific realist ontology and epistemology. This means that the social world exists independently of the observer and can be studied using objective measurements. The consequences for this are the emphasis of this study on objective, measures, standardised experimental protocols, and statistical modelling. The aim is to reduce subjective bias, and produce objective research that accurately describes reality.

4.4 Methodological approaches used in prior research on gratitude and cardiovascular health

In the context of research on gratitude and cardiovascular reactivity, there have been three prior studies examining the relationship between gratitude and reactivity (Gallagher et al., 2020; Ginty et al., 2020). All three studies have made use of primary data. As well, all three studies have been based in a laboratory and made use of an experimental design. While the present set of studies makes use of similar methods, it also expands and innovates on previous work methodologically by making use of a secondary data source in order to connect gratitude's association with reactivity to cardiovascular outcomes. Similarly, while prior research has made use of experimental methods, no long-term study has been carried out and no randomised controlled trial has yet been conducted. These studies expand on this by leveraging secondary the Mid Life in the United States dataset to examine the long term associations between trait gratitude and acute myocardial infarction through reactivity in Study One and by incorporating a randomised controlled trial design in Study Three.

4.5 The present set of research studies

This research used three related but distinct methodological approaches across the three studies in order to address its various aims:

1. Assess the impact of trait gratitude on the risk of suffering acute myocardial infarction indirectly through cardiovascular reactivity.
2. Assess the impact of state gratitude on cardiovascular reactivity and recovery.
3. Assess the interaction between affect balance and state gratitude on cardiovascular reactivity and recovery.
4. Conduct a randomised-control trial study to examine the effectiveness of gratitude interventions in modulating cardiovascular reactivity and recovery in response to stress.

4.5.1 Secondary data

The first aim this research is to assess the long-term, indirect relationship between trait gratitude and the risk of acute myocardial infarction through cardiovascular reactivity. While prior research has made use of primary data (Gallagher et al., 2020, 2021; Ginty et al., 2020), these studies have been interested only in the relationship between gratitude and reactivity in the laboratory. As such, they capture short-term cardiovascular responses but do not examine longer term effects. To do so requires substantial resources to follow participants over longer time spans. Fortunately, there are secondary data sets available that can be accessed and analysed. Study One in this thesis aimed to examine the relationship between gratitude, reactivity and the risk of acute myocardial infarction longitudinally. To do so, it makes use of secondary data: Mid Life in the United States (Radler, 2014).

Launched in 1995, the Midlife in the United States dataset is a longitudinal project examining the physical, emotional, cognitive, and social factors influencing health during midlife (Radler, 2014). Utilizing a national sample, Midlife in the United States integrates various components like psychological assessments, biomarker data, and cognitive functioning to explore the complex

interplay of factors affecting health (Radler, 2014). Usefully, it contains both measures of gratitude, taken psychometrically, as well as laboratory assessments of cardiovascular reactivity and subsequent measures of whether a participant had suffered from a heart attack (Dienberg-Love et al., 2010; Radler & Ryff, 2010).

While cardiovascular reactivity can be reliably elicited and measured in laboratory settings (Kamarck & Lovallo, 2003; Whittaker et al., 2021), questions arise when translating these findings to natural, everyday environments. For instance, in a controlled setting, the type and intensity of stressors can be very different from what individuals encounter in their daily lives (Gerin, 2022). Consequently, the magnitude and pattern of cardiovascular reactivity observed in the lab might differ from real-world scenarios (Kamarck et al., 2003). While this study made use of a laboratory-based assessment of reactivity, it also followed up on average 6.7 years later to assess health outcomes, allowing for a more ecologically valid study. Thus, making use of a longitudinal design, study one goes beyond the previous laboratory studies (i.e. Gallagher et al., 2020, 2021; Ginty et al., 2020) while also validating the model of positive well-being's prediction that gratitude will be associated with lowered adverse health outcomes through stress buffering (Schache et al., 2019).in order to assess the relationship between gratitude, reactivity, and adverse cardiovascular outcomes, it is necessary to assess cardiovascular reactivity. Indeed, it is a key component of the four aims of this research to do so. As such, assessing cardiovascular stress responses in a consistent manner will be highly important.

4.5.2 Cardiovascular reactivity and recovery

A key component of each aim of this thesis is the measurement of cardiovascular reactivity primary goal of this research involves exploring the association between gratitude and cardio-

vascular reactivity. Therefore, measuring reactivity accurately and scientifically is essential for any conclusions this thesis wants to draw. In the context of research on cardiovascular reactivity, there is a long tradition of laboratory-based research (e.g. Carroll et al., 2012), with reactivity being

typically measured in a laboratory (Kamarck et al., 2003). This is necessary given how difficult reactivity would be to measure outside the laboratory, which provides a controlled environment to measure blood pressure responses free from error (Schmidt & Hunter, 1996). Given the practical advantages, the research in this thesis assesses cardiovascular reactivity in a laboratory context.

Reactivity is typically computed as an arithmetic change score (Llabre et al., 1991; Lovallo, 2005). In this case, reactivity is computed as: Mean Cardiovascular Measures when stressed – Mean of baseline cardiovascular measures = Cardiovascular Reactivity. Studies suggest reactivity to be a reliable measure across heart rate, systolic blood pressure and diastolic blood pressure (Ginty et al., 2013). For example, test-retest correlations for HR reactivity have ranged from 0.32 to 0.91 (Manuck et al., 1993). Similarly, in a sample of 136 individuals, one study reports a Cronbach's alpha value of .99 for heart rate, .93 for systolic blood pressure, and .91 for diastolic blood pressure, demonstrating acceptable levels of internal consistency (Kelsey et al., 2007).

Cardiovascular recovery is similarly defined as the cardiovascular response to the cessation of stress (Hocking Schuler & O'brien, 2007). It can be measured as a change score like reactivity (Christenfeld et al., 2000). It has also demonstrated acceptable (r = .22 - .35) (Rutledge et al., 2000).

While research suggests that cardiovascular reactivity and recovery represents a stable disposition, their manifestations are context-dependent, with various factors influencing outcomes in different scenarios (Kamarck et al., 2003). Thus, optimizing testing conditions, like minimizing physical triggers (e.g., muscle movement) while maximizing psychological effort, is recommended (Kamarck & Lovallo, 2003). Similarly, aggregating data across tasks and sessions strengthens the reliability by reducing individual measurement variances and highlighting trait characteristics (Gallagher & Ashford, 2021; Llabre et al., 1991). These elements are integrated into the study protocols in the papers of these studies.

4.5.3 Experiment methods

The laboratory experiment is one of the most powerful tools available to researchers (Howitt & Cramer, 2010). Fundamentally, a laboratory-based experiment involves varying the level of the in- dependent variable systematically and then measuring the impact of this varying (Easterby-Smith et al., 2021; Howitt & Cramer, 2010). The advantages associated with the laboratory experiment consist in the standardisation of procedures (Howitt & Cramer, 2010). A general problem with conducting non-laboratory-based research is that researchers cannot control for confounding variables in the design of the study (Easterby-Smith et al., 2021). Ideally, experimental studies set in the laboratory can control and keep constant all variables apart from the ones being investigated (Howitt & Cramer, 2010). Given these advantages, the studies in this thesis all incorporate some aspect of experimental design.

The prior three studies on gratitude and reactivity made use of within-subjects correlation designs (Gallagher et al., 2020; Ginty et al., 2020). An experimental study can have a between-subjects or within-subjects design. A between-subjects design means that participants can receive different conditions of the experiment (Howitt & Cramer, 2010). A within-subjects design means that participants all receive the same conditions of the study (Howitt & Cramer, 2010). In line with past studies, Studies One and Two incorporated a within-subjects, correlational design into their methodology. While this design does not allow us to conclusively determine a causal relationship between reactivity and gratitude, it does possess the advantages of being easier to run and setting up preliminary evidence (Wang & Wu, 2020). Similarly, the within-subjects design allows for the comparison of effects within each person (Curran et al., 2010; Howitt & Cramer, 2010).

It is worth emphasizing that the accurate measurement of cardiovascular stress responses benefits strongly from an experimental design in a laboratory context. As it is a key goal of all studies in this research to assess cardiovascular reactivity, the use of experimental is highly appropriate.

Laboratory inducement of stress

When researching the physiological responses to stress, it is useful to have a method of inducing a considerable amount of stress in a controlled and ethical manner (Brouwer & Hogervorst, 2014); that is to say, as all of the aims of this research require measuring cardiovascular responses to stress, it is critical to have a valid method of doing so. Generally, acute stressors are examined under standardized laboratory conditions (Epel et al., 2018). This approach has the advantages of conforming to a positivist epistemology by standardising stress exposure and environmental factors, while also maintaining an ethical level of stress exposure (Weber et al., 2022). It is important to identify what features of a stressor are needed in order to manipulate the stress response (Epel et al., 2018). Otherwise, we may not actually be manipulating what we think we are (Brouwer & Hogervorst, 2014). A meta-analysis of 208 laboratory-based studies of acute stress concluded that a combination of social-evaluative threat (i.e. being judged by others) and uncontrollability (i.e. not being able to change the situation) are the two factors that produced the strongest stress response in humans (Dickerson & Kemeny, 2004). fMRI evidence suggests that controllability reduces key responses in threat-related brain areas (Limbachia et al., 2021) and, similarly, manipulating the social-evaluative threat of an acute stressor alters stress reactivity (Craw et al., 2021). This has led to standardised protocols used to elicit stress responses (Allen et al., 2016). The Trier Social Stress Test (TSST) is regarded as a gold-standard laboratory protocol for the induction of stress that combines both social-evaluative threat and uncontrollability (Allemand & Hill, 2016; Kirschbaum et al., 1993). In this protocol, participants must deliver an impromptu speech to an audience of judges followed by an arithmetic test (Allen et al., 2016), thus combining uncontrollability and social-evaluative threat (Brouwer & Hogervorst, 2014). Participants are given three minutes to prepare a speech on why they are a perfect candidate for a job and five minutes to deliver it to a panel of committee members; this is followed by a five minute arithmetic task (Allen et al., 2016). The TSST has been shown to successfully increase heart rate, between 10 and 25 beats per minute (BPM) on average (Brouwer & Hogervorst, 2014).

Limitations of the TSST include it being somewhat difficult and complicated to run (Allen et al., 2016). Due to the difficulty in running the TSST, this research has made modifications to

the protocol in studies Two and Three. However, these were in line with prior empirically valid modifications. For example, in lieu of requiring a panel of committee members, some researchers opt to make use of deceptions such as informing participants that their performance will be recorded and reviewed by experts at a later date (Narvaez Linares et al., 2020), with some research suggesting this to be associated with greater social-evaluative threat (Biondi & Picardi, 1999). Similarly, researchers have also omitted the speech component of the protocol, opting instead for just an arithmetic test (e.g. S. Gallagher et al. (2020); Lipovac et al. (2022)) or pairing an arithmetic test with a Stroop test (Hamid et al., 2019). The speech instructions have also been varied successfully to include a description of a participant's three best and three worst qualities (e.g. Gallagher et al., 2021). The time-spans for each component suggested above have also been inconsistently applied by past research, leading to a lack of agreement regarding how long these periods should last (Narvaez Linares et al., 2020).

Alternate stress-testing paradigms include orthostatic tests where participants are asked to stand with their hand on their head have also been used (Dimitrow & Sorysz, 2013), although tasks such as these constitute non-psychological, biogenic stressors (Everly, & Lating, 2019). Biogenic stressors are stressors which evoke a stress response without a cognitive appraisal, for example, extreme temperatures exert sympathomimetic effects (Everly, & Lating, 2019). Alternate psychological stressors include internet versions of the TSST (Almazrouei et al., 2023), which are more cost-effective to deploy, although this is not very useful if biological measures of stress responses are being used. However, as the TSST is the gold standard approach to studying cardiovascular responses to stress (Allen et al., 2016) and has been used in past research on state gratitude and cardiovascular stress responses (Ginty et al., 2020), this is the method of which this research will make use.

Biological assessment of stress

Stress is an umbrella term that captures the experience of individuals when the environment

places demand upon them that exceed their perceived ability to cope (Cohen et al., 2016). An important distinction is between the stressful events and the response to them. Stressors represent discrete, stressful events such as a traffic jam or job interview (Doan, 2021). Stress responses are the cognitive, emotional and biological responses to stressors (Doan, 2021). In the context of this thesis, the measurement of the stress response is more relevant (see Crosswell & Lockwood, 2020 for a discussion of measuring stressor exposure).

Stress responses can be measured psychometrically using self-report scales (Crosswell & Lock- wood, 2020). For example, the perceived stress scale is a ten-item, easy to use scale with established psychometric properties (Lee, 2012). However, stress responses can also be measured biologically, such as via electrocardiograph, cortisol, and blood pressure (Gormally & Romero, 2020), as is the case in the studies presented in this thesis.

Measuring stress responses biologically via blood pressure and heart rate makes for excellent science driven by a positivist epistemological paradigm. These measurements provide objective, quantifiable data, reducing the potential for subjective biases inherent in self-report measures (Arza et al., 2019; Llabre et al., 1991). Blood pressure and heart rate readings also deliver immediate and real-time insights into an individual's autonomic nervous system response, offering a direct window into the body's physiological reactions to stressors (McEwen, 2000; Szabo et al., 2020). Additionally, the non-invasive nature of these measurements makes them suitable for repeated assessments, allowing for the examination of dynamic changes over time or in response to various stimuli (Kamarck et al., 2003). This can be especially valuable in longitudinal studies aiming to understand the interplay between stress and health outcomes (Szabo et al., 2020). Furthermore, by employing standardized equipment and protocols, these biological markers ensure consistency and comparability across diverse study populations and settings (Howitt & Cramer, 2010). Overall, utilizing blood pressure and heart rate as indices of stress furnishes researchers with robust and reliable tools to gauge the body's immediate and direct response to environmental challenges. While the use of within-subjects experimentation in this context permits the leveraging for the experimental context for the purposes of stress induction and the biological assessment of stress,

a natural yet hitherto unused extension is the use of a randomised controlled trial design (Hariton & Locascio, 2018). While this approach had been adopted by prior research (Ginty et al., 2020), a randomised controlled trial offers a number of distinct advantages which will extend and solidify current research on the subject of gratitude and cardiovascular stress responses.

Randomised controlled trials

Study three in this thesis extends prior research methodologically by making of a randomised control design. A randomized controlled trial is useful due to its capability to ascertain causal relationships between variables (Hariton & Locascio, 2018). These designs can be regarded as a species of between-subjects experiment with the important characteristics of having random assignment to two groups for the purposes of causal interpretation (Akobeng, 2005; Kendall, 2003). This distinguishes it from the between-subjects experiment, which does not necessarily imply random assignment (Howitt & Cramer, 2010). Effective randomization will minimise confounding variables (Kendall, 2003), as it ensures that all confounding variables, whether observed or un- observed, are equally distributed across the groups, thus minimizing potential biases (Sibbald & Roland, 1998). A further important characteristic is blinding (Hariton & Locascio, 2018; Kendall, 2003). The researcher, participant, and anyone conducting data collection should be unaware what group a participant has been assigned to and what condition (either control or intervention) (Kendall, 2003). Within psychology, randomized controlled trials have been pivotal in evaluating the efficacy of therapeutic interventions, behavioural modifications, and a variety of other treatments, reinforcing their status as a gold standard in research methodology (Akobeng, 2005).

In the context of research on gratitude and reactivity, it would be expected that state gratitude should modulate cardiovascular reactivity (Gallagher et al., 2020). However, prior research has not investigated the efficacy of a gratitude intervention on reactivity, and it is prudent to do so in a randomised controlled trial context (Hariton & Locascio, 2018). Evaluating a gratitude

intervention in this context will distribute characteristics of participants which may bias the results evenly among the intervention and control groups, balancing potential baseline systematic differences (Akobeng, 2005; Kendall, 2003). The use of a randomised control trial design in Study Three allows it to assess the effects of a gratitude intervention on cardiovascular reactivity under highly rigorous conditions, thus contributing to prior research while making use of excellent scientific practice.

Open science practices

Study three was pre-registered with clinical trials.gov (ID: NCT05133063). The data for Studies 2 and 3 and code for Study 2 are available at the Open Science Framework at: osf.io/eg3au. The Mid Life in the United States data for Study 1 are publicly available through the Institute for Aging.

4.6 Conclusion

In conclusion, this thesis embraced a scientific realist ontology and positivist epistemology, assert- ing that objective reality exists independently and can be quantitatively measured. It emphasized the use of standardized experimental protocols and statistical modelling to mitigate subjective bias and produce objective, accurate descriptions of reality. By leveraging both primary and secondary data sources, and integrating methodologies like laboratory experiments and randomized control trials, this research contributes to the understanding of the interplay between gratitude, stress reac- tivity, and cardiovascular health outcomes.

Chapter 5

Heart rate reactivity mediates the relationship between trait gratitude and acute myocardial infarction[1]

5.1 Introduction

5.1.1 Evaluating the cardiovascular stress buffering effects of trait gratitude

Myocardial infarctions, also known as heart attacks, are defined by the WHO as the "demonstration of myocardial cell necrosis due to significant and sustained ischaemia" (Mendis et

[1] Note: This study has been accepted for publication: Leavy, B., O'Connell, B. H., & O'Shea, D. (2023). Heart rate reactivity mediates the relationship between trait gratitude and acute myocardial infarction. *Biological Psychology, 108663*. Advance online publication. https://doi.org/10.1016/j.biopsycho.2023.108663

al., 2011). In the United States, it is estimated that 660,000 patients suffer heart attacks for the first time each year and that 1 in 7 deaths are due to acute myocardial infarction (Mozaffarian et al., 2016; Smilowitz et al., 2017). Furthermore, the rate at which myocardial infarction occurs has also been found to be growing (Kuhn et al., 2022), leading researchers to call for cost-effective policies and interventions in order to meet the UN's goal of reducing premature mortality due to non-communicable deaths by a third (Roth et al., 2020).

Positive psychological constructs such as optimism, purpose in life, and positive thoughts (Boehm & Kubzansky, 2012) have been identified as potential low-cost areas of intervention that have positive associations with cardiovascular health Celano et al. (2017). Similarly, gratitude has been identified as a potentially useful area of intervention (Gallagher et al., 2020). Gratitude can be conceptualized at both state and trait levels (Wood et al., 2010). As a state, gratitude refers to momentary feelings of appreciation for the good things one has in their life. As a trait, gratitude refers to a predisposition to notice and appreciate what is good in the world (Wood et al., 2010). The potential value of gratitude lies in it being a straight-forward, low-cost, and clinically usable intervention (Boggiss et al., 2020; Wood et al., 2010). Recent research has found evidence that gratitude can play a role in cardiovascular health (Cousin et al., 2021; Redwine et al., 2016), and in modulating the cardiovascular response to acute stress (Cousin et al., 2021; Gallagher et al., 2020).

5.1.2 Stress buffering and cardiovascular reactivity

One pathway by which positive constructs may influence health is by buffering the negative effects of psychological or perceived stress (Boehm et al., 2011). This is called the stress-buffering hypothesis, and it proposes that positive emotions can mitigate negative reactions to stress, and thus protect individuals from the potential deleterious effects of stressful events (Fredrickson et al., 2000; Pressman et al., 2019).

Stress is a major risk factor in the development of cardiovascular illness (Phillips et al., 2011; Steptoe & Kivimäki, 2012), comparable to risks associated with obesity and hypertension (Osborne

et al., 2020). In the context of stress and cardiovascular health, it is important to consider cardiovascular reactivity due to its well-established relationship with cardiovascular disease (Phillips & Hughes, 2011). Cardiovascular reactivity refers to the magnitude of the change between an individual's baseline cardiovascular state and their state during acute psychological stress (Carroll et al., 2012). Until recently, research predominantly considered heightened cardiovascular reactions to stress to be associated with increased risk of onset and progression of cardiovascular disease (Hughes & Lü, 2017; Phillips et al., 2011). For example, exaggerated cardiovascular reactivity has been associated with negative health outcomes such as atherosclerosis, hypertension, and coronary heath disease mortality (Carroll et al., 2012; Hocking Schuler & O'brien, 2007; Jennings et al., 2004; Phillips et al., 2011) and myocardial infarction (Canto et al., 2012; Krantz et al., 1991; Manuck et al., 1992; Sundin et al., 1995).

However, recent research has suggested that blunted or 'too low' reactivity can also be associated with a range of adverse outcomes (O' Riordan et al., 2022). For example, in individuals with poorer cardiovascular health, blunted reactivity predicts a range of adverse cardiovascular outcomes such as cardiac arrest, cardiovascular hospitalization and death, angina, and myocardial infarction (Ahern et al., 1990; Kupper et al., 2015; Sherwood et al., 2017). One posited explanation for these relationships is that lower reactivity reflects the inability of the cardiovascular system to produce an appropriate response, which may be due to a pre-existing condition, for example (O' Riordan et al., 2022).

5.1.3 Gratitude and cardiovascular reactivity

Gratitude has been associated with coping more successfully with stress and adversity (Wood et al., 2010), and gratitude expressions are positively related to emotional regulation strategies such as reappraisal (Bryan et al., 2018) as well as goal-directed activities, which reduce the frequency and intensity of stress (Wood et al., 2007). Research has only recently begun to investigate physiological aspects of these relationships, with research to date showing that state gratitude has

a significant, inverse relationship with cardiovascular reactivity (Gallagher et al., 2020; Ginty et al., 2020). As heightened reactivity has traditionally been associated with poorer cardiovascular outcomes (Carroll et al., 2012) – although as has been discussed, blunted reactivity can be also - this suggests that gratitude may play a protective role for physical health. This association is consistent with the cognitive model of stress which posits that an individual's internal resources and characteristics influence how one copes and manages with stress (Lazarus, 1999), with positive emotions playing an important, restorative role in this model (Folkman, 2008).

Thus, gratitude's relationship with lower stress has been proposed as a potential mechanism by which gratitude is indirectly associated with cardiovascular health (Schache et al., 2019). Gratitude may not have a direct relationship with cardiovascular health, but rather operate through mechanisms such as improving health behaviours, improving physiological functioning, and buffering the negative effects of stress on physical health (Boehm, 2021; Schache et al., 2019; Soo et al., 2018).

5.1.4 Trait gratitude and cardiovascular health

Although three studies have shown that *state* gratitude has an inverse relationship with cardio- vascular reactivity (Gallagher et al., 2020; Ginty et al., 2020), a recent psychophysiological study found an association between *trait* gratitude and an increase in reactivity (Gallagher et al., 2021). As there is an established relationship between increased reactivity and cardiovascular disease (Carroll et al., 2012), this may lead to the confusing claim that trait gratitude may actually be worsening cardiovascular health. The inconsistencies in previous research may reflect the more recent discovery that both blunted reactivity – too low – and exaggerated reactivity – too high – may result in poorer health outcomes (O' Riordan et al., 2022; Whittaker et al., 2021).

Nonetheless, it has yet to be established whether the increase in reactivity associated with *trait* gratitude has any relationship with cardiovascular health outcomes. Our research explored this by examining the prospective, indirect relationship between trait gratitude, reactivity and

myocardial infarctions. Myocardial infarction is a serious health problem which causes substantial morbid- ity and mortality (Chi & Kloner, 2003), with studies estimating that a significant portion of sudden deaths globally are caused by myocardial infarctions (Solomon et al., 2005; Zaman & Kovoor, 2014). Exploring how gratitude may be associated with reductions in the likelihood of the occurrence of myocardial infarctions contributes to programmes of research in both preventative cardiology and positive psychology which examine how positive psychological constructs such as gratitude can cultivate cardiovascular health, including the occurrence of myocardial infarctions (Kubzansky et al., 2018; Labarthe et al., 2016).

Our research aimed to clarify whether trait gratitude is indirectly associated with cardiovascular health through reactivity**.** The claim that gratitude, as a positive emotion, is statistically associated with a lower rate of myocardial infarction through its capacity to modulate blood pressure reactivity and heart rate reactivity is evaluated (Cousin et al., 2021; Gallagher et al., 2020; Schache et al., 2019). Thus, this study is novel in that it tests these cardiovascular reactivity pathways to understand whether trait gratitude is associated with the likelihood of suffering acute myocardial infarction. Moreover, a longitudinal study design is used to evaluate the occurrence of myocardial infarctions as an outcome. In so doing, I hope to clarify how increases in reactivity associated with trait gratitude are related to the occurrence of acute myocardial infarctions. As such, this study proposes:

Hypothesis 1: Systolic blood pressure reactivity mediates the relationship between trait gratitude and the occurrence of acute myocardial infarction.

Hypothesis 2: Diastolic blood pressure reactivity mediates the relationship between trait gratitude and the occurrence of acute myocardial infarction.

Hypothesis 3: Heart rate reactivity mediates the relationship between trait gratitude and the occurrence of acute myocardial infarction.

5.2 Method

5.2.1 Study overview and design

This study made use of the publicly available Mid-life in the United States study (referred to as MIDUS) dataset (Radler, 2014). Between 1995 and 1996 the first wave of the Mid-life in the United States study was carried out using telephone interviews and questionnaires with over 7,000 participants. The aim of these studies was to investigate the roles of behavioural, psychological, and social factors in understanding age-related differences in both mental and physical health. Detailed information on retention and response rates can be found in Radler & Ryff (2010). Participants were contacted to participate in a second wave in 2004 (MIDUS 2; N = 4963). These MIDUS 2 participants were invited to complete a separate biological assessment called the MIDUS 2 Biomarker Project (N = 1054) (Radler, 2014). The primary reasons for not participating in this biological assessment were (1) participants did not wish to travel to the clinic, (2) had family obligations, (3) were too busy, or (4) were not interested (Dienberg-Love et al., 2010). Between 2013 and 2014, MIDUS 3 (N = 3295) completed a third wave of data collection on the same sample. Of the 1,255 participants who were part of the Biomarker Project, 945 were retained at MIDUS 3. The present study uses participants who completed MIDUS 2, the MIDUS 2 Biomarker Project and MIDUS 3. Detailed information on the study protocol and measures are found in and colleagues (2018).

1,255 individuals participated in the in the MIDUS 2 Biomarker project, comprising two subsamples: the longitudinal sample (N = 1,054) and the Milwaukee sample (N = 201) (Ryff et al., 2010). The Milwaukee sample does not have data at MIDUS 3, and hence, was not included in the present study. Thus, this study used 1,054 from MIDUS 2 (1,255-201 = 1,054). Of these, 945 were retained at MIDUS 3. 20 individuals who completed a different protocol and 13 individuals who had heart attacks prior to the MIDUS 2 study (to ensure that the sample only included individuals who suffered heart attacks after the biological assessment) were excluded. This

resulted in a sample of 912 participants used in the present study. Ages ranged from 35 to 86 (M = 57.06, SD = 10.97). 55.71% were female, 22.6% had high school education or less, and the mean number of chronic conditions was 2.16 (SD = 2.13), with 43% reporting a hypertension diagnosis and 9.6% reporting a diabetes diagnosis.

5.2.2 Participants

Table 5.1: Descriptive statistics of sample.

Name	Mean	SD	Min	Max	N (%)	Missing (%)
Heart attack (Yes)					23 (2.5)	0
Years between measure	6.67	1.3	4	9		0
Trait gratitude	6.29	0.81	2	7		0
Systolic blood pressure reactivity	13.87	10.6	-18.3	65.6		27
Diastolic blood pressure reactivity	6.52	4.03	-7.15	21.4		27
Heart rate reactivity	3.83	3.91	-6.4	29.4		12
Age	57.06	11.97	35	86		0
BMI	28.98	5.97	16.49	60.39		0
Education	2.33	0.82	1	3		0
High school or less					206 (22.6)	
Some college					198 (21.7)	
College degree minimum					506 (56.6)	
Diabetes (Yes)					87 (9.6)	0
Sex (Female)					483 (55.71)	0
High blood pressure (yes)					297 (32.9)	1
Number of chronic conditions	2.16	2.13	0	16		0
Ever smoked (Yes)					394 (43.2)	0

Table 5.2: Comparison of reactivity scores (means, SDs) for those who suffered acute myocardial infarctions compared to those who did not.

Acute myocardial infarction	N	SBP reactivity mean	SBP reactivity SD	SBP reactivity min/max	DBP reactivity mean	DBP reactivity SD	DBP reactivity min/max	HR reactivity mean	HR reactivity SD	HR reactivity min/max
No	889	13.95	10.66	-0.278963415	6.54	4.06	-0.33411215	3.86	3.93	-0.217687075
Yes	23	9.68	5.9	2.35/22.95	5.51	2.21	2/10.8	2	1.65	-0.226190476

Note. SBP = Systolic blood pressure (mmHG), DBP = Diastolic blood pressure (mmHG), HR = Heart rate (BPM).

5.2.3 Measures

Occurrence of acute myocardial infarction was measured in telephone interviews in both MIDUS 2 and MIDUS 3. Participants were asked if they had any heart trouble and if so, whether they had been diagnosed as having had a heart attack. To establish which participants had suffered acute myocardial infarction between being assessed at MIDUS 2 and MIDUS 3, participants who reported heart attacks at MIDUS 2 were excluded. This left only participants who suffered heart attacks between MIDUS 2 and MIDUS 3.

Cardiovascular measurement. Heart rate was measured using a beat-to-beat electrocardiogram (ECG). Beat-to-beat analogue ECG signals were collected and then digitised at a sampling rate of 500 Hz. This was conducted using a 16-bit National Instruments analogue-to-digital board attached to a micro-computer. Heart rate was then calculated as the average of all valid inter-beat intervals and then translated to beats-per-minute. Systolic and diastolic blood pressure were recorded using a Finometer monitor (Finapres Medical Systems, Amsterdam, Netherlands) which accurately as- sesses absolute blood pressure (Schutte et al., 2003). A finger cuff was placed on the middle finger of the non-dominant hand and an arm cuff on the upper arm on the same side. In the MIDUS 2 dataset, this resulted in two baseline averages corresponding to the mean blood pressure readings for the first and last 6 minutes of the baseline period. Similarly, the data set contains two averages for the stress tasks.

Cardiovascular reactivity is defined as the arithmetic difference between task and baseline averages (Gallagher et al., 2020). In line with previous research, reactivity was computed for systolic blood pressure, diastolic blood pressure and heart rate (Gallagher et al., 2020; Ginty et al., 2020). In the MIDUS 2 dataset, an average score for two stress tasks - the Stroop task and an Arithmetic task - is provided. To arrive at a single, overall score for these stress tasks, the average of the average scores was taken for each of these tasks, as this has been suggested to increased reliability and generalizability (Kamarck & Lovallo, 2003). Cardiovascular reactivity was then

calculated. To do this, the average baseline (or 'resting') score was subtracted from the overall stress score for systolic blood pressure, diastolic blood pressure, and heart rate.

Trait Gratitude was assessed using two items from the Subjective Well-Being scale (McCullough et al., 2002) which were extracted from the gratitude questionnaire (GQ-6) (Jans-Beken et al., 2015). Participants were asked to rate their agreement with two statements on a 7-point Likert scale (1 = strongly disagree to 7 = strongly agree). These statements were: "I have so much in life to be thankful for", and "I am grateful to a wide variety of people." Cronbach's alpha for this scale was 0.73. This was measured as part of MIDUS 2.

Stress tasks. The stress tasks comprised a Stroop task and an arithmetic task. For the Stroop task participants were seated in front of a computer and coloured words appeared on screen. These words either matched the colour or did not and were subsequently judged as either congruent or incongruent (i.e., the word "red" written in red letters was congruent, but the word "red" written in yellow letters was incongruent). Participants used a keypad to respond to find the colours of the letters, not the name of the colour. Participants were also informed that the computer "will score your responses for speed and accuracy. If you don't respond quickly enough, it will score your response as incorrect and present a new problem." This task lasted 6 minutes and was followed by a 6-minute recovery period.

The arithmetic task used the Morgan and Turner Hewitt mental arithmetic task, which requires participants to complete several addition and subtraction problems (Turner et al., 1986). A problem was presented on screen with an equal to sign and participants pressed a key to indicate whether the answer presented was correct or incorrect. Problem difficulty varied. If participants gave a correct answer they were subsequently presented with a more difficult problem. If an incorrect answer was given, a less difficult problem was subsequently presented. Participants were informed that if they did not answer sufficiently quickly their answer would be scored as incorrect (Coyle et al., 2020). This task lasted 6-minutes and was followed by a 6-minute recovery period.

Perceived stress was assessed at baseline and after each stress task. Participants were verbally asked by the researcher for a stress rating from 1 to 10, with 1 being not stressed at all and 10 being

extremely stressed. These were captured to confirm that the stress tasks were psychologically stressful. Such items have been used in similar studies (e.g. Gallagher et al., 2021). An overall average was computed for both stress tasks.

Control variables were selected based on their well-established relationships with cardiovascu- lar reactivity and cardiovascular health. These control variables used were: socio-economic status (Coughlin & Young, 2020), diabetes (Jacoby & Nesto, 1992), body mass index (BMI) (Bucholz et al., 2012), age, sex (Canto et al., 2012), and high blood pressure (Creaven et al., 2020), and whether participants had ever smoked (Elkhader et al., 2016).

Education was used as a proxy for socioeconomic status and coded as 'high school or less', 'some college', and 'college and higher'. It was measured at MIDUS 2. Diabetes was assessed by asking participants had they ever been diagnosed with diabetes. It was coded as either 'yes' or 'no' and it was measured at the MIDUS 2 biomarker project. BMI was calculated by dividing weight by height squared it was measured at the MIDUS 2 biomarker project. Smoking status was coded as ever having been a smoker or not it was measured at the MIDUS 2 biomarker project.

5.2.4 Procedure

Participants at MIDUS 2 were admitted for a two-day hospital stay in one of three participating sites. On day one, they completed self-administered questionnaires which assessed various psychological constructs and demographic questions, as well as a 45-minute medical exam which included a medical history and physical exam. After breakfast on the second day, participants received a standardised experimental protocol examining the response to cognitive challenges similar to stressors experienced in everyday life. The session lasted 90 minutes. Participants sat quietly for 11 minutes for a formal baseline before undergoing their first cognitive stress task followed by a 6-minute recovery period followed by the second cognitive stress task and a 6-minute recovery. The stress tasks were presented in random order.

Following the stress tasks, participants were asked to hand in their completed self-

administered questionnaires and were then debriefed. This protocol has been outlined in detail elsewhere (e.g. Dienberg-Love et al., 2010, Ryff et al., 2011), and includes further details on the collection of blood samples, urine samples, saliva samples, the measurement of respiration, and heart rate variability. The sections relevant to the current study are focused on.

5.2.5 Data reduction and analysis

R version 4.2.0 was used to prepare the data. This study used MPlus (version 8.2) for all analyses. Checks for normality and assumption checking were carried out using inspection of Q-Q plots, histograms, and Shapiro tests, where all variables had p-values > .05. Manipulation checks were carried out using paired-samples t-tests to confirm that the stress tasks increased blood pressure. Exploratory comparisons between participants suffering myocardial infarctions and those who did not were conducted were conducted using independent samples t-tests. In cases where equal variances were not assumed, Welch's two-sample t-test is reported which can result in degrees of freedom which are smaller or in decimal form (Whitlock & Schluter, 2015).

To test the hypotheses, a logistic regression parallel mediation model was conducted in Mplus using a maximum likelihood estimator. Gratitude (assessed at MIDUS 2) was entered as the predictor variable, systolic and diastolic blood pressure reactivity, and heart rate reactivity (assessed during the MIDUS 2 Biomarker Project) were entered as parallel mediators, and myocardial infarction (assessed at MIDUS 3) was entered as a dichotomous outcome variable. All previously mentioned control variables were entered as control variables.

Maximum likelihood estimation was used and was appropriate as it makes use of all available data, meaning participants with some missing data were not excluded. 27% of observations were missing for systolic and diastolic reactivity, and 12% for heart rate reactivity. As Dong & Peng (2013) and Newman (2014), when >10% of observations are missing, full information maximum

likelihood should be used to ensure unbiased estimates. Additionally, to account for the binary outcome, the mediation model was estimated using logistic regression (Feingold et al., 2019)

Preacher & Hayes (2008) recommend using bias corrected confidence intervals to test indirect effects. Following these recommendations, 1,000 bootstrapped samples with bias corrected 95% confidence intervals were used. As our mediators were entered in parallel, each of our three indirect effects were assessed while accounting for the other two. This is important for building parsimonious models (Preacher & Hayes, 2008), and reducing parameter bias owing to omitted variables (Judd & Kenny, 1981). Standardised estimates are reported for direct effects with continuous outcomes. Estimates for categorical outcomes are reported in log-odds scale. This includes estimates for indirect effects which are also reported in odds ratio scale meaning that they are regarded as statistically significant if the confidence intervals do not intersect zero.

Ethical approval was not required form Maynooth Biomedical and Life Sciences Research Ethics Sub-Committees the "Please note: Ethical approval is not required for secondary use of anonymous data" (Maynooth University, 2024).

5.3 Results

5.3.1 Descriptive statistics

Descriptive statistics for the study variables are reported in Table 5.1, and correlations are reported in the appendices. On average, 6.7 years elapsed between participation in the MIDUS 2 Biomarker project and MIDUS 3, with a minimum of 4 and maximum of 9 years. 2.5% of the sample reported suffering a heart attack between MIDUS 2 and MIDUS 3. At MIDUS 3, 76.32% reported having at least one chronic underlying condition in the past 12 months, with 7.7% reporting a diabetes diagnosis. Women reported a larger number of chronic health conditions at MIDUS 2, $t(789.32) = 3.61$, $p < .001$.

Table 5.3: Means and standard deviations for systolic blood pressure, diastolic blood pressure, and heart rate.

Name	Mean	SD
SBP baseline	123.9	19.2
DBP baseline	61.5	12
HR baseline	72.8	10.7
SBP pooled task	136.7	21.6
DBP pooled task	67.7	12.3
HR pooled task	76.6	11.3
SBP Stroop task	138.9	22.2
DBP Stroop task	68.4	12.5
HR Stroop task	77.2	11.6
SBP math task	134.9	21.6
DBP math task	66.8	12
HR math task	76	11.2

Note. SBP = Systolic blood pressure (mmHG), DBP = Diastolic blood pressure (mmHG), HR = Heart rate (BPM).

There was a high correlation between systolic blood pressure reactivity and diastolic blood pressure reactivity (N = 660, r = .79, p <.001). Trait gratitude had a positive and significant correlations with systolic, diastolic and heart rate reactivity (see Table A.1 in Appendix A). The average heart rate reactivity and diastolic blood pressure were quite low in the sample, with values beneath those that recent research has found to constitute a threshold for blunted reactivity (O' Riordan et al., 2022), see Table 5.2. Those who suffered heart attacks by MIDUS 3 had lower heart rate reactivity at MIDUS 2 than those who did not, t(14.35) = 3.89, p = .002; there were no significant differences for systolic or diastolic reactivity.

5.3.2 Manipulation checks

Paired samples t-tests were conducted on the baseline perceived stress and the mean of the perceived stress ratings for the two stress tasks, confirming that participants experienced stress during the tasks t(853) = 44.25, p <.001, d = 1.51. Paired samples t-tests between baseline cardiovascular measures and the cardiovascular stress responses averaged across the two stress tasks confirmed that the stress tasks increased cardiovascular responses for systolic blood pressure, t(661)= 33.67, p <.001, d = 1.31, diastolic blood pressure t(661) = 41.60, p<.001, Cohen's d = 1.62, and heart rate t(801) = 27.75, p <.001, Cohen's d = 0.98, See Table 5.4 for descriptive statistics and cardio- vascular parameters.

5.3.3 Hypothesis testing

It was hypothesised that gratitude would have an association with acute myocardial infarction through systolic reactivity, diastolic reactivity, and heart rate reactivity. This was tested using the MODEL INDIRECT command in Mplus version 8.2. Looking first to the direct effects, the paths from trait gratitude to systolic blood pressure reactivity (—= 0.09, p = 0.012), diastolic blood pressure reactivity (— = 0.08, p = 0.043), and heart rate reactivity (— = 0.10, p = 0.003) were statistically significant and positive in nature. Furthermore, there was no direct association between trait gratitude and the occurrence of acute myocardial infarction in the logistic regression analysis. Coefficients for the paths are illustrated in Figure 5.1, and summarised in Tables 5.4 and 5.5.

Regarding the indirect effects, (see Table 5.5), there were no significant indirect effects for either systolic blood pressure reactivity, — = -0.079, 95%CI [-0.253, 0.015], or diastolic blood pressure reactivity — = 0.032, 95%CI [-0.050, 0.174]. Thus, our findings do not support hypotheses 1 and 2. In support of hypothesis 3, there was a significant indirect effect through heart rate reactivity, — = -0.098, 95%CI [-0.331, -0.010], meaning that, while statistically controlling for age, sex, BMI, high blood pressure, education, diabetes, whether or not participants had ever smoked, trait gratitude was associated with a lower likelihood of suffering a heart attack through its effect

on heart rate reactivity. Furthermore, because the mediators were included in parallel, this effect also account for the effects of systolic and diastolic blood pressure reactivity.

Table 5.4: Model Coefficients for the Parallel Mediation Model with three mediators and covariates

	Consequent											
	Parallel mediator									Outcome		
Antecedent	SBP reactivity			DBP reactivity			HR reactivity			Myocardial infarction		
	Coeff	SE	P	Coeff	SE	P	Coeff	SE	P	Log odds	SE	P
Baseline	-0.02	0.05	0.73	-0.03	0.04	0.45	-0.03	0.04	0.44			
Age	0.25	0.04	<0.001	0.19	0.04	<0.00	-0.08	0.04	0.03	0.09	0.03	<.001
Sex	0.08	0.04	0.05	0.01	0.04	0.79	-0.02	0.04	0.51	1.08	0.81	0.18
BMI	0.05	0.04	0.1	-0.01	0.04	0.85	-0.06	0.04	0.08	0.06	0.04	0.14
Trait gratitude	0.09	0.04	0.01	0.08	0.04	0.04	0.1	0.03	<0.001	-0.12	0.36	0.74
Education										-0.43	0.3	0.15
Ever smoked										0.09	0.53	0.86
High blood pressure										-0.39	0.59	0.52
Diabetes										0.51	1.19	0.67
SBP reactivity										-0.07	0.04	0.13
DBP reactivity										0.09	0.13	0.48
HR reactivity										-0.21	0.13	0.11
	R^2=0.08			R^2=0.05			R^2=0.020			R^2=0.45		
	P = < .001			P = 0.002			P = 0.064			P =< .001		

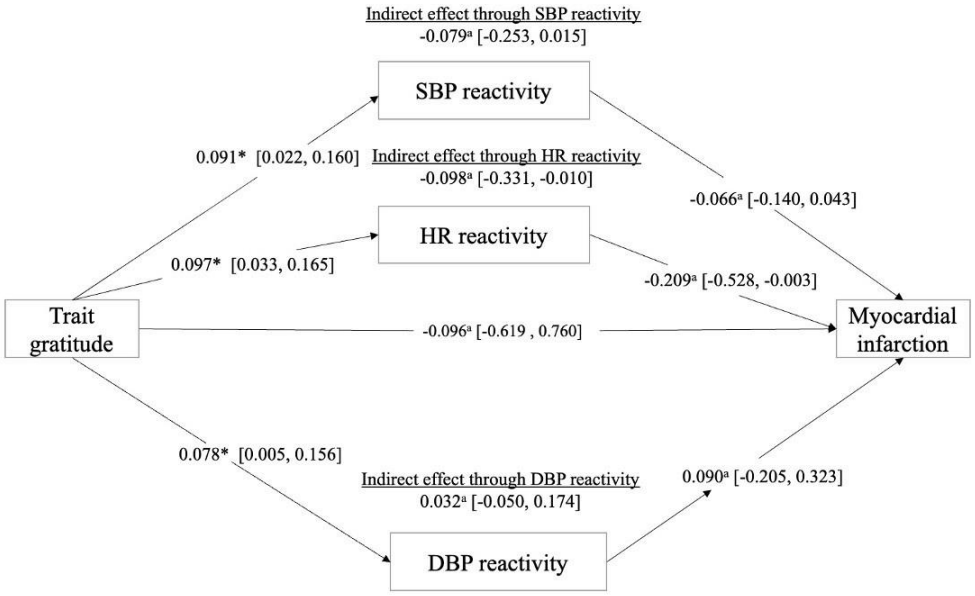
Note. SBP = Systolic blood pressure (mmHG), DBP = Diastolic blood pressure (mmHG), HR = Heart rate (BPM).

Table 5.5: Results of mediation analysis predicting myocardial infarction: indirect relationships between trait gratitude and myocardial infarction through three reactivity measures.

			BC 95% CI	
	Estimate	SE	Lower	Upper
Indirect effects	Predictor: Trait gratitude			
Total indirect effect	-0.142	0.359	-0.662	0.766
Unique effects:				
1. Systolic blood pressure reactivity	-0.079	0.064	-0.253	0.015
2. Diastolic blood pressure reactivity	0.032	0.058	-0.050	0.174
3. Heart rate reactivity	-0.098	0.080	-0.331	-0.010

Note. BC 95% CI refers to the bias-corrected 95% confidence interval using 1,000 bootstrap samples. All estimates are reported in log-odds scale; estimates with CIs that do not include zero are statistically significant and bolded.

Figure 5.1: Parallel mediation analysis of the relationship between trait gratitude and myocardial infarction through the cardiovascular reactivity parameters



Note: SBP = Systolic blood pressure (mmHG), DBP = Diastolic blood pressure (mmHG), HR = Heart rate (BPM). ᵃ indicates that the estimate is given in log odds scale. Otherwise, standardised estimates are reported. * Indicates $p < .016$.

5.3.4 Supplementary Analyses

Several additional analyses were conducted to ensure the robustness of our results and also to rule out alternative explanations. Further details and statistical output regarding the supplementary analyses can be found in Appendix A.

First, due to the high correlation between systolic and diastolic blood pressure reactivity, three additional mediation models were conducted to examine separately systolic blood pressure reactivity, diastolic blood pressure reactivity, and heart rate reactivity. Findings showed that heart rate reactivity continued to mediate the relationship between trait gratitude and myocardial infarction; there was also a significant indirect effect for systolic blood pressure reactivity, but no effect was detected for diastolic reactivity (see appendix A).

Second, an additional model controlling for positive affect was run, as previous research

suggests a relationship between positive affect, gratitude and cardiovascular health (Ginty et al., 2020; Pressman et al., 2019; Schache et al., 2019). Findings show that heart rate reactivity continued to mediate the relationship between trait gratitude and myocardial infarction, but no effect was detected for systolic or diastolic reactivity (see Appendix A, tables A.2 and A.3).

Finally, an additional model controlling for depressive affect was run, as previous research suggests a relationship between depressive affect, gratitude, and cardiovascular health (Bouzinova et al., 2015; Ginty et al., 2020). Findings show that heart rate reactivity continued to mediate the relationship between trait gratitude and myocardial infarction, but no effect was detected for systolic or diastolic reactivity (Appendix A, tables A.4 and A.5).

5.4 Discussion

Evidence from a growing body of research suggests that gratitude can be associated with cardiovascular health outcomes through its modulation of stress responses (Jans-Beken et al., 2020; Schache et al., 2019). However, the underlying processes have not been fully examined. The potential indirect associations between trait gratitude on acute myocardial infarction through systolic blood pressure reactivity, diastolic blood pressure reactivity, and heart rate reactivity were tested. A significant indirect association between trait gratitude and myocardial infarctions, through heart rate reactivity, was detected, meaning that increased trait gratitude was associated with decreased risk of suffering a heart attack through increases in heart rate reactivity. However, no significant indirect effects through either systolic blood pressure reactivity or diastolic blood pressure reactivity were found. Significant, positive direct associations between trait gratitude and systolic blood pressure reactivity, diastolic blood pressure, and heart rate reactivity were also found. There were no direct associations between trait gratitude and risk of myocardial infarction.

Looking first to the direct effects, the results of our present study suggest that trait gratitude was associated with increased reactivity, and this increase was associated with a reduced risk of

suffering acute myocardial infarction. This finding is seemingly inconsistent with the stress buffering hypothesis insofar as trait gratitude has a positive relationship with all cardiovascular reactivity parameters and increases in cardiovascular reactivity are associated with negative outcomes (Phillips & Hughes, 2011). However, previous research has suggested that an increase in reactivity as- sociated with trait gratitude may reflect moderate or healthy responses to stress (S. Gallagher et al., 2021). These authors suggest that responding to stress requires the mobilization of resources and positive emotions facilitate this. For example, happiness has previously been associated with increased cardiovascular reactivity (Framorando & Gendolla, 2019). Framorando and Gendolla (2019) suggest that emotions like happiness lead to individuals appraising tasks as less demanding and subsequently mobilizing higher effort, leading to higher blood pressure. This leads S. Gallagher et al. (2021) to suggest that gratitude may increase engagement. Thus, the result of our study may reflect the capacity of trait gratitude to buffer against the deleterious effects of stress by helping to mobilise resources complete challenging tasks, which is consistent with the stress buffering hypothesis.

Consistent with this line of reasoning, it was found that increases in heart rate reactivity mediated the relationship between trait gratitude and a decreased risk of acute myocardial infarction. In the context of the previously discussed direct effects, this implies that – despite increases in reactivity – trait gratitude is associated with more positive cardiovascular outcomes, providing further evidence for gratitude playing a health-protective role. This is consistent with predictions that positive emotions like gratitude are associated with better health outcomes (Fredrickson, 2004; Jans-Beken et al., 2020; Schache et al., 2019).

Concomitantly, it was found that individuals who suffered acute myocardial infarctions had significantly lower heart rate reactivity than those who did not. This is inconsistent with some previous research which reported positive associations between reactivity and risk of myocardial infarction (Carroll et al., 2012). However, while increased reactivity has previously been found to be cardio-toxic (Phillips & Hughes, 2011), some recent research suggests that blunted reactivity is associated with an increased risk of myocardial infarctions (O' Riordan et al., 2022). For example,

a study of 100 patients found that increased heart rate reactivity was associated with a reduced risk of cardiovascular morality (Kupper et al., 2015). This may reflect chronotropic incompetence, or the inability to increase heart rate to match cardiac output to metabolic demands (Brubaker & Kitzman, 2011; Kupper et al., 2015). Chronotropic incompetence has been associated with cardiovascular disease (Brubaker & Kitzman, 2011), including myocardial infarction (Savonen et al., 2008).

This may partially align with the suggestion that gratitude reduces cardiovascular disease by improving physiological function (Boehm, 2021; Schache et al., 2019), implying that increases in reactivity associated with trait gratitude reflect the adequacy of the cardiovascular system to respond to acute mental exertion. As such, higher trait gratitude may more holistically reflect healthier lifestyles and physiological functioning rather than stress coping resources (Boehm, 2021). For example, research has found that other positive constructs like optimism are associated with more frequent exercise, healthier foods and a lower likelihood of smoking cigarettes (Virani et al., 2020).

Although it was found that heart rate reactivity mediated the relationship between trait gratitude and a decreased risk of acute myocardial infarction, no indirect relationships between systolic blood pressure reactivity or diastolic blood pressure reactivity and risk of myocardial infarction were found, which is inconsistent with previous predictions. Nonetheless, there was also high correlation between both diastolic and systolic reactivity in our study. In a simulation study exploring how bootstrapping methods are impacted by correlated mediators, results showed that when mediators are highly correlated, there was a lower likelihood of the confidence intervals to include the true values of the correlated parameters (Beasley, 2014). To investigate this, the analyses were re-runs, while excluding systolic blood pressure reactivity or diastolic reactivity, and confirmed that the pattern of results remained the same. However, it is worth noting that when looking at the supplementary analyses, which included only systolic blood pressure reactivity as the mediating variable, this mediator emerged as a significant indirect pathway by which gratitude reduces acute myocardial infarctions. This is more consistent with past findings (Gallagher et al.,

2020), and may suggest that the high correlations between systolic blood pressure reactivity may have masked their effects. Nonetheless, this requires further investigation before any conclusions may be drawn.

Moreover, additional supplementary analyses were conducted to ensure that our findings were specific to gratitude (see Appendix A). Positive affect was controlled for to demonstrate that trait gratitude continued to have a significant indirect effect on the risk of the occurrence of acute myocardial infarction. In addition, it was checked that our findings were not just the reverse of the statistical effects of depressive affect by conducting supplementary analyses with depression as an additional control variable, see table A5 in Appendix A. Here, our findings continue to demonstrate that gratitude continues to have an indirect relationship with myocardial infarction through heart rate reactivity. Thus, there is reasonable confidence in the robustness of our findings. Taken together, our findings suggest that higher levels of trait gratitude are associated with higher heart rate reactivity, and through this increase are associated with a lowered risk of acute myocardial infarction.

5.4.1 Strengths and Limitations

This study has several limitations. The MIDUS 2 survey only used two questions from the GQ- 6 scale to assess gratitude, making it difficult to compare it to other studies which used the full scale. The present study only included individuals who suffered heart attacks and survived. There was no laboratory-based manipulation of gratitude. It would be preferable if there were more measurement periods as this would allow for a more complete picture of participant health, over time. A further limitation is that the stress tasks used to induce stress (i.e., the Stroop test and arithmetic task) can be viewed as non-evaluative, asocial, and low threat. This may help to explain why stress responses were relatively muted compared to other large studies (Creaven et al., 2020). The present study did not examine the stress buffering hypothesis using the one-item self-report stress measure reported in MIDUS due to limitations associated with simple self-report measures

of stress (Epel et al., 2018).

While gratitude has shown consistent beneficial effects for health (Boggiss et al., 2020; Hill et al., 2013), this is not to say that one should only practice gratitude for the putative health benefits. Rather, it is to acknowledge exploring the determinants of health mean, examining both negative and positive psychological constructs (Fredrickson, 2004). In this case, the gap in past research is that it has predominantly focused on gratitude and self-reported health (Boggiss et al., 2020; O'Connell & Killeen-Byrt, 2018), and the novelty of our paper is that it shows how gratitude can also have effects on objective physical health outcomes in cardiovascular reactivity.

Finally, due to the design of the study, causal relationships cannot be inferred. However, this study also has several strengths and novel contributions. It makes use of a standardised and well- controlled laboratory-based stress tasks to assess reactivity. It is the first study to longitudinally assess the association between trait gratitude and the risk of heart attacks. Furthermore, it helps to clarify that increases in cardiovascular reactivity associated with trait gratitude do not necessarily result in poorer cardiovascular outcomes.

5.4.2 Future directions

It would be useful to extend the present analysis by examining other cardiovascular outcomes such as hypertension. Accordingly, it would also be useful to explore the extent to which trait gratitude impacts reactivity by reducing stress or by improving physiological functioning. For example, in previous stress-buffering models (Pressman et al., 2019a), one way by which gratitude may buffer the effects of stress is by interacting with how stressful the task is perceived. It would be useful to assess this relationship in a stress-testing protocol context, as well as assessing the relationship between trait gratitude and chronotropic incompetence. Additionally, with further research suggesting that the relationship between reactivity and health may be curvilinear (Phillips et al., 2013), it would be helpful to explore how gratitude is related to both exaggerated reactivity and blunted reactivity. It would be useful to address how gratitude, depressive affect, and positive

affect influence each other's relationships to cardiovascular outcomes.

It is also recommended that future research investigate the association between gratitude and reactivity in the context of a randomised control trial using a gratitude induction. Finally, it would be useful to explore this relationship in a latent variable modelling framework in order to more accurately examine how systolic and diastolic reactivity may mediate the relationship between trait gratitude and acute myocardial infarctions.

Finally, these findings have clinical utility. Gratitude interventions are low-cost and easy to use (A. M. Wood et al., 2010). For example, gratitude lists whereby individuals write down three to five things for which they are grateful have been shown to have a number of beneficial effects (Kerr et al., 2015; Manthey et al., 2016). Previous research shows that the use of gratitude journals in cardiac samples improves outcomes (Redwine et al., 2016). Combined with the results of this study and previous work, gratitude may constitute a useful point of intervention for the improvement of cardiovascular health.

5.5 Conclusions

In conclusion, this study found that heart rate reactivity significantly mediated the relationship be- tween trait gratitude and the occurrence of acute myocardial infarction. Higher trait gratitude was associated with lower likelihood of suffering acute myocardial infarction 6.7 years later, through changes in heart rate reactivity, even when controlling for age, sex, BMI, education, high blood pressure and diabetes. This suggests that gratitude may buffer the negative physiological consequences of stress and overall improving cardiovascular outcomes.

These novel findings help further clarify that increases in cardiovascular reactivity, associated with trait gratitude, do not necessarily result in poorer cardiovascular outcomes. They also demonstrate that positive psychological constructs have beneficial impacts of cardiovascular health. In sum it may be said that this study contributes to our understanding

of how gratitude impacts physical health.

Chapter 6

Gratitude, affect balance, and stress buffering: a growth curve examination of cardiovascular responses to a laboratory stress task[2]

6.1 Abstract

Previous research has indicated that gratitude and affect-balance play key stress-buffering roles. However, to date there is limited research on the impact of gratitude and affect balance on cardiovascular recovery from acute psychological stress, and whether affect balance moderates the

[2] This study has been accepted for publication: Leavy, B., O'Connell, B. H., & O'Shea, D. (2023). Gratitude, affect balance, and stress buffering: A growth curve examination of cardiovascular responses to a laboratory stress task. *Inter- national Journal of Psychophysiology: Official Journal of the International Organization of Psychophysiology, 183*, 103–116. https://doi.org/10.1016/j.ijpsycho.2022.11.013

relationship between gratitude and cardiovascular reactions to acute psychological stress. In this study, 68 adults completed measures of state gratitude, positive and negative affect, and completed a laboratory-based cardiovascular stress-testing protocol. This incorporated a 20-minute acclimatization period, a 10-minute baseline, a 6-minute arithmetic stress task, and an 8-minute recovery period. Mixed-effects growth curve models were fit and the results indicated that state gratitude predicted lower systolic blood pressure responses throughout the stress-testing period. Affect balance was found to moderate the association between state gratitude and diastolic blood pressure responses to stress, amplifying the effects of state gratitude. These findings suggest that state gratitude has a unique stress-buffering effect on both reactions to and recovery from acute psychological stress.

6.2 Introduction

Psychological stress refers to situations where an individual perceives that the demands placed upon them exceed their ability to cope (Cohen et al., 2007). While stress cannot be avoided, it can have a negative impact on an individual's health and well-being. Notably, epidemiological data shows that stress predicts increased cardiovascular morbidity and coronary heart disease (Kivimäki & Steptoe, 2018; Steptoe & Kivimäki, 2012). The cardiovascular stress response is a complex and dynamic process; two important aspects of this response are cardiovascular reactivity to stress and cardiovascular recovery from stress (Felt et al., 2017; Llabre et al., 2004; Woody et al., 2018). Where cardiovascular stress reactivity can be defined as the response to challenging conditions, cardiovascular stress recovery refers to the response following cessation of the stressor (Panaite et al., 2015). It is proposed that state gratitude and affect balance modulate both of these components of the stress response.

Past studies have consistently shown that large-magnitude responses to stress are associated with cardiovascular outcomes such as high blood pressure, hypertension, and cardiovascular dis-

ease mortality (Chida & Steptoe, 2010; Treiber et al., 2003). Increased cardiovascular reactivity has been associated with poorer health outcomes such as high blood pressure and hypertension (Carroll et al., 2012; Hocking Schuler & O'brien (2007); Yuenyongchaiwat, 2015). Similarly, impaired cardiovascular recovery from stress is associated with serious health problems such as hypertension and even cardiovascular death (Hocking et al., 2007; Kivimäki et al., 2006). Taken together, this suggests that the relationship between physiological responses to stress and health has an inverted "U" shape (Whittaker et al., 2021) whereby the 'steepness' of the upward slope represents cardiovascular reactivity (with greater reactivity representing a greater response or reaction to a stressor) and the downward slope representing how fast or slow the physiological recovery following the reactivity (with slower recovery representing a poorer physiological response).

The stress-buffering hypothesis proposes that positive emotions have the capacity to mitigate negative reactions to stress and thus protect individuals from the potential pathogenic effects of stressful events (Gellert et al., 2018; Pressman et al., 2019). Positive emotions dampen the autonomic nervous system and hypothalamic-pituitary-adrenal axis responses to stress (Okely et al., 2017) and facilitate the utilization of stress coping mechanisms such as the reappraisal of stressful situations (Pressman et al., 2019). In line with this, past research has demonstrated that positive affect was associated with faster physiological recovery from acute psychological stress (Ong & Allaire, 2005; Papousek et al., 2010). Regardless of which aspect of cardiovascular health is considered (reactivity or recovery), identifying the stress-buffering effects of positive emotions is important to reduce the overall burden of cardiovascular disease, which is already the leading cause of death globally (Mathers & Loncar, 2006).

With regard to affect, previous research has connected both positive and negative affect to health outcomes (Danhauer et al., 2013; Pressman et al., 2019). There is evidence that negative affect is associated with poorer cardiovascular health outcomes (DeSteno et al., 2013). In contrast, increased positive emotions have been associated with positive health outcomes (DuBois et al., 2012; Teoh & Hilmert, 2018) such as reduced sleep problems (Steptoe, 2008), positive overall health (Cohen, 2006), and reduced mortality risk (Zhang & Zhang, 2016). Positive affect is also

associated with reduced cardiovascular reactions to stress (Brummett et al., 2009), decreased hospital readmission following cardiovascular issues (Middleton & Byrd, 1996), and overall is thought to play an independent, protective role against cardiovascular disease (Boehm & Kubzansky, 2012). Hence, affect is thought to play a critical stress buffering role, both reducing reactions to stress and hastening recovery (Pressman et al., 2019).

Past research has been criticised for a sole focus on either positive or negative affec (Kolanowski et al., 2014). Rather, what is important is the balance of positive to negative emotions (Fredrickson, 2001). This concept is supported by findings that positive and negative affect are associated with separate outcomes (Russell & Carroll, 1999; Schlauch et al., 2013). Thus, rather than examining either positive or negative affect independently, this study examined the balance between them, or affect balance (Diener et al., 2010).

Affect balance has been conceptualised as a key component of well-being insofar as it measures the relative frequency of experiencing positive affect over negative affect (Kolanowski et al., 2014). It is also frequently attached to the "positive ratio" (Garland et al., 2010; Veilleux et al., 2020). When an individual experiences more positive than negative affect, they tend to experience better mental health (Diehl et al., 2011), well-being (Meeks et al., 2012), life satisfaction (Plessis & Guse, 2017). Affect balance has also been found to negatively predict stress (Veilleux et al., 2020). Furthermore, a specific body of work has demonstrated that greater experience of positive constructs such as social support (Phillips et al., 2009) and positive affect (Pressman & Cohen, 2005) are associated with lowered cardiovascular reactivity and improved recovery.

Gratitude is a specific positive emotion described as the recognition that something good has happened to an individual and which is perceived as costly, altruistic, or valuable (Wood et al., 2010). There is growing evidence that gratitude has a significant impact on individual health. Cross-sectional studies indicate that dispositional gratitude in adults is associated with positive self-reported physical health (O'Connell & Killeen-Byrt, 2018). This link is also seen in numerous studies concerning cardiovascular health (Celano et al., 2017; Jackowska et al., 2016). Importantly,

recent research has uncovered evidence regarding gratitude's stress-buffering effects, with state gratitude, as opposed to trait gratitude, being associated with decreased cardiovascular reactivity to acute psychological stress in a laboratory setting (Gallagher et al., 2020; Ginty et al., 2020).

These effects are theoretically consistent with the neurovisceral integration model, which charts the brain-heart link (Park et al., 2013; Thayer & Lane, 2009). From this perspective, gratitude may play a role in regulating central autonomic nervous system activity (Kyeong et al., 2017), with some studies having found an association between gratitude and increased parasympathetic heart rate variability and cardiac coherence (Rash et al., 2011; Redwine et al., 2016). Importantly, previous research indicates that state gratitude can be manipulated in individuals by simple interventions (Davis et al., 2016; Hussong et al. (2019)). As such, gratitude may constitute plausible low-cost health intervention (Wood et al., 2010), with a greater understanding of the stress- buffering effects of gratitude having the potential to improve individual and community health and well-being.

The effects of gratitude and affect balance are consistent with the cognitive model of stress which states that an individual's internal characteristics and resources influence how one copes and manages stress (Folkman, 2013). For example, individuals expressing higher levels of gratitude appear to more successfully cope with stress and adversity (Wood et al., 2010). For example, gratitude expressions are correlated with coping actions such as reappraisal (Bryan et al., 2018), as well as planning and goal-directed strategies which reduce the frequency and intensity of stress (Wood et al., 2007). Similarly, positive affect (Pressman et al., 2019), negative affect (Diehl & Hay, 2010), and the balance between them (Amai & Hojo, 2022) have been found to be associated with stress coping.

In past research investigating the impact of gratitude on cardiovascular reactivity, positive affect has typically been modelled as a control variable in order to establish the independent effect of gratitude on cardiovascular reactivity (Ginty et al., 2020). Ginty and colleagues (2020) found that including positive affect in the regression model did not change the significance of state gratitude as a predictor. This suggests that gratitude has an impact on the cardiovascular stress

response that is relatively independent of positive affect.

However, previous research has also suggested that positive affect may moderate the effects of gratitude on well-being (Rash et al., 2011). This is based on the resistance hypothesis which states that individuals who already experience a high amount of positive emotions are unlikely to be affected by additional positive experiences such as experiencing gratefulness (McCullough et al., 2004; Rash et al., 2011). For example, Froh and colleagues (2009) conducted a randomized-control study assessing the impact of a gratitude letter on positive and negative affect. They found that pre-test positive affect moderated the effect of the intervention on post-test affect whereby individuals lower in positive affect benefitted more from the intervention. As such, recent research has called for greater investigation into how affect might moderate the effects of gratitude on positive outcomes (Klibert et al., 2019). Based on this idea, it is posited that gratitude and affect interact in buffering the effects of acute stress on cardiovascular reactivity and cardiovascular recovery. It is expected that individuals with poorer affect balance (i.e., those who experience more negative than positive affect on balance) will benefit more from experiencing state gratitude.

In sum, exaggerated cardiovascular stress reactions and delayed cardiovascular recovery are associated with poorer health outcomes. Positive psychological constructs such as gratitude and affect balance appear to have an important cardiovascular stress-buffering effect, including reducing cardiovascular stress reactions and hastening cardiovascular stress recovery. Previous research has uncovered inverse relationships between gratitude and cardiovascular reactions (Cousin et al., 2021). However, previous research has not examined gratitude's effect on cardiovascular recovery. As such, the interactive relationship between gratitude, affect balance, and cardiovascular reactivity is unclear. Therefore, this paper has the following aims: (1) to assess the impact of state gratitude and affect balance on the cardiovascular response to stress, including both reactivity and recovery, and (2) to assess whether affect-balance moderates the relationship between state gratitude and the cardiovascular response to stress.

The cardiovascular stress response has an inverted u-shape, whereby when an individual experiences a stressful situation, they experience a corresponding cardiovascular reaction (e.g.

increase in heart rate or blood pressure) which is typically termed 'reactivity'. Once the stressor has abated, the individual's cardiovascular response slowly returns to normal but how quickly this occurs varies across individuals. This is termed recovery and, as such, a faster recovery phase (e.g. lowering of heart rate) is better for one's health. Overall, lower responses are considered healthier than higher responses. Thus, using multilevel growth curve models is a useful way of modelling the relation- ship between state gratitude, affect balance and the cardiovascular response to acute stress (Curran et al., 2010). Multilevel growth curve models allow for the indexing of reactivity and recovery through the analysis of the shape and significance of time course patterns for cardiovascular measures (Hoogerwerf et al., 2018; Woody et al., 2018). This is advantageous over traditional methods of analysing repeated measures data as it permits researchers to examine various patterns of change (e.g. linear, quadratic) and allows for within-person variability (Lehman et al., 2015). In the context of this paper, it is expected that state gratitude and affect balance would predict lower responses throughout the laboratory-testing period (Woody et al., 2018). To our knowledge, this is the first paper to assess the impact of state gratitude and affect balance on the cardiovascular stress response using this method.

Accordingly, this paper has the following hypotheses:

H1: State gratitude will predict lower overall cardiovascular responses to acute psychological stress.

H2: Affect balance will predict lower overall cardiovascular responses to acute psychological stress.

H3: Affect-balance will moderate the relationship between state gratitude and the cardiovascular response to stress, with individuals with lower scores benefitting more from the effects of state gratitude.

6.3 Method

6.3.1 Design

This study used a correlational design. The experimental design induced stress and measured individuals' cardiovascular reactivity and recovery in response to this. The predictor variables (state gratitude and affect balance) were assessed in the lab via a paper-and-pencil survey prior to the stress task. This study focused on state gratitude as previous research has found it to play an important role in stress buffering (Gallagher et al., 2020). Affect can be conceptualised as either a state or a trait depending on the temporal phrasing of survey items (Merz & Roesch, 2011) and state gratitude experienced over the previous 24 hours was assessed in this study. Affect balance was measured 'over the past week' and as such reflects the general affective state individuals experienced in the week prior to their participation in the study. This ensured that affect was captured as opposed to more momentary emotions. The dependent variables were cardiovascular reactivity and cardiovascular recovery to the induced stress.

6.3.2 Participants

A convenience sample of 68 undergraduate students studying psychology in an Irish university volunteered to take part. Of these, 24 were male and 44 were female. The ages ranged from 18 to 57 (M = 22.87, SD = 8.07). The inclusion criteria included individuals who were 18 years old or older and able to consent. Participants were excluded if they (1) consumed alcohol in the twelve hours before the study took place, (2) engaged in vigorous exercise in the twelve hours before the study took place, (3) consumed caffeine or smoked cigarettes less than two hours before the study took place, (4) consumed food one hour before the study, (5) were pregnant, or (6) currently held a diagnosis of cardiovascular disease. These precautions were to control for confounding variables and are in line with previous research (Creaven & Hughes, 2012; Gallagher et al., 2020). Three participants were excluded for violating the exclusion criteria; having eaten less than an hour before the study commenced. This left a sample of 65 individuals.

Studies that utilize repeated measures, as the current study does, usually have higher

power than comparable between-subjects studies (Murphy et al., 2014). A priori power analyses were conducted using *G*Power* (Faul et al., 2007). F-test was selected and R^2 increase examined for effect size estimation. Based on studies of the effects of positive emotion on the physiological response and mood response to stress, it was estimated that a medium effect size was justified (Fredrickson et al., 2000; Monfort et al., 2015). An alpha level of .05, a power of .80 and medium effect sizes of .15 for two tested predictors and a total of seven predictors overall was set. The suggested sample size was 68. Additionally, as the analysis was run in long format, this yielded a total of 189 observation. This suggests that the study attained sufficient power.

6.3.3 Measures and Materials

Stress task The study used an adaptation of the serial subtraction task whereby participants were asked to subtract in increments of thirteen from 1,222 (al'Absi et al., 1995). If participants made an error, or forgot their place, they were asked to begin the task again. Mental arithmetic tasks, such as the one used in this study, are commonly used to elicit a physiological stress response in the lab (Mathias et al., 2017; Whittaker et al., 2021).

Lab setting The laboratory added pressure on each participant by: (1) including a fake leader board directly opposite the participant, (2) using a small lab with two researchers present, and (3) switching off the main laboratory light during the task, leaving the room illuminated by a lamp in front of the participant.

Cardiovascular Assessment Systolic blood pressure, diastolic blood pressure and heart rate in beats per minute were measured using a GE Dinamap Pro 400 V2 vital signs monitor (GE Medical Systems, Freiburg, Germany. A stopwatch was used to ensure that measurements were taken at consistent times. A standard blood pressure cuff was placed over the brachial artery on the participant's non-dominant arm. After acclimatization, four baseline measurements were taken over a ten-minute period. Three measurements were taken during the six-minute stress task. Four

measurements were taken immediately after the stress task in the ten-minute recovery period.

State gratitude was assessed using the Gratitude adjective checklist (GAC; McCullough, 2002). This scale has demonstrated high internal reliability (a = .83) in prior research (Froh et al., 2010) and this was confirmed in the current study (a =.86). The scale is the sum of three adjectives: grateful, thankful, and appreciative, assessed using a 5-point Likert scales (1 = not at all to 5 = extremely) with higher scores reflecting higher levels of state gratitude. The stem of this scale assessed gratitude over the past day. However, the measure was completed in the same lab session as the stress test. The scale was summed in line with previous research and scores ranged from 3 to 15 (Gallagher et al., 2020)

Affect balance was assessed using the Positive and Negative Affect Schedule Short Form (PANAS-SF; Watson et al., 1988). This is a 20-item scale where participants rate the extent to which they have felt certain emotions on a 5-point scale from very slightly/not at all (1) to extremely (5) over the past week. The measure was completed in the same lab session as the stress test. The scale demonstrated acceptable reliability for both positive affect (a = .82) and negative affect (a = .82). Affect balance was computed by subtracting the negative affect score from the positive affect score (Veilleux et al., 2020).

Stress task measures were immediately taken after the completion of the stress task with a three-item scale (a = .83). Participants were asked to rate how stressful they found the task on 7-point Likert scale 0 (Not at all) to 6 (Extremely).

6.3.4 Procedure

Prior to attending, participants were instructed to refrain from drinking alcohol or exercising in the 12 hours prior to the study and to refrain from smoking and consuming caffeine two hours before, or eating one hour before the study took place. Upon arrival, the acclimatization period began at the laboratory. During this time, participants were greeted by the primary experimenter and instructed to read study information sheets to confirm eligibility and provide consent. The

researcher recorded the participant's height and weight measurements for the computation of BMI. They were then seated and the blood pressure cuff was placed on their upper, non-dominant arm, and they were instructed to place their feet in a box under the table in order to control for movement during the study (Pickering et al., 2005). During this period, acclimatization measures were taken, demographic details were recorded, and psychometric scales were completed. The acclimatization period lasted 20 minutes. The participant was then asked to refrain from speaking for the rest of the experiment. This was followed by a formal ten-minute baseline period where measures were taken every two minutes.

Once the formal baseline was completed, the researcher turned off the lights, turned on a spotlight and explained to the participant that they would be completing a serial sub- traction task. The task lasted for six minutes. Blood pressure measurements were taken throughout at two-minute intervals, yielding three measurement points. Prompts to continue were delivered by the researcher if the participant stopped engaging. After the task, the lights were switched back on and participants were asked to rate how stressful they found the task. This was immediately followed by an eight-minute recovery period where participants sat quietly and had a blood pressure measurements taken every two minutes, yielding four measurement points. They then filled out a self-report stress and motivation questionnaire, and were debriefed and thanked for their time after study completion.

Ethical approval was received from The Biomedical and Life Sciences Research Ethics Sub-Committee (BSRESC-2018-014).

6.3.5 Data reduction and data analysis

R (Version 4.2.0; R Core Team, 2022) was used for data analysis. The packages *lme4* (*Version 1.1.30;* Bates et al., 2015) and *lmerTest* (*Version 3.1.3;* Kuznetsova et al., 2017) were used to fit mixed-effects growth models. Four resting baseline measures for each of the cardiovascular parameters were averaged to yield baseline values for each participant. The same was done with the three stress task measures and the four recovery measures, in line with similar

approaches taken in previous research (Phillips et al., 2009). Data were screened and checked for normality using the visual inspection of histograms, QQ-plots, and by utilizing the Shapiro test for normality. Outliers were assessed using z-scores with a z score >= |3| constituting an outlier. One outlier was removed as a result. Repeated measures ANOVAs were used to confirm that the stress task increased cardiovascular responses from baseline, and confirmatory factor analysis was used to confirm that state gratitude, positive affect, and negative affect were best modelled as three distinct constructs. Correlations between study variables were examined prior to moving to hypothesis testing.

A series of mixed-effects growth curve models were used to test the hypotheses and assess the impact of state gratitude and affect balance on the cardiovascular stress response. Growth curve models can help gain insight into the reactivity and recovery periods by the specification of non-linear growth parameters and their interactions with other predictors (Bolger & Laurenceau (2013); Felt et al., 2017). Mixed-effect growth curves are advantageous as they allow a more parsimonious test of the effects of state gratitude and affect balance over the whole testing session (i.e., baseline, stress task, and recovery) and estimate the effects of the predictor variables on the trajectory of the changes over time (Verkuil et al., 2014). They also more elegantly control for baseline cardiovascular differences by allowing varying intercepts at baseline (by coding mean baseline measures as time 0 and varying slopes for individual participants; Gueorguieva & Krystal (2004)). Mixed-effects models have also been shown to perform well with smaller sample (McNeish (2017)), particularly by making use of restricted maximum likelihood estimation (REML; Snijders, 2005). As such, all models will be fit using REML estimation.

As it was likely that some non-linearity would need to be incorporated into the models, firstly it was formally tested whether a linear or quadratic growth fitted best, using a normal chi-square distribution.

The data were modelled at two levels, with the most detailed level comprising level one (Snijders & Bosker, 2011). As such, the measurement occasion (time) was modelled at level one, which thus represented the within-person, random effects. The intercept was allowed to vary in

order to account for different baseline cardiovascular measures. Measurement occasions were nested within people, and so level two represented the between-person, fixed effects or growth parameters. The primary predictor variables were state gratitude and affect balance. The control variables were task stress ratings, age and sex. Both the predictor variables and control variables were person-level variables and so, only possible to model at level 2. To test the hypotheses, the interaction between affect balance, state gratitude and the growth parameters were of main interest (i.e. Systolic blood pressure = Growth ◊ State gratitude + Growth} ◊ affect_balance). For interactions, state gratitude and affect balance scores were standardised as z-scores as recommended by Bauer & Curran (2005). Models were fitted hierarchically, with the growth parameters and control variables entered into the model first, then the cross-level interactions between state gratitude and time and affect balance and time second, and finally adding the interaction between state gratitude, affect balance and time in step 3.

Restricted maximum likelihood estimation were used to analyse the main effects and moderation analyses. Overall model fit is evaluated using the Akaike information criterion on a smaller-is-better basis and the Loglikelihood on a bigger-is-better basis (Woody et al., 2018. Model comparison was also formally tested using likelihood ratio tests for nested models which make use of an ordinary ‰^{2} distribution, with significant differences indicating that the full model fits better than the nested model (Bliese, 2022). A marginal coefficient of determination R^2 was used to compute an effect size, flR^2. This quantifies the variance explained by the fixed effects of the models. An alpha level of 0.05 was selected.

6.4 Results

6.4.1 Preliminary analyses

Descriptive statistics and correlations between the study variables are outlined in tables 6.1 and 6.2. State gratitude had significant correlations with affect balance and systolic blood pressure during the stress task; it did not correlate with other cardiovascular measures. Affect balance did not correlate with any cardiovascular measures. A series of repeated measures ANOVAs were used to check if the stress task increased cardiovascular responses from baseline, to stress, and decrease to recovery (see Figure\ref{fig:study2fig1), for systolic blood pressure $F(2, 128) = 134.05$, $p < .001$, $\eta_p^2 = .67$, diastolic blood pressure $F(2, 128) = 59.94$, $p < .001$, $\eta_p^2 = .48$, and heart rate $F(2, 128) = 81.21$, $p < .001$, $\eta_p^2 = .56$ with results indicating statistically significant changes from baseline, stress, and recovery epochs. The intraclass correlation coefficients (ICC1) for systolic blood pressure, diastolic blood pressure and heart rate were .61, .65, and .67 respectively. This indicates that 61% of the variation in systolic blood pressure was at level 2 (person level), 65% for diastolic blood pressure, and 67% for heart rate.

Table 6.1: Descriptive statistics of all variables

Name	Mean	SD	Min	Max
Age	22.79	8.23	18	57
Affect balance	13.11	.4	-17	30
Gratitude score	10.98	2.57	3	15
Task stress rating	2.79	0.89	0.25	4.25
Baseline SBP	117.44	0.09	98.5	142.5
Stress task SBP	126.58	12.11	103	148.67
Recovery SBP	115.09	9.68	97.25	132.5
Baseline DBP	65.46	7.6	53.75	83
Stress task DBP	71.84	9.58	55.67	94.67
Recovery DBP	65.05	8.25	54.5	87
Baseline HR	80.37	.3.07	53.5	120
Stress task HR	89.03	.4.51	57.33	131
Recovery HR	77.38	.2.53	51.5	112

Note. SBP = systolic blood pressure (mmHG), DBP = dias- tolic blood pressure (mmHG), HR = heart rate (BPM), stress task = average measurement during the study's stress task.

Table 6.2: Correlations between study variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Age															
2. Gender	-0.09														
3. Positive affect	0.02	.37**													
4. Negative affect	-0.19	0.1	-0.1												
5. Affect balance	0.13	-.33**	.77**	-.71**											
6. State gratitude	-0.18	0.07	.39**	-0.11	.35**										
7. Stress task rating	-0.19	.33**	-.26*	.35**	-.41**	-0.24									
8. Baseline SBP	0.18	-0.24	0.1	-0.06	0.11	-0.23	-0.09								
9. Stress task SBP	.29*	-.33**	0	-0.09	0.05	-.38**	-0.19	.81**							
10. Recovery SBP	0.19	-.28*	0.03	-0.08	0.07	-.28*	-0.12	.91**	.83**						
11. Baseline DBP	.35**	0.19	-.26*	-0.06	-0.15	-0.2	-0.03	.63**	.56**	.64**					
12. Stress task DBP	.38**	-0.04	-0.18	-0.11	-0.06	-0.19	-0.03	.53**	.60**	.59**	.75**				
13. Recovery DBP	.31*	0.16	-.26*	-0.06	-0.15	-0.2	-0.09	.53**	.46**	.60**	.87**	.79**			
14. Baseline HR	-0.19	.42**	-0.16	-0.05	-0.08	0.09	.25*	0.03	-0.2	-0.06	0.16	-0.02	0.09		
15. Stress task HR	-0.02	.38**	-0.17	-0.09	-0.06	0.01	.31*	-0.01	-0.04	-0.05	0.19	0.05	0.01	.80**	
16. Recovery HR	-0.15	.40**	-0.18	-0.01	-0.12	0.11	.26*	0	-0.21	-0.05	0.17	0.02	0.12	.95**	.77**

Note. SBP = systolic blood pressure (mmHG), DBP = diastolic blood pressure (mmHG), HR = heart rate (BPM), stress task = average measurement during the study's stress task. * indicates $p < .05$. ** indicates $p < .01$.

To ensure that gratitude, positive affect, and negative affect were best captured as three distinct constructs, three confirmatory factor analyses (CFAs) were carried out (see Table 6.3). The first CFA loaded all the items onto a single factor. The second loaded gratitude and positive affect onto one factor and negative affect onto a second factor. The third loaded gratitude, positive affect, and negative affect onto three distinct factors. As both AIC and BIC are lowest for the three-factor model, this model was accepted (see table 6.3).

Table 6.3: Confirmatory factor analysis model fit statistics of state gratitude, positive affect and negative affect.

	‰^{2}	df	p	CFI	TLI	RMSEA (90% CI)	SRMR	AIC	BIC	ssBIC
One-factor model	621.03	30	<.001	0.318	0.25	.162 (.146 - .177)	0.181	4080.393	4230.426	4013.231
Two-factor model	463.85	29	<.001	0.59	0.548	. 126 (.109- .142)	0.131	3925.212	4077.419	3857.077
Three-factor model	389.89	27	<.001	0.716	0.683	.105 (.087 - .123)	0.121	3855.246	4011.802	3785.164

Note. Estimator = MLR; ‰^{2} = Chi-square Goodness of Fit statistic; df = degrees of freedom; p = Statistical significance; CFI = Compara- tive Fit Index; TLI = Tucker Lewis Index; RMSEA (90% CI) = Root-Mean-Square Error of Approximation with 90% confidence intervals; SRMR = Standardized Root-Mean Square Residual; AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion; ssaBIC = sample size adjusted BIC. One-factor model = gratitude adjective checklist, positive and negative affect on one factor. Two-factor model = gratitude adjective checklist and positive affect on one factor and negative affect on a second factor. Three-factor model = gratitude adjective checklist, positive affect and negative affect loaded onto three separate factors.

6.4.2 Assessing the pattern of growth

The test if a non-linear growth parameter would fit the data better, linear and quadratic growth parameters were fitted to model systolic blood pressure, diastolic blood pressure and heart rate over time. The addition of the quadratic growth pattern yielded significant improvements to model fit for systolic blood pressure (‰^{2}(1) = 140.43, $p < 0.001$), diastolic blood pressure (‰^{2}(1) = 88.08, $p < 0.001$) and heart rate (‰^{2}(1) = 97.36, $p < 0.001$). As such, the quadratic growth parameter was retained for the rest of the analysis. All models were fitted with random intercepts

and linear random slopes; linear random slopes were fitted in order to circumvent issues of singularity and create more parsimonious models (Bates et al., 2015; Matuschek et al., 2017). Additionally, for all models, the addition of autocorrelated error terms did not improve model fit and as such were not retained in the final model.

6.4.3 Hypothesis testing

To test the hypotheses, multilevel growth models were fit to the data in a procedure similar to hierarchical regressions. For step one, the model with the growth parameters and the control variables were entered into the model. For step two, the cross-level interactions between state gratitude and growth, and affect balance and growth were entered into the model. Finally, in the third step, the interaction between affect balance, state gratitude and growth was entered into the model.

Hypothesis 1 proposed that state gratitude would predict lower overall cardiovascular responses to acute psychological stress. The cardiovascular response was tested via the growth curves of diastolic blood pressure, systolic blood pressure and heart rate. The relevant results for this can be found in step 2 of Tables 6.4, 6.5, and 6.6, and Figure 6.1 State gratitude significantly interacted with linear growth (B =-1.57, 95%CI [-2.66, -0.49], p = 0.005), and quadratic growth (B = - 0.74, 95%CI [0.21, 1.26], p = 0.007) to predict systolic blood pressure. Visual inspection of the interaction plot revealed that higher levels of state gratitude were associated with a lower systolic blood pressure response to stress. Adding this interaction to the model decreased the AIC and increased the LL fit indices and resulted in a 6% increase in the variance explained (ΔR^2 =0.06, $\chi^2(6)$ = 21.62, p = 0.001). State gratitude did not interact with linear growth to predict either diastolic blood pressure (B = 1.73, 95%CI [-0.90, 4.35], p = 0.195), or heart rate (B = -2.80, 95%CI [-6.63, 1.03], p = 0.151) stress responses. Similarly, state gratitude did not interact with quadratic growth to predict either diastolic blood pressure (B = 0.67, 95%CI [-0.60, 1.95], p = 0.299), or heart rate (B = 1.45, 95%CI [-0.39, 2.39], p = 0.123) responses. Taken together, partial

support for hypothesis 1 was found as higher state gratitude predicted a lower systolic blood pressure response to stress, but did not predict diastolic blood pressure or heart rate responses.

Hypothesis 2 proposed that affect balance would predict lower overall cardiovascular responses to acute psychological stress. The relevant results for this can be found in step 2 of tables 6.4, 6.5 and 6.6 and Figure 6.2. Across the three models, there was no evidence that affect balance moderated the growth trajectory of the cardiovascular response to stress. Thus, hypothesis 2 was not supported.

Hypothesis 3 proposed that affect balance and state gratitude would interact with growth to predict the cardiovascular stress response. The relevant results for this can be found in step 3 of tables 6.4, 6.5 and 6.6 and Figure 6.3. There was a significant three-way interaction for diastolic blood pressure for linear (B = -2.46, 95%CI [-4.81, -0.10], p = 0.041), and quadratic growth (B = 1.31, 95%CI [0.18, 2.45], p = 0.023). Adding this interaction to the model decreased the AIC and increased the LL fit indices and resulted in a 7% increase in the variance explained ($fl R^2$ = 0.07, $‰^2(1)$ = 14.86, p = 0.002). Inspection of the interaction plots (see Figure 6.3B) implies that the more an individual experienced positive emotion over negative emotion, the stronger the effect of state gratitude on the diastolic blood pressure response to stress was. The three way interaction was not significant for systolic blood pressure, although inspection of the interaction plot in Figure

6.3A demonstrates a similar (albeit non-significant) pattern of interaction whereby more positive emotion on balance amplified the effects of state gratitude.

The three way interaction with quadratic growth for heart rate was marginally significant (B = 1.70, 95%CI [0.05, 3.34], p = 0.043), but the change in R^2 was less than 0.1% so this finding was interpreted as non-significant. Thus, there was some support for hypothesis 3 with regard to diastolic blood pressure but not for systolic blood pressure or heart rate.

Table 6.4: Multi-level growth model results looking at the cross-level inter- actions between state gratitude, affect balance and growth in systolic blood pressure

Step	1				2				3			
	Est	95% CI	t	p	Est	95% CI	t	p	Est	95% CI	t	p
Fixed Effects												
Intercept	121.58	[108.06, 135.10]	17.8	<.001**	23.57	[109.78, 137.35]	17.75	<.001**	26.51	[111.61, 141.41]	16.82	<.001**
Age	0.26	[-0.05, 0.56]	1.68	0.098	0.17	[-0.13, 0.47]	1.11	0.271	0.12	[-0.19, 0.44]	0.79	0.435
Gender	-5.81	[-11.28, -0.33]	-2.12	.038*	4.19	[-9.80, 1.42]	-1.5	0.14	-3.97	[-9.59, 1.65]	-1.41	0.163
Perceived Stress	-0.11	[-3.08, 2.85]	-0.08	0.939	-1.04	[-4.12, 2.03]	-0.68	0.499	-1.74	[-5.07, 1.60]	-1.04	0.301
Linear growth	19.46	[16.80, 22.12]	14.48	<.001**	9.56	[16.94, 22.17]	14.82	<.001**	19.77	[16.99, 22.55]	14.09	<.001**
Quadratic growth	-10.32	[-11.59, -9.04	-15.98	<.001**	-10.36	[-11.62, -9.11]	-16.34	<.001**	-10.42	[-11.76, -9.09]	-15.46	<.001**
Affect balance					0.82	[-2.18, 3.83]	0.55	0.587	0.63	[-2.40, 3.66]	0.42	0.678
State Gratitude					2.58	[-5.47, 0.31]	-1.79	0.079	-2.9	[-5.88, 0.07]	-1.95	0.056
Affect balance × Linear growth					0.63	[-2.19, 3.45]	0.44	0.659	0.58	[-2.26, 3.42]	0.4	0.687
Affect balance × Quadratic growth					0.37	[-1.73, 0.98]	-0.55	0.584	-0.36	[-1.73, 1.00]	-0.52	0.601
State gratitude × Linear growth					4.08	[-6.93, -1.24]	-2.84	.005**	-4.18	[-7.07, -1.29]	-2.86	005**
State gratitude × Quadratic growth					.91	[0.54, 3.28]	2.76	.007**	1.94	[0.55, 3.33]	2.76	.007**
Affect balance × State gratitude									-1.01	[-3.69, 1.67]	-0.76	0.452
Affect balance × State gratitude × Linear growth									-0.62	[-3.20, 1.96]	-0.48	0.634
Affect balance × State gratitude × Quadratic growth									0.17	[-1.07, 1.41]	0.27	0.788
Random effects												
Intercept (σ^2)	9.33				9.01				8.99			

Time (σ^2)	0.03	.04	0.04
Residual (σ^2)	4.18	4.11	4.13
Model fit indices			
AIC	1259.36	1249.75	1250.93
LL	-619.68	-608.87	-606.46
$\chi^2(df)$		21.61(6); p <.001**	4.82(3); p =0.185
R^2 (ΔR^2)	0.27	0.33 (0.06)	0.33 (0.00)

Note. AIC = Akaike information criterion, LL = log likelihood, R^2 = marginal coefficient of determination representing variance explained by fixed effects, σ^2= standard deviation, * = p < .05, ** = p < .01.

Table 6.5: Multi-level growth model results looking at the cross-level inter- actions between state gratitude, affect balance and growth in diastolic blood pressure.

Step	1				2				3			
	Est	95% CI	t	p	Est	95% CI	t	p	Est	95% CI	t	p
Fixed effects												
Intercept	52.6	[42.26, 62.94]	10.07	< .001**	55.22	[44.38, 66.06]	10.09	< .001**	61.05	[49.95, 72.15]	0.89	< .001**
Age	0.39	[0.16, 0.62]	3.33	.001**	0.36	[0.12, 0.60]	3.03	.004**	0.27	[0.04, 0.51]	2.33	.023*
Gender	2.65	[-1.53, 6.83]	1.27	0.21	2.77	[-1.63, 7.18]	1.26	0.213	3.2	[-0.98, 7.39]	1.53	0.13
Perceived Stress	-0.15	[-2.41, 2.12]	-0.13	0.896	-0.92	[-3.33, 1.50]	-0.76	0.45	-2.25	[-4.74, 0.23]	-1.82	0.074
Linear growth	12.95	[10.53, 15.37]	10.59	< .001**	12.99	[10.55, 15.42]	10.56	< .001**	13.85	[11.31, 16.38]	10.81	< .001**
Quadratic growth	-6.58	[-7.74, -5.42]	-11.2	< .001**	-6.59	[-7.76, -5.42]	-11.16	< .001**	-7.05	[-8.27, -5.83]	11.46	< .001**
Affect balance					-1.08	[-3.48, 1.33]	0.9	0.374	-1.47	[-3.78, 0.83]	-1.28	0.206
State Gratitude					-0.9	[-3.21, 1.42]	-0.78	0.441	.59	[-3.86, 0.68]	-1.41	0.165
Affect balance × Linear growth					1.73	[-0.90, 4.35]	1.3	0.195	.53	[-1.06, 4.12]	1.17	0.245
Affect balance × Quadratic growth					-0.86	[-2.12, 0.41]	-1.34	0.182	0.75	[-2.00, 0.50]	1.19	0.236
State gratitude × Linear growth					-1.59	[-4.24, 1.07]	-1.18	0.239	-1.97	[-4.61, 0.67]	1.48	0.141
State gratitude × Quadratic growth					0.67	[-0.60, 1.95]	1.04	0.299	.88	[-0.39, 2.15]	1.37	0.173
Affect balance × State gratitude									-2.35	[-4.39, -0.31]	-2.31	.025*
Affect balance × State gratitude × Linear growth									-2.46	[-4.81, -0.10]	-2.07	.041*
Affect balance × State gratitude × Quadratic growth									1.31	[0.18, 2.45]	2.3	.023*
Random effects												

Intercept (σ^2)	7.02	6.97	6.54
Time (σ^2)	0.15	0.06	0.16
Residual (σ^2)	3.81	3.83	3.77
Model fit indices			
AIC	1203.47	1204.45	1195.58
LL	-591.74	-586.22	-578.79
$\chi^2(df)$		11.02 (6); p =0.088	14.86 (3); p =0.002**
R^2 (σR^2)	0.24	0.27 (0.03)	0.34 (0.07)

Note. AIC = Akaike information criterion, LL = log likelihood, R^2 = marginal coefficient of determination representing variance explained by fixed effects, σ^2= standard deviation, * = p < .05, ** = p < .01.

Table 6.6: Multi-level growth model results looking at the cross-level interactions between state gratitude, affect balance and growth in heart rate.

Step	1				2				3			
	Est	95% CI	t	p	Est	95% CI	t	p	Est	95% CI	t	p
Fixed effects												
Intercept	58.59	[42.53, 74.65]	7.22	< .001**	5.61	[38.48, 72.74]	.43	< .001**	6.16	[37.46, 74.86]	5.95	< .001**
Age	-0.06	[-0.42, 0.31]	-0.32	0.753	-0.06	[-0.43, 0.32]	-0.3	0.765	-0.07	[-0.46, 0.33]	-0.34	0.735
Gender	9.83	[3.34, 16.33]	3.03	.004**	10.44	[3.47, 17.41]	3	.004**	10.49	[3.44, 17.54]	2.98	.004**
Perceived Stress	2.4	[-1.12, 5.92]	1.36	0.178	3.09	[-0.73, 6.91]	1.62	0.11	2.93	[-1.26, 7.11]	1.4	0.167
Linear growth	18.82	[15.32, 22.32]	10.64	< .001**	18.88	[15.37, 22.40]	10.63	< .001**	20.04	[16.36, 23.73]	10.77	< .001**
Quadratic growth	-10.16	[-11.84, -8.47]	-11.96	< .001**	-10.19	[-11.88, -8.50]	-11.94	< .001**	-10.78	[-12.55, -9.01]	-12.06	< .001**
Affect balance					1.54	[-2.22, 5.31]	0.82	0.415	1.53	[-2.29, 5.36]	0.8	0.426
State Gratitude					0.74	[-2.88, 4.36]	0.41	0.685	0.72	[-3.03, 4.48]	0.39	0.701
Affect balance × Linear growth					1.53	[-2.27, 5.32]	0.8	0.427	1.26	[-2.51, 5.02]	0.66	0.51
Affect balance × Quadratic growth					-0.9	[-2.72, 0.92]	-0.98	0.33	-0.76	[-2.57, 1.05]	-0.83	0.406
State gratitude × Linear growth					-2.8	[-6.63, 1.03]	-1.45	0.151	-3.32	[-7.16, 0.51]	-1.72	0.089
State gratitude × Quadratic growth					1.45	[-0.39, 3.29]	1.56	0.123	1.71	[-0.13, 3.56]	1.84	0.068
Affect balance × State gratitude									0.17	[-3.21, 3.55]	0.1	0.92
Affect balance × State gratitude × Linear growth									-3.32	[-6.75, 0.10]	-1.92	0.057
Affect balance × State gratitude × Quadratic growth									1.7	[0.05, 3.34]	2.05	.043*

Random effects			
Intercept (σ^2)	11.02	1.11	11.22
Time (σ^2)	0.04	0.04	0.04
Residual (σ^2)	5.5	5.53	5.49
Model fit indices			
AIC	1346.82	1344.84	1342.01
LL	-663.41	-656.42	-652.01
$\chi^2(df)$		13.98(6); p =0.030*	8.82(3); p =0.032*
R^2 (ΔR^2)	0.27	0.28 (0.01)	0.28 (0.00)

Note. AIC = Akaike information criterion, LL = log likelihood, R^2 = marginal coefficient of determination representing variance explained by fixed effects, σ^2= standard deviation, * = p < .05, ** = p < .01.

Figure 6.1: Interaction plots for low (-1SD), average (mean) and high (+1SD) levels of state gratitude and how the trajectory of growth and de- cline changes at each level for cardiovascular measures

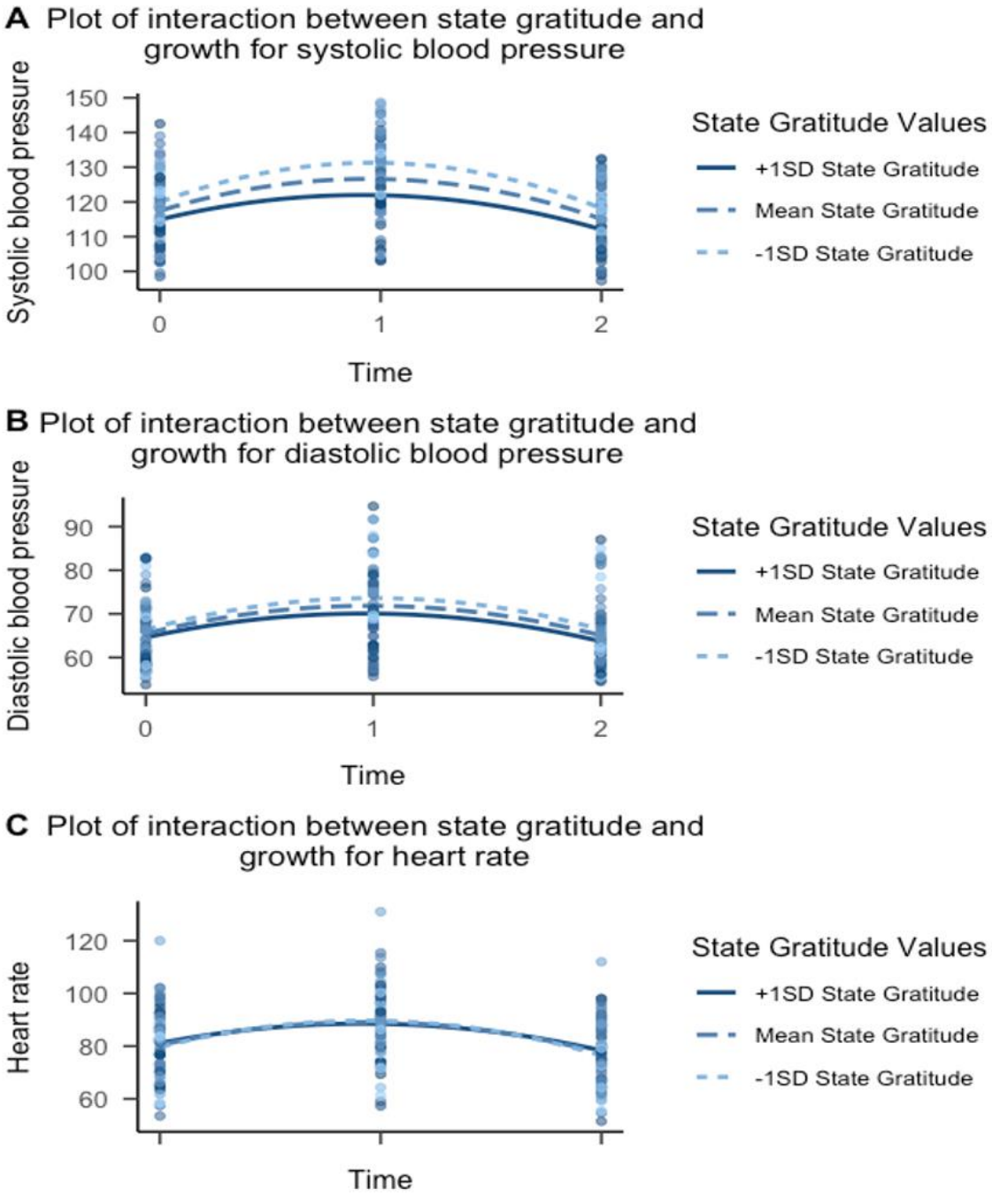


Figure 6.2: Interaction plots for low (-1SD), average (mean) and high (+1SD) levels of affect-balance and how the trajectory of growth and de- cline changes at each level for cardiovascular measures

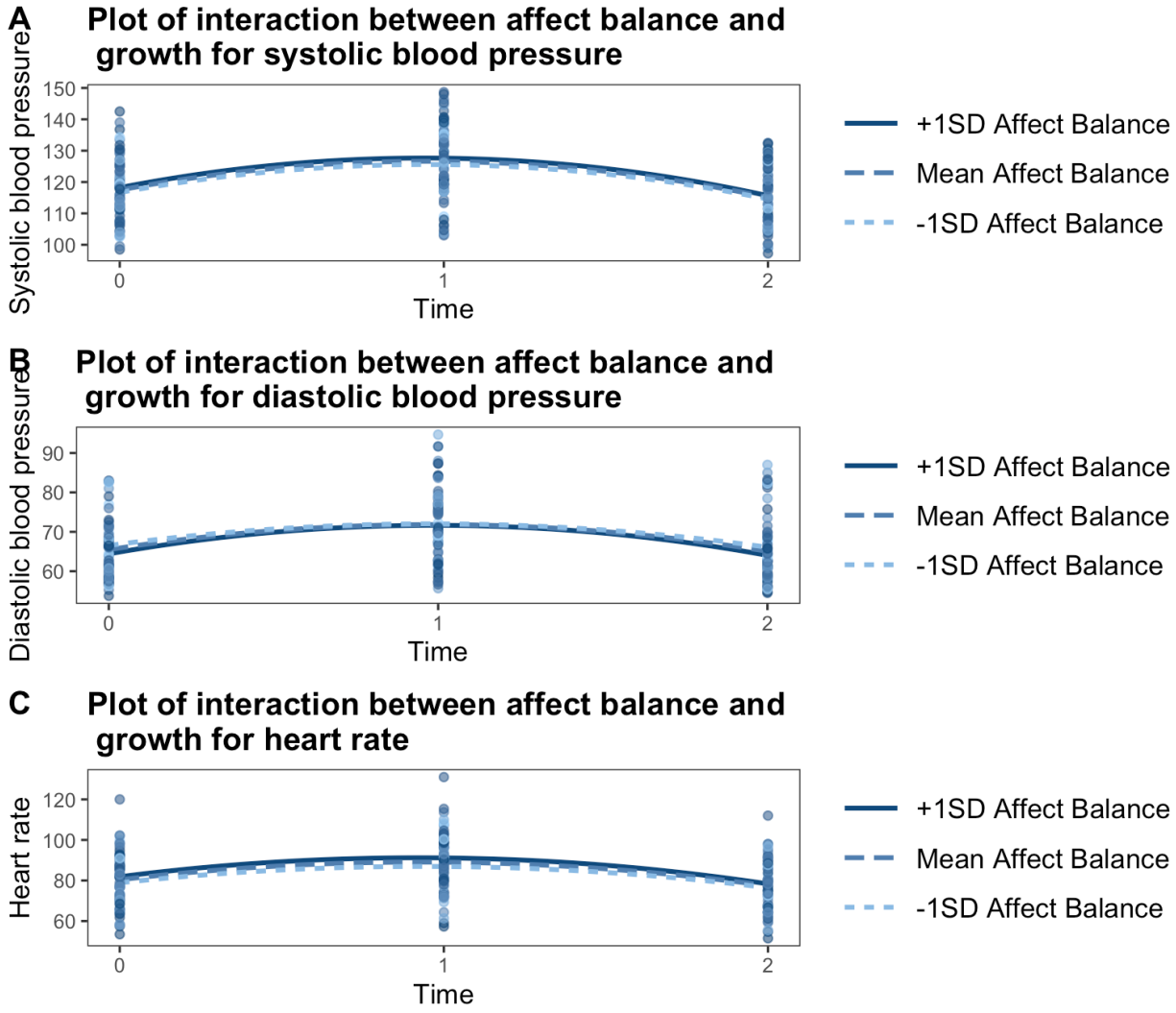
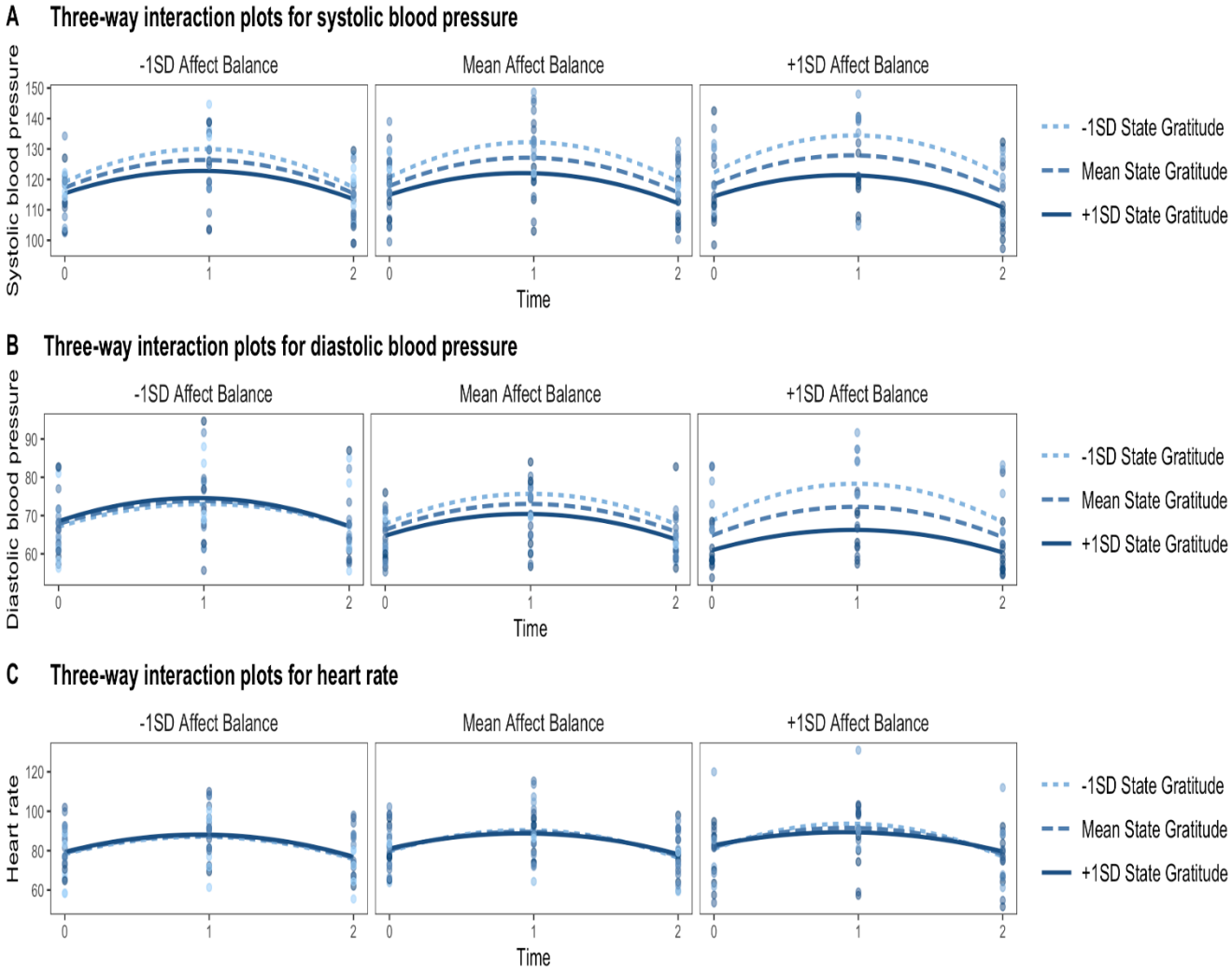


Figure 6.3: Interaction plots for low (-1SD), average (mean) and high (+1SD) levels of affect balance and state gratitude and how and how the trajectory of growth and decline changes at each level for cardiovascular measures.



6.5 Discussion

This study aimed to assess the stress buffering functions of state gratitude and affect balance. It found that state gratitude buffered the impact of induced stress on systolic blood pressure, indicated by a 'flatter' curve in systolic blood pressure (lower reactivity and faster recovery) at higher levels of state gratitude compared to moderate and low levels. This study also uncovered evidence of a moderating effect of affect balance on state gratitude's effect on diastolic blood pressure, where the more positive emotion an individual experienced, the greater the effect of higher levels of state gratitude on the diastolic blood pressure response to stress.

However, the study did not find evidence for affect balance's role as a moderator of the relationship between gratitude and the trajectory of systolic blood pressure or heart rate responses to acute stress. Similarly, there was no evidence of a direct buffering effect of affect balance on any cardiovascular outcome.

Overall, this study provides some evidence that state gratitude plays a unique stress-buffering role on systolic blood pressure during an acute stress response, where the response consists of both the reaction to and recovery from the stressor. Moreover, it found that this same response for diastolic blood pressure was moderated by the balance of positive to negative emotion, where more positive than negative emotion amplifies the stress-buffering effects of state gratitude.

State gratitude was found to have a significant interaction with the linear and quadratic growth parameters for systolic blood pressure. This means that individuals higher on state gratitude had lower responses to stress and had lower systolic blood pressure during the recovery period compared to those who reported moderate and low levels of state gratitude. It is worth noting that to our knowledge, no other study has examined how gratitude impacts cardiovascular recovery. While previous research has noted the effects of state gratitude on cardiovascular reactivity to stress (Gallagher et al., 2020; Ginty et al., 2020), no other study has examined the recovery period. Considered in the light of research indicating that exaggerated responses to stress are associated with negative cardiovascular health outcomes (Chida & Steptoe, 2010; Treiber et al., 2003), this

implies that state gratitude has an important stress-buffering function. This is consistent with prior work on gratitude and cardiovascular reactivity where gratitude played a unique role (Gallagher et al., 2020; Ginty et al., 2020). This also coheres with other previous research where positive psychological well-being has been associated with reduced cardiovascular mortality (Chida & Steptoe, 2008) and previous work has also noted how positive emotional states are important for the stress process (Folkman, 2008). Importantly, this finding was robust to the adjustment for control variables (Gallagher et al., 2020) and was adequately powered. The findings for diastolic blood pressure, while statistically non-significant, followed a similar pattern to that found for systolic blood pressure, whereby higher levels of state gratitude predicted lower diastolic blood pressure responses to the stress task. This interaction between state gratitude and quadratic growth explained 3% of the variance in diastolic blood pressure. Similarly, there was no evidence of a buffering effect on heart rate.

Affect balance did not moderate the growth trajectory for systolic blood pressure, diastolic blood pressure, or heart rate. This may reflect the fact that the stem of the affect-balance measures referred to positive and negative affect over the past week and as such was not as proximally relevant as state gratitude, leading to smaller effects. For example, there is some research describing the different effects of state and trait affect on outcomes such as cardiovascular stress responses (Määttänen et al. (2021); Papousek et al. (2010)). Future research should examine more proximal measures of affect-balance in order to further explore this relationship.

However, there was evidence that affect balance moderated the effects of state gratitude on diastolic blood pressure, but not systolic blood pressure or heart rate. The results indicate that a balance of more positive emotions to negative emotions amplified the effects of higher gratitude for diastolic blood pressure. A similar pattern is observable for systolic blood pressure and, although it did not reach statistical significance, it suggests that in a larger sample, this effect would be significant. Similarly, while the interaction for heart rate was significant, as the effect was less than .01, we cautiously interpret this as non-significant. This interaction may indicate that affect-balance does not directly impact the cardiovascular response to stress, but instead works to amplify

the effects of gratitude on the stress response.

Interestingly, this result is the opposite of what was expected. It was expected that individuals who experienced more positive than negative emotion would not benefit as much from experiences of gratefulness, in line with the resistance hypothesis (McCullough et al., 2004). For example, previous research found that low baseline positive affect amplified the relationship between a gratitude intervention on positive affect two weeks later (Froh et al., 2009). However, the results show that a balance of *more* positive to negative emotion amplifies the effects of state gratitude. Thus, the findings are more consistent with the broaden-and-build theory (Fredrickson, 2004) which posits that positive emotions can beget upward spirals which lead to optimal functioning (Garland et al., 2010; O'Connell et al., 2016). Additionally, and in line with past research suggesting the importance of considering positive and negative affect (Fredrickson, 2001; Kolanowski et al., 2014), this research tentatively suggests a role for considering affect balance when looking at the effect of state gratitude on cardiovascular stress responses.

A notable outcome of these results is that there are differing outcomes for systolic blood pressure, diastolic blood pressure and heart rate. This discrepancy may reflect differences in the cardiovascular and autonomic profiles of positive emotions (Shiota et al., 2011; Sinha et al., 1992), or differences in the cardiovascular profile of different emotional regulation strategies in response to stress (Griffin & Howard, 2022). For example, the discrepancy in the results may reflect differences in how gratitude and affect balance impact cardiac output, which is a determinant of systolic blood pressure, and total peripheral resistance, which is a determinant of diastolic blood pressure (Chaudhry et al., 2022; Magder, 2018; Tortora & Derrickson, 2019). Similarly, there are multiple determinants of heart rate, such as through parasympathetic nervous system activity or through the release of hormones like epinephrine and norepinephrine (Tortora & Derrickson, 2019). Research has found that positive emotions are differentially associated with cardiac output and total peripheral resistance (Kreibig, 2010), as well as parasympathetic nervous system activity (Levenson, 2014). Similarly, emotional regulation strategies in response to stress are differentially associated with cardiovascular outcomes (Griffin & Howard, 2022). For example, one study shows

that using a reappraisal emotional regulation strategy is associated with decreased total peripheral resistance (Mauss et al., 2007), and another shows that emotional suppression is associated with greater total peripheral resistance (Peters et al., 2014). As such, the differences in this study's findings may reflect the differences in the autonomic and cardiovascular profiles state gratitude and affect balance.

It is also worth noting that a recent review found that the kind of physiological measure utilized determined the strength of the effect for positive emotions on the cardiovascular response to stress (Behnke et al., 2022). Composite indices of the cardiovascular response to stress are constructed from multiple measurements such as finger pulse amplitude, blood pressure, and heart period are associate with larger effect sizes (Fredrickson et al., 2000; Fredrickson & Levenson, 1998). The reasoning behind their utilization is that they provide a better measure of sympathetic activation than any single measure alone (Behnke et al., 2022; Fredrickson & Levenson, 1998). This is in contrast to the use of studies which focus on heart rate and blood pressure, which index a combination of sympathetic and parasympathetic nervous system activity (Shiota & Danvers, 2014). This indexing of sympathetic and parasympathetic nervous system activity may also explain the differentiated findings in the current study (Shiota et al., 2011).

However, it is also possible that the moderating effect of affect balance and the buffering effects of state gratitude would emerge in a larger sample. Buffering effects have been detected for systolic and diastolic blood pressure and heart rate in larger samples (e.g. Ginty et al., 2020). Moreover, the effects of positive and negative affect on systolic and diastolic blood pressure, and heart rate are similarly undifferentiated (Brummett et al., 2009; Hilmert et al. (2014)). Indeed, it is possible that the effects are much smaller than anticipated, with one meta-analysis indicating that the undoing effects of positive emotions on cardiovascular responses to stress were small (Behnke et al., 2022). Nonetheless, this finding suggests that further research into gratitude, affect balance and the cardiovascular determinants of blood pressure and heart rate is warranted such as cardiac output and total peripheral resistance, as well as through the use of composite measures.

These results can be viewed as a stepping-stone to extend to clinical utility. There are a number

of low-cost gratitude interventions which can contribute to well-being (Wood et al., 2010). For example, gratitude lists whereby individuals write down three to five things for which they are grateful have been shown to have a number of beneficial effects (Kerr et al., 2015; Manthey et al., 2016). Previous research has shown how cardiac patients who make use of gratitude journals have better cardiovascular outcomes than those who do not (Redwine et al., 2016). Combined with the results of this study and previous work, gratitude may constitute a useful point of intervention for the improvement of cardiovascular health.

6.5.1 Limitations

There are some limitations of this study. First, it made use of self-report measures. These have several generic criticisms attached to them (e.g. Easterby-Smith et al. (2021)). However, self- report measures are frequently used in research and the measures of gratitude and positive and negative affect were psychometrically robust instruments. Additionally, their usage allows the comparison with other research (i.e. Gallagher et al., 2020). Second, this study did not make use of a gratitude induction; however, it did induce stress in a laboratory setting. It would be beneficial to induce gratitude in a randomised control trial context to assess its impact on cardiovascular responses to stress. Third, only blood pressure and heart rate were measured in this study. A fuller range of cardiovascular outcomes might prove instructive, for example, cardiac output and total peripheral resistance. However, the study was well-controlled and followed a standardised stress-testing protocol design. It also uncovered effects consistent with previous research.

6.5.2 Future directions

Future research to replicate and extend the findings with a larger, more diverse sample of participants would be useful to provide further investigate the interaction between affect balance

and gratitude in modulating cardiovascular responses to stress. According to previous stress-buffering models (Pressman et al., 2019), one way by which gratitude may buffer the effects of stress is by interacting with how stressful the task is perceived as. It would be useful to assess this relationship in a stress-testing protocol context. It is also recommended that future research investigate the impact of gratitude in the context of a randomised control trial using a gratitude induction. This will aid in untangling the causal direction at work and explicate the pathways by which gratitude may buffer the deleterious impact of stress.

6.6 Conclusion

This study found that state gratitude decreases the trajectory of the systolic blood pressure stress responses in terms of reactivity to stress and recovery from stress. Participants with higher state gratitude had lower systolic blood pressure responses to stress during the task and during the recovery period. This implies that gratitude is reducing reactivity and hastening recovery. This provides support for gratitude's stress-buffering role, as these effects withstood adjustment for age, gender and baseline measures. Additionally, this research detected a three-way interaction between gratitude, affect-balance and the growth trajectory for diastolic blood pressure. These novel findings suggest that state gratitude can act as a buffer against the negative cardiovascular effects of acute stress and a higher balance of positive to negative emotions amplifies this effect (at least for diastolic blood pressure). This contributes to our overall understanding of how gratitude impacts physical health.

Chapter 7

Does a short gratitude intervention impact cardiovascular reactivity?[3]

7.1 Introduction

Psychological stress is increasingly recognized as a significant contributor to cardiovascular health issues (Dimsdale, 2008; Steptoe & Kivimäki, 2012), a concern underscored by the World Health Organization's identification of cardiovascular diseases as the leading cause of death globally, ac- counting for 17.9 million deaths in 2019 (WHO, 2021). Although we cannot avoid stress com- pletely, our reaction to stress can be modified through various means, and this can impact on physical health and wellbeing (Dimsdale, 2008; Kop (1999); Steptoe & Kivimäki, 2012_. Past research on interventions to intervene to modify stress reactions has tended to focus on perceived stress, with less research focusing on biobehavioural reactions. Cardiovascular reactivity refers to the magnitude of change in an individual's cardiovascular state in response to

[3] Note: This study has been submitted for publication: Leavy, B., O'Connell, B.H., & O'Shea, D. (under review).
Does a short gratitude intervention impact cardiovascular reactivity? Submitted to *Journal of Happiness Studies* in Spring 2024. ISI IF: 3.86

acute psychological stress (Hughes & Lü, 2017). Atypical cardiovascular reactivity to stress (if exaggerated or prolonged) has negative health-related implications such as cardiovascular disease mortality and hypertension (Carroll et al., 2012; O' Riordan et al., 2022; Yuenyongchaiwat, 2015).

Previous research has identified stable characteristics (e.g. age, personality) that render certain people more susceptible to the harmful effects of stress than others (Soliemanifar et al., 2018; Uchino et al., 2010). Similarly, psychophysiological scholars have explored the role of psychosocial factors, like positive affect and social support, in buffering against pathogenic effects of stress (e.g. Brummett et al., 2009; Uchino et al., 2011). Of these, gratitude has recently been associated with the cardiovascular stress response (Gallagher et al., 2020; Ginty et al., 2020). However, previous research has been predominantly cross-sectional in nature (Ginty et al., 2020). While cross sectional studies have significantly contributed to our understanding of the relationship be- tween state gratitude and cardiovascular reactivity, their inherent limitations underscore the need for experimental research (Easterby-Smith et al., 2021; Solem, 2015). The present study aimed to fill this gap by experimentally manipulating state gratitude in a laboratory setting to clarify the relationship between state gratitude and cardiovascular reactivity.

7.1.1 Gratitude and cardiovascular reactivity

Gratitude is a positive emotion that focuses on appreciation for the good someone has in their life and can be viewed as both a state and as a trait (Wood et al., 2010). As a state, gratitude refers to feelings of appreciation experienced in response to the good things in a person's life. As a trait, it refers to an individual's disposition to appreciate those same good things (Wood et al., 2010). Gratitude has been associated with improved cardiovascular health (Cousin et al., 2021), including lowered pro-inflammatory biomarkers (Redwine et al., 2016) and lowered ambulatory diastolic blood pressure (Jackowska et al., 2016).

Recent research suggests that gratitude plays a pivotal role in stress appraisal (Wood et al., 2010), and subsequent cardiovascular outcomes (Cousin et al., 2021), aligning with both the Stress

and Coping Model (Biggs et al., 2017; Lazarus, 1999) and the Broaden and Build theory (Fredrickson, 2001, 2004). According to the Stress and Coping Model, stress is a subjective experience involving both primary appraisal—evaluating the personal significance of an event—and secondary appraisal—assessing the available resources to cope with the event (Lazarus, 1999). Within this framework, gratitude emerges as a valuable positive emotion that influences these appraisals, as it helps individuals to reframe stressful events as challenges rather than threats and to perceive higher levels of available coping resources (Wood et al., 2010). The Broaden and Build theory complements this perspective by proposing that positive emotions like gratitude 'broaden' cognitive and emotional repertoires, thereby helping individuals to 'build' resources that are beneficial for both psychological and physiological resilience (Fredrickson, 2004).

Based on the integration of these theories, it is expected that gratitude should promote effective coping by expanding cognitive flexibility (Hartanto et al., 2020), encouraging healthy behaviours, such as better diet, (Boggiss et al., 2020) and increasing social support (A. M. Wood, Maltby, Gillett, et al., 2008). As such, gratitude may not only mitigate the psychological distress associated with stress but may also serve as a buffer against the harmful cardiovascular effects of stress (Froh, 2008; Kubzansky et al., 2018; Schache et al., 2019).

Recent observational research has confirmed that state gratitude is inversely associated with cardiovascular stress reactivity. A laboratory-based study conducted by Gallagher et al. (2020) uncovered an inverse relationship between state gratitude and systolic blood pressure reactivity, indicating that state gratitude is beneficial for cardiovascular health. Similarly, Ginty et al. (2020) also found an inverse relationship between systolic blood pressure reactivity and state gratitude, while additionally finding an inverse association between state gratitude and diastolic blood pressure reactivity and heart rate reactivity.

Although there has been previous cross-sectional research on the subject, these studies are limited in their ability to establish causal relationships between gratitude interventions and cardiovascular reactivity (Ginty et al., 2020). A randomized control trial offers a robust framework for making causal inferences with less bias and confounding (Spieth et al., 2016). Importantly, in the

absence of a randomized control trial, one could argue that the observed association between state gratitude and cardiovascular reactivity may actually be a case of reverse causality, where reactivity causes state gratitude (Antonakis et al., 2010; Sattar & Preiss, 2017). Therefore, a randomized control trial is crucial for providing stronger insights into the relationship between state gratitude and cardiovascular reactivity.

Previous studies have shown that gratitude can be successfully manipulated through interven- tions (Cregg & Cheavens, 2021; Davis et al., 2016; Kirca et al., 2023). Gratitude interventions are structured activities designed to cultivate feelings of appreciation and thankfulness (Boggiss et al., 2020). They aim to shift individuals' focus from negative aspects to positive aspects of their lives, fostering a sense of gratitude and well-being (Cregg & Cheavens, 2021; Kirca et al., 2023). Examples include gratitude journaling, gratitude letters, and mindfulness-based practices (Davis et al., 2016). Evidence suggests that gratitude interventions have positive associations with physical health (Boggiss et al., 2020). As the demand increases to find new interventions that may reduce the impacts of cardiovascular disease (Kubzansky et al., 2018), positive psychology can offer low- cost interventions that are largely endorsed by their practitioners (Boggiss et al., 2020; Wood et al., 2010), and which can be implemented alongside medical interventions.

7.1.2 Study aims and hypotheses

In sum, recent evidence underscores the potential protective role of state gratitude against the detrimental effects of stress on cardiovascular reactivity (Gallagher et al., 2020; Ginty et al., 2020). These effects are broadly consistent with predictions from the model of psychological wellbeing (Boehm & Kubzansky, 2012), and the stress and coping model (Folkman, 2008). However, little research on gratitude interventions to date has moved beyond self-report indicators of stress by focusing on biobehavioural indicators of stress, as is done in this study. In doing so, this study addresses limitations of past research examining gratitude interventions and cardiovascular reactivity

(Ginty et al., 2020). In line with this, the present study aims to experimentally manipulate state gratitude using an intervention to assess its relationship with cardiovascular reactivity. As such, this study has the following hypotheses:

H1: A gratitude intervention will be associated with reductions in systolic blood pressure re- activity.

H2: A gratitude intervention will be associated with reductions in diastolic blood pressure reactivity.

H3: A gratitude intervention will be associated with reductions in heart rate reactivity.

7.2 Methods

7.2.1 Design

This study used a randomized control trial design in a laboratory setting, and specifically made use of a parallel group trial design (Nair, 2019). The study experimentally manipulated state gratitude in a laboratory setting that induced stress. Participants received either instructions to write a gratitude letter (experimental condition) or were given a neutral writing activity (active control condition). Following this, all participants underwent two separate stress tasks in counterbalanced order, denoted as the stress task period. Blood pressure and heart rate readings were taken to assess the cardiovascular response to stress. The independent variable was treatment group, either control or experimental group. The outcomes were systolic blood pressure, diastolic blood pressure, and heart rate.

7.2.2 Participants

This study used a convenience sample of 129 participants who volunteered to partake in the study. Of these 35 were male and 93 were female. The ages ranged from 18 to 58 (M = 23.24, SD = 6.29). The inclusion criteria included individuals who were 18 years old or older and able to consent. Participants were excluded if they (1) consumed alcohol in the twelve hours before the study took place, (2) engaged in vigorous exercise in the twelve hours before the study took place, (3) consumed caffeine or smoked cigarettes less than two hours before the study took place, (4) consumed food one hour before the study, (5) were pregnant, or (6) currently held a diagnosis of cardiovascular disease. These precautions were to control for confounding variables and are in line with previous research (Creaven & Hughes, 2012; Gallagher et al., 2020). The power analysis was based on an analysis of power conducted in *G*power* (Faul et al., 2007) with a modest effect size of .25, an error probability of 0.05, and a desired power of 0.8. With two groups (experimental and control), the numerator degrees of freedom was set to 1. Additionally, the analysis included five covariates. This resulted in a sample of 128. An effect size of .25 was selected optimistically as previous gratitude interventions had modest effect sizes (e.g. Davis et al., 2016).

7.2.3 Procedure

The study was advertised via a departmental scheme whereby participants received credit for participation in the study. It was advertised as a study about the effects of a writing exercise on the cardiovascular response to stress. Potential participants were not informed that the study was examining gratitude. Potential participants were told about the restrictions on diet and exercise they would have to follow prior to participation, and that they would need to attend a laboratory session in-person to take part.

Following their agreement to take part, participants were asked to attend a laboratory session. Prior to attending, participants were instructed to refrain from drinking alcohol or exercising in the 12 hours prior to the study and to refrain from smoking and consuming caffeine two hours before, or eating one hour before the study took place. Experimental materials including questionnaires

and the instructions for the experimental condition were placed in opaque sealed envelopes to ensure concealment of condition (Schulz, 2001). Blocks of envelopes were utilized to ensure randomization to the control and experimental conditions (Kim & Shin, 2014).

Figure 7.1 provides a visual overview of the procedure. Participants were recruited using flyers, posters, and through a participation-for-credit programme operating in the department of psychology. Students were given the option to participate in two hours of studies in return for credit, or writing an essay instead. Randomisation was ensured by simple randomisation (Beller et al., 2002). Blocks of eight packets of surveys and intervention instructions were printed and stored in opaque envelopes. These blocks contained four control packets and four experimental intervention packets. The researchers were unaware of which packet corresponded to which intervention.

Upon arrival, the acclimatization period began at the laboratory. During this time, participants were greeted by the research and instructed to read an information sheet to confirm eligibility and provide their informed consent. The researcher recorded the participant's height and weight measurements for the computation of BMI. They were then seated and the blood pressure cuff was placed on their upper, non-dominant arm, and they were instructed to place their feet in a box under the table in order to control for movement during the study (Pickering et al., 2005). They were asked to remain as stationary as possible as excessive movement would interfere with the measurements (Dienberg-Love et al., 2010). Following this, they were handed a package of surveys which included instructions on the experimental manipulation the participant would receive. The packets of surveys were randomised so the researcher would not know which condition the participant received. The surveys were also hidden by use of a divider to ensure the researchers could not see the instructions that the participants received. The surveys included demographic details and psychometric scales.

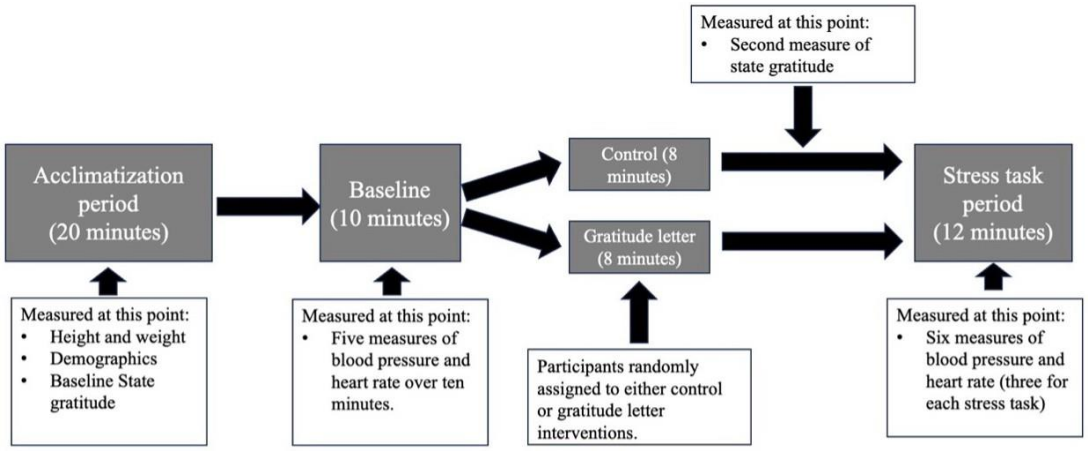
The acclimatization period lasted 20 minutes. The participant was asked to refrain from speaking for the remainder of the experiment. During this period, the participant completed a questionnaire comprising demographic variables and the Gratitude Adjective Checklist (the first

measurement of state gratitude). This was followed by a formal ten-minute baseline period where cardiovascular measures were taken every two minutes. Once the formal baseline was completed, the participant completed either a gratitude letter or an exercise where they described the room, which served as the active control condition. Both the gratitude letter condition and control condition lasted eight minutes. The instructions for both conditions can be found in Appendix A. After completing the intervention, both groups completed the Gratitude Adjective Checklist again to assess whether the intervention had successfully manipulated state gratitude.

Following this, the stress task period began. The researcher turned off the lights, turned on a spotlight and explained to the participant that they would be completing a serial sub- traction task and a public speaking task. The order was randomly determined by the researcher by means of a coin toss. The stress tasks lasted for six minutes each. Blood pressure measurements were taken throughout at two-minute intervals, yielding three measurement points. Researchers were instructed to behave in a neutral manner; this was due in part to the necessity of mask-wearing to remain in compliance with departmental covid-19 safety protocols. Prompts to continue were delivered by the researcher if the participant stopped engaging at any stage. After the task, the lights were switched back on and participants were asked to rate how stressful they found the task. They then filled out a self-report stress questionnaire and were debriefed and thanked for their time after study completion.

Ethical approval was granted by the Biomedical & Life Sciences Research Ethics Subcommittee (BSRESC-2021-2441521).

Figure 7.1: Overview of the experimental procedure detailing each phase and tis duration



7.2.4 Intervention

Participants in the experimental condition were asked to complete a brief, 8-minute gratitude intervention comprising writing a gratitude letter. Participants were instructed to "Think of someone in your life who you feel like you have never fully or properly thanked for something meaningful or important that they did for you. In the space provided below and on the next page, please write a note to this person that describes why you feel like you never properly thanked them and letting them know why you feel thankful for something important that they did for you." Gratitude letters have been associated with increases in feelings of gratitude as well as decreases in depression, anxiety, and increases in feelings of well-being (Froh et al., 2008). This study used an adaptation of the Froh et al. (2009) intervention, with the main difference that participants did not send their gratitude letters. An important factor for gratitude interventions is dosage – for how long the intervention is carried out (Davis et al., 2016). It has been suggested that positive psychological interventions of longer duration have larger effects than brief interventions (Carr et al., 2021). However, recent studies have shown that brief gratitude interventions of 5 to 10 minutes can also have significant associations with stress (Fekete & Deichert, 2022; Komase et al., 2021).

As a control condition, the study used a neutral, active control. In the active control condition, participants were asked to complete a writing task for eight minutes describing the laboratory in

which the study was conducted. Utilizing an active control conditions responds to criticisms of past research which used measurement-only controls, or control interventions that utilize hassle lists as they may increase stress (Davis et al., 2016).

7.2.5 Measures

Stress tasks. This study used two stress tasks and participants completed both stress tasks. The order in which they received the tasks was counterbalanced. These tasks were adaptations of the Trier Stress Test, which asks participants to give an interview-style presentation followed by an arithmetic test (Allen et al., 2016). The tasks were adapted in line with prior research such that the order in which the tasks were presented was counterbalanced (Gallagher et al., 2021).

Serial subtraction task: Participants were asked to subtract in increments of thirteen from 1,222 (al'Absi et al., 1995). If an error was made, they were required to start over. This type of mental arithmetic task is commonly used to elicit a physiological stress response in a lab setting (Mathias et al., 2017; Whittaker et al., 2021). The task lasted for six minutes, not including the time taken to explain the task.

Public speaking task: Participants had two minutes to prepare a speech about three of their best and worst qualities. They then had four minutes to deliver this speech to the primary researcher (Bosch et al., 2009). To increase stress levels during the task, the researcher wore a white lab coat, decreased the room lighting, and directed a light toward the participant. The task lasted for six minutes, excluding time for explanation. This task has been used in prior research on gratitude and stress (Gallagher et al., 2021).

The laboratory setting also added pressure by: (1) including a fake leader board directly opposite the participant, (2) using a small lab with two researchers present, and (3) switching off the main laboratory light during the tasks, leaving the room illuminated by a lamp in front of the participant.

Cardiovascular Assessment. Systolic blood pressure, diastolic blood pressure and heart

rate in beats per minute were measured using a GE Dinamap Pro 400 V2 vital signs monitor (GE Medical Systems, Freiburg, Germany). A stopwatch was used to ensure that measurements were taken at consistent times. A standard blood pressure cuff was placed over the brachial artery on the participant's non-dominant arm. After acclimatization, five baseline measurements were taken over a ten-minute period. Six measurements were taken during the two six-minute stress tasks during the stress task period.

State gratitude was measured using the Gratitude adjective checklist (GAC; McCullough et al., 2002). The scale is the sum of three adjectives: grateful, thankful, and appreciative, assessed using a 5-point Likert scale (1 = not at all to 5 = extremely) with higher scores reflecting higher levels of state gratitude. Participants were instructed to rate their feeling "right now". The scale was summed in line with previous research and scores ranged from 3 to 15 (Gallagher et al., 2020). This was assessed twice during the study. Once during the acclimatization period to get a baseline measure for state gratitude, and once after the intervention to test whether individuals in the gratitude group had increased levels of state gratitude. This scale has demonstrated high internal reliability (a = .83) in prior research (Froh et al., 2010) and this was confirmed in the current study at both the first (a = 0.87) and second (a = 0.88) measurement points.

Control variables were selected based on their well-established relationships with cardiovascular reactivity and cardiovascular health. These are as follows: *Age* and *sex* (Canto et al., 2012). *Body mass index* (BMI), which was assessed using a weighing scales and measuring tape (Bucholz et al., 2012).

A single item self-report health scale was used to assess *overall health*. This asked participants to rate their health on a scale from 1-5 (Ahmad et al., 2014).

Two single-item measures ranging from 0-6 (0 =Not stressful at all, 6 = Extremely stressful) were used to assess how *stressful* participants found each stress task (Gallagher et al., 2020).

7.2.6 Data analysis

Data screening

Data were screened, outliers removed, normality checked, and manipulation checks were carried out using R version 4.2.0. Manipulation checks were carried out in line with best practice (Gallagher et al., 2020; O' Riordan et al., 2022). Five resting baseline measures for each of the cardiovascular parameters were averaged to yield baseline values for each participant. The same process was used for the six stress task measures from the stress task period (Phillips et al., 2009). Data were screened and checked for normality using the visual inspection of histograms, QQ-plots, and by utilizing the Shapiro test for normality. Outliers were assessed for the average of the stress task period measures using z-scores, with a z-score >= |3| constituting an outlier. No outliers were detected.

Manipulation checks

T-tests to compare the conditions across age, baseline state gratitude, and baseline cardiovascular parameters were conducted to confirm that the randomization procedure was successful. A t-test was also conducted on the second measure of state gratitude to ensure that the intervention had successfully manipulated state gratitude. Repeated measures ANOVAs across two timepoints were used to confirm that the stress task manipulation was successful and increased cardiovascular responses from baseline. Correlations between study variables were examined prior to hypothesis testing.

Hypothesis testing

JASP version 0.16.3 (JASP Team, 2023) was used to assess the hypotheses. Two-way mixed between-within ANOVAs were used in examine the effects of the intervention (gratitude letter or active control) and time (from mean baseline to the mean of the stress task period) on systolic blood pressure, diastolic blood pressure and heart rate.

The intervention type was the between-subjects factor; participants were grouped based on

which intervention they received either the gratitude letter or the active control. This helped us compare the overall differences in cardiovascular responses between these two distinct groups. Time was the within-subjects factor, comparing two specific time points - from the mean baseline to the mean of the stress task period for each participant. This allowed us to assess how cardiovascular responses changed over time within each individual, regardless of which intervention they received. An alpha level of .05 was selected. Effect sizes are reported in partial eta square.

7.3 Results

7.3.1 Preliminary analyses and randomization checks

Descriptive statistics and correlations between the study variables are outlined in Table 7.1. There was a significant, positive correlation between baseline state gratitude and health ($r = 0.21$, $p = 0.016$) and positive affect, ($r = 0.36$, $p = <.001$), but there were no significant correlations between baseline state gratitude and baseline cardiovascular parameters, or post-intervention state gratitude and any baseline cardiovascular parameters.

Tests of baseline homogeneity were conducted and no significant differences between control and intervention groups were found in age, $t(90.93) = -1.40$, $p = 0.164$; baseline state gratitude $t(126.40) = -0.98$, $p = 0.331$, baseline systolic blood pressure $t(126.33) = 0.00$, $p = 0.997$, baseline diastolic blood pressure $t(126.82) = -1.53$, $p = 0.128$, or baseline heart rate, $t(126.80) = -0.12$, $p = 0.905$. Thus, the randomization procedure was successful.

Table 7.1: Correlations with descriptive statistics

Variable	Mean	SD	Min	Max		1	2	3	4	5	7	8	9	10	11
1. Age	23.24	6.29	18	58											
2. BMI	205.78	62.1	118.5	398.54	0.15										
3. Health	4.21	0.68	2	5	-0.02	-.33**									
4. Baseline state gratitude	10.22	2.53	3	15	0.15	-0.11	.21*								
5. Post intervention state gratitude	11.08	2.43	6	15	-0.01	-0.1	.21*	.58**							
6. Baseline SBP	111.71	9.09	91.6	137.8	0.15	.35**	-.24**	0.02	0.01						
7. Baseline DBP	65.36	7.41	52	87.4	0.14	0.07	-.17*	0.05	0.1	.71**					
8. Baseline HR	78.54	11.63	54.4	109.6	-0.09	-0.11	0.09	-0.09	-0.02	.27**	.40**				
9. Stress tasks SBP	127.34	11.36	103.8	154.4	0.13	.33**	-0.13	-0.03	-0.01	.75**	.52**	0.09			
10. Stress tasks DBP	75.67	7.99	56.4	98.4	-0.02	0.16	-.21*	-0.01	0.01	.63**	.78**	.22*	.65**		
11. Stress tasks HR	89.8	12.92	60.8	133	-.20*	-0.16	0	-0.1	-0.11	.21*	.25**	.76**	.24**	.20*	

Note. * indicates $p < .05$. ** indicates $p < .01$. SBP = Systolic blood pressure (mmHG), DBP = Diastolic

7.3.2 Manipulation checks

A series of repeated measures ANOVAs were used to check if the stress task increased cardiovascular responses from the baseline period to stress period, for systolic blood pressure F(1, 128) = 557.23, < .001, η_p^2 = 0.81, diastolic blood pressure F(1, 128) = 527.53, < .001, η_p^2 = 0.80, and heart rate F(1, 128) = 218.53, < .001, η_p^2 = 0.63, with results indicating statistically significant changes from baseline to stress task phases.

Welch's independent samples t-tests indicated that levels of state gratitude were significantly higher in the gratitude letter group (M = 11.88) post intervention compared to the active control group (M = 10.28), t(118) = 3.80, p <.001, with a moderate effect size, Cohen's d = 0.70, 95% CI[0.34, 1.09].

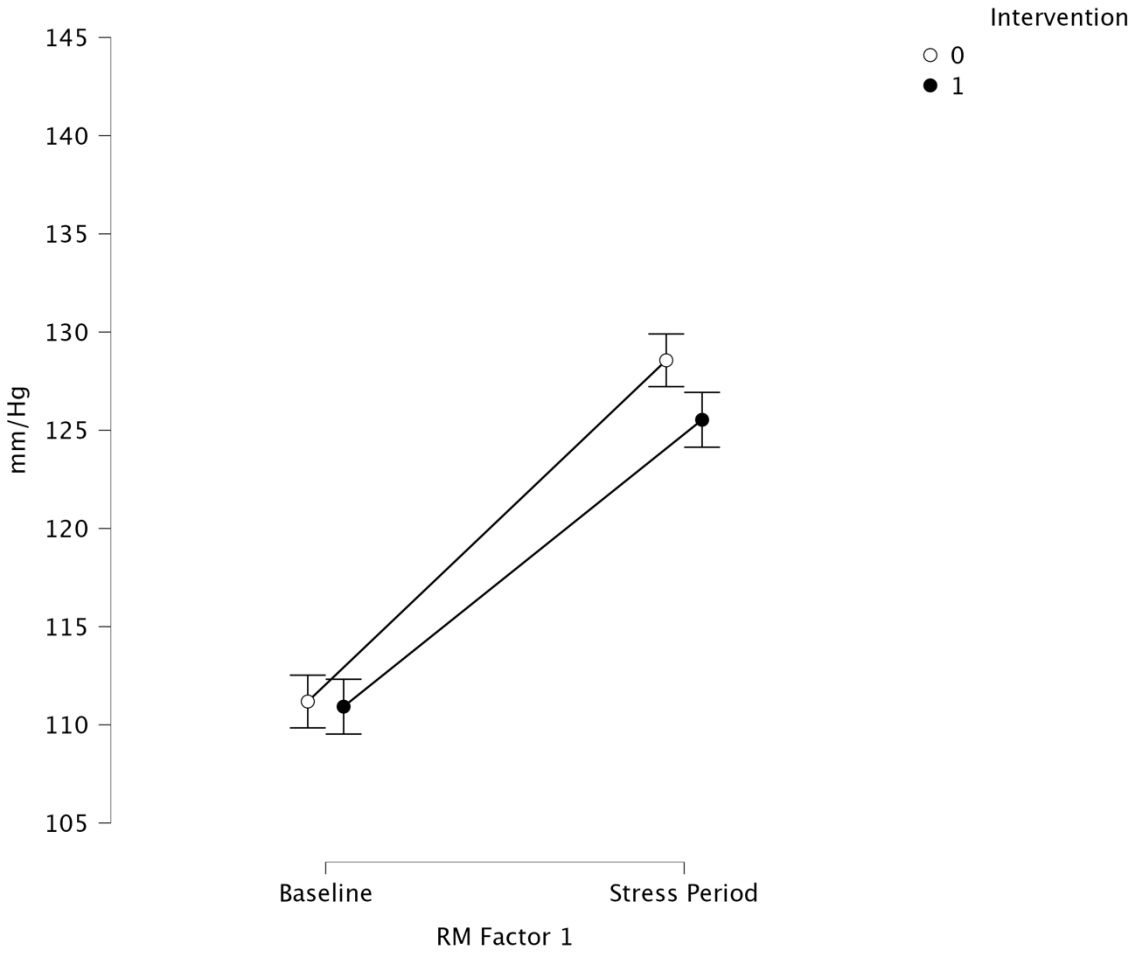
7.3.3 Hypothesis testing

A 2 (Time: Baseline vs. Stress task period) x 2 (Treatment: Control vs. Gratitude letter) mixed ANOVA, controlling for age, sex, BMI, health status, and how stressful each stress task was perceived to be, was conducted to examine the impact of the intervention on the cardiovascular response to stress from the baseline to the stress task period for systolic blood pressure, diastolic blood pressure, and heart rate (see Tables 7.2 and 7.3, and Figure 7.2)

Looking first at the main effects, the effects of the gratitude intervention on systolic blood pressure, $F(1, 120) = 0.908$, $p = .343$, $\eta^2_p = 0.006$, diastolic blood pressure $F(1, 109) = 0.908$, $p = .343$, $\eta^2_p = 0.006$, and heart rate $F(1, 109) = 0.812$, $p = .369$, $\eta^2_p = 0.006$, were not significant, suggesting that the two treatment groups did not significantly differ in their overall cardiovascular responses (see Table 7.2).

Hypothesis 1 predicted that the intervention would be associated with changes in systolic blood pressure between baseline and stress periods (see Table 7.3). For systolic blood pressure, the interaction of time X intervention was significant, $F(1, 120) = 4.573$, $p = .035$, the effect was small, $\div^2 = 0.006$. This implies that the pattern of increase for systolic blood pressure differed by experimental condition. Inspection of Figure 7.2 indicates that individuals who write a gratitude letter had smaller systolic blood pressure increases. This supports hypothesis 1.

Figure 7.2: Interaction plot showing the increase from baseline to stress period for systolic blood pressure



Hypotheses 2 and 3 predicted that the intervention would be associated with changes in both diastolic blood pressure and heart rate between baseline and stress periods. Looking at the within-person effects, there were no significant time X intervention interactions for diastolic blood pressure $F(1, 109) = 1.234$, $p = .269$, $\eta^2_p = 0.001$, or heart rate $F(1, 109) = 1.964$, $p = .164$, $\eta^2_p = 0.002$) indicating that the pattern of increase from the baseline to the stress task period did not differ significantly between the gratitude letter and control condition for these parameters. These results did not support hypothesis 2 or hypothesis 3. Results can be found in Table 7.3.

Table 7.2: Main effects for the mixed effects ANOVAs for systolic blood pressure, diastolic

blood pressure and heart rate.

Between Subjects Effects	Systolic			Diastolic			Heart rate		
Variables	F	p	η_p^2	F	p	η_p^2	F	p	η_p^2
Age	2.35	0.13	0.02	0.12	0.73	0	1.3	0.26	0.01
Sex	0.38	0.54	0	0.84	0.36	0.01	0.13	0.72	0
BMI	10.25	0	0.09	2	0.16	0.02	0.33	0.56	0
Health	0.11	0.74	0	2.97	0.09	0.03	0.77	0.38	0.01
Speech task perceived stress	0.69	0.41	0.01	0	1	0	2.67	0.11	0.02
Maths task perceived stress	0.93	0.34	0.01	0.7	0.4	0.01	0.01	0.93	0
Intervention	0.91	0.34	0.01	0.37	0.54	0	0.81	0.37	0.01

Note. Type III sum of squares. ÷^{2} = Partial Eta Squared.

Table 7.3: Within-subjects effects including interactions for the mixed effects ANOVAs for systolic blood pressure, diastolic blood pressure and heart rate

	Systolic			Diastolic			Heart Rate		
Variables	F	p	η_p^2	F	p	η_p^2	F	p	η_p^2
Time	0.37	0.54	0	3.01	0.09	0.03	0.31	0.58	0
Time*Age	0.12	0.73	0	7.82	0.01	0.07	3.27	0.07	0.03
Time*Sex	0	0.95	0	0.08	0.79	0	0.02	0.9	0
Time*BMI	0.51	0.48	0.01	3.9	0.05	0.04	0.01	0.94	0
Time*Health	1.41	0.24	0.01	0.01	0.91	0	1.95	0.17	0.02
Time*Stress task perceived stress	2.89	0.09	0.03	2.8	0.1	0.03	1.1	0.3	0
Time*Math task perceived stress	0	0.95	0	0.05	0.82	0	0.33	0.57	0
Time*Intervention	4.57	0.04	0.04	1.23	0.27	0.01	1.96	0.16	0.02

Note. Type III sum of squares. ÷^{2} = Partial Eta Squared.

7.4 Discussion

In this study, the effects of a gratitude intervention on cardiovascular responses to stress were examined using a randomized controlled trial design in a laboratory setting. Our preliminary analyses revealed significant increases in systolic blood pressure, diastolic blood pressure, and heart rate from baseline to the stress task phases, indicating that the stress task manipulation was successful. Additionally, the results suggest that participants in the gratitude letter condition exhibited significantly higher levels of state gratitude post-intervention compared to the control group, implying the successful induction of gratitude. Finally, it was found that participants who wrote a gratitude letter had a smaller increase in systolic blood pressure compared to the control group. This effect was robust to adjustment for age, sex, BMI, and overall health. Effects were not detected for condition across time for diastolic blood pressure or heart rate.

The relationship between inducing state gratitude (via writing a gratitude letter) and systolic blood pressure is consistent with both the Stress Coping theory (Lazarus, 1999) and the Broaden and Build theory (Fredrickson, 2004b). In this context, gratitude may help individuals to reframe stressful events as challenges rather than threats and enhances perceptions of available coping resources (Wood et al., 2010). This may, in turn, lessen the impact of stress on systolic blood pressure, reinforcing gratitude's role as a buffer against cardiovascular stress effects. Similarly, Broaden and Build theory supports our findings by suggesting that positive emotions like gratitude can 'broaden' cognitive flexibility and emotional repertoires (Fredrickson, 2001, 2004a; Fredrickson, 2004b). By doing so, gratitude could facilitate effective coping mechanisms like reappraisal, planning, and goal-directed strategies, thereby reducing the harmful cardiovascular effects of stress (Bryan et al., 2018; A. Wood et al., 2007). This interpretation is not only supported by our findings but is also consistent with a growing body of evidence, including studies by Gallagher et al. (2020, 2021) and Ginty et al. (2020), which similarly suggest an inverse relationship between gratitude and systolic blood pressure reactivity.

Importantly, while past studies are predominantly cross-sectional in nature, a strength of our

study is its use of a randomised control trial design. In the absence of a randomized control trial the observed association between state gratitude and cardiovascular reactivity may be due to reactivity causing gratitude (Antonakis et al., 2010; Sattar & Preiss, 2017). The randomized control trial design of this study isolates the effect of state gratitude on cardiovascular reactivity, mitigating the risk of reverse causality (Hariton & Locascio, 2018). Therefore, it can be concluded with greater confidence that induced state gratitude has a protective effect on cardiovascular health (Sattar & Preiss, 2017; Spieth et al., 2016).

The effect was small, consistent with previous research on the effect sizes of gratitude interventions (Cregg & Cheavens, 2021). The small effect size reflects that brevity of the intervention, lasting only eight minutes. Arguably, a daily intervention over at least a week or several weeks should be associated with larger effect sizes (Dickens, 2017; O'Connell et al., 2016; O'Connell & Killeen-Byrt, 2018). For example, Redwine et al. (2016) detected a large effect size for an eight- week gratitude intervention on proinflammatory biomarkers (d = 1.03). Similarly, a three-week gratitude intervention was associated with a large effect on sleep quality (d =0.81) (Southwell & Gould, 2017). A meta-analysis found dosage to be an important factor, with daily interventions over three to five days more likely to be associated with significant relationships with health (Boggiss et al., 2020). However, our study demonstrates that even with a short duration gratitude activity, changes can be seen in the systolic blood pressure reaction to stress.

Importantly, research indicates that relatively modest effects can have clinical significance. While typically focusing on changes of 10 mm/Hg for systolic blood pressure (Williams et al., 2018), determining clinically relevant effects of blood pressure decreases depends on variables like age and pre-existing blood pressure (Law et al., 2009; Williams et al., 2018), making estimating an absolute threshold of clinical significance difficult. Law et al. (2009) present a way of calculating expected reductions in disease events for 3 mm/hH reduction in blood pressure:

$$1 - Relative\ Risk\ Reduction = S^{d/20}$$

Where S is the age specific slope showing the association between blood pressure reduction and relative risk in either stroke or coronary heart disease (see appendix in Law et al., (2009)) and d is the reduction in blood pressure. For example, a S slope value of 0.5, this specifically means that

for every 20 mm/Hg reduction in systolic blood pressure (SBP), the relative risk of cardiovascular events (such as coronary heart disease or stroke) decreases by 50% (since 0.5 * 20 = 10, and 10/20 = 0.5, indicating a 50% reduction for a full 20 mm/Hg drop).

While the reduction of 3 mm/hg in systolic blood pressure is modest, using the equation in Law et al., (2009), to calculated expected reduction in disease events for 3 mm/Hg reduction in blood pressure in 40-49 year olds, we get an 14% reduction in the relative risk of stroke:

$$1 - \ Relative\ Risk\ Reduction \ = \ 0.36^{3/20} \ = \ .14$$

And 10% reduction in coronary heart disease incidents:

$$1 - \ Relative\ Risk\ Reduction \ = \ 0.49^{3/20} \ = \ .10$$

Thus, while 3mm/Hg reduction is modest, depending on age-specific relationships with disease events, it can result in significant effects.

As previously mentioned, effects for diastolic blood pressure or heart rate were not detected. Previous research has also found differential effects for different cardiovascular parameters with systolic blood pressure being the parameter most commonly found to have a relationship with gratitude. For example, Gallagher et al. (2020) only detected relationships between state gratitude and systolic blood pressure reactivity. Collectively, these findings may suggest that the effects of state gratitude on diastolic blood pressure and heart rate are more difficult to detect than systolic blood pressure, or that such effects might only emerge over longer periods of time perhaps. Certainly, future research is warranted to explore these tentative explanations.

7.4.1 Strengths and Limitations

This study has several strengths and novel contributions. It makes use of a standardised and well- controlled laboratory-based stress tasks to assess cardiovascular reactivity. is the first to show how the experimental manipulation of gratitude impacts the cardiovascular stress response. The

use of a randomized control trial design in this study adds robustness to the findings, allowing for stronger inferences about the relationship between state gratitude and cardiovascular outcomes. This methodological choice strengthens the argument that gratitude serves as a protective mechanism.

However, the study used a convenience sample, which may introduce bias and limit the generalizability of the findings. Participants volunteered based on their availability and proximity to the study location, which may not represent the broader population. The study relied on self-report measures for variables such as state gratitude. Self-report measures can be influenced by various biases, such as social desirability bias or recall bias (Paulhus, 2017). However, the measures had previously been found to be both reliable and valid (Froh et al., 2011; Watson et al., 1988). The intervention was also brief, with further work needed to clarify the long-term cardiovascular health benefits of gratitude interventions. Similarly, the validity of the control group intervention warrants further examination with lists of daily activities constituting a more common active control (Regan et al., 2023). However, the advantage of the control intervention in Study 3 included being able to exercise greater control over what participants wrote.

7.4.2 Future directions

Future research would do well to replicate and extend the findings with a larger, more diverse sample of participants. It would also be helpful to assess a greater range of cardiovascular parameters such as total peripheral resistance, cardiac output, and heart rate variability. These are useful to connect gratitude to autonomic nervous system activity (Thayer et al., 2009; Uphill et al., 2019). Similarly, future research should examine the long-term relationships between gratitude, cardiovascular reactivity, and cardiovascular health outcomes. Finally, future research should explore how daily interventions over a larger period of time are associated with reactivity (Boggiss et al., 2020).

7.5 Conclusion

This study provides preliminary evidence suggesting that state gratitude may have a beneficial im- pact on cardiovascular responses to stress. Cardiovascular diseases are a leading cause of global mortality, emphasizing the need for effective interventions to reduce their burden. Stress is a significant risk factor for cardiovascular illness, highlighting the importance of investigating factors that can mitigate the adverse effects of stress. Participants who engaged in a gratitude intervention showed lower systolic and diastolic blood pressure during the stress task compared to the control group. These findings align with previous research, supporting the notion that state gratitude could be a protective factor for cardiovascular health. However, it is important to acknowledge the limitations of the study, such as the convenience sample and reliance on self-report measures. Further research with larger and more diverse samples is necessary to confirm and expand upon these findings, including the exploration of additional cardiovascular parameters and long-term relationships between gratitude, cardiovascular reactivity, and health outcomes.

Chapter 8

Overall discussion

The overarching aim of this thesis was to examine how gratitude impacts the cardiovascular response to stress. This thesis built on a growing body of research highlighting the health benefits of gratitude and the potential of gratitude interventions to promote positive cardiovascular health. However, prior to this thesis, little research had been conducted on this subject and particularly there is a paucity of research on gratitude and cardiovascular recovery, the longitudinal cardiovascular outcomes associated with gratitude through reactivity, and the effects of gratitude interventions on cardiovascular stress responses. This thesis contributes to this with three studies examining gratitude and the cardiovascular response to stress.

8.1 Overall summary of findings

Building on past research, this thesis had the following aims:

Assess the impact of trait gratitude on the risk of suffering acute myocardial infarction indirectly through cardiovascular reactivity.

Assess the impact of state gratitude on cardiovascular reactivity and recovery.

Assess the interaction between affect balance and state gratitude on cardiovascular reactivity and recovery.

Conduct a randomised-control trial study to examine the effectiveness of gratitude interventions in modulating cardiovascular reactivity and recovery in response to stress.

Study One addressed the first aim as it examined the long-term, indirect relationship between trait gratitude and the risk of acute myocardial infarction through cardiovascular reactivity over 6.7 years. This study found that heart rate reactivity mediated the relationship between trait gratitude and risk of acute myocardial infarction. This is important as it provides evidence for trait gratitude playing a long-term protective role against cardiovascular strain caused by daily stress (Whittaker et al., 2021). Similarly, prior research had found that trait gratitude, unlike state gratitude, had been associated with increases in reactivity (Gallagher et al., 2021). This finding suggests that trait gratitude is associated with reduced adverse cardiovascular outcomes through reactivity, despite the associated increases (Whittaker et al., 2021).

Study Two investigated the relation- ship between state gratitude and cardiovascular reactivity and recovery within a within-subjects experimental framework. This study addressed the second and third aims of this research. Study Two demonstrated that state gratitude not only was associated with reduced systolic blood pressure reactivity but also expedited recovery, thereby enhancing the overall cardiovascular stress response profile. Moreover, it found that the balance of positive to negative emotions significantly interacts with state gratitude, amplifying its beneficial effects on the diastolic blood pressure stress response. This novel insight further evidences predictions that gratitude buffers the deleterious effects cardiovascular stress responses and highlights the amplification effect of positive emotions in conjunction with state gratitude on cardiovascular health.

Study Three, a randomized controlled trial, further corroborated the beneficial impact of state gratitude on cardiovascular health by demonstrating that a brief gratitude intervention could reduce systolic blood pressure reactivity in comparison to a control group. This finding is pivotal as it

confirms the causal relationship between state gratitude and reduced reactivity using best scientific practice in the form of a randomised controlled trial.

8.2 Contributions to theory

Applying Colquitt & Zapata-Phelan‘s (2007) framework, this thesis on gratitude and cardiovascular health significantly advances our theoretical understanding by acting as a “builder” and “tester”. According to Colquitt & Zapata-Phelan (2007), theory building is about creating new frameworks or models to explain phenomena, introducing new constructs, relationships, or enhancing existing theories. Theory testing involves evaluating these theories’ validity through empirical research. This thesis on gratitude and cardiovascular health can be seen as both building and testing theory. The three studies underscore gratitude’s role in modulating stress’s impact on cardiovascular re- activity, a mechanism not fully explained in prior research (Cousin et al., 2021). By integrating gratitude into the Transdisciplinary Model of Stress and the Model of Psychological Well-Being, this work not only tests but also expands existing theories, placing gratitude’s stress-mitigating effects at the forefront of cardiovascular health research.

This thesis builds on the existing Transdisciplinary Model of Stress (Epel et al., 2018) by adding gratitude as a psychosocial buffer and tests this addition through empirical studies, showing how gratitude affects cardiovascular reactivity and recovery, thereby contributing to both the advancement and validation of theoretical constructs within the domain of stress and cardiovascular health. The Transdisciplinary Model of Stress suggests that large magnitude responses to stress and delayed recovery, collectively comprising maladaptive stress responses, impact the cardiovascular system through wear and tear (Epel et al., 2018). For example, over time, chronic, maladaptive stress responses can lead to the deterioration of the cardiovascular system through endothelial dysfunction and subsequent platelet activation (Zupancic, 2009), ultimately contributing the rupturing of vulnerable atherosclerotic plaques, leading to myocardial infarction (Osborne et al., 2020). However, the Transdisciplinary Model of Stress also posits that there are psychosocial buffers of stress, which can play protective roles (Epel et al., 2018). This thesis extends this by providing empirical support for gratitude acting as a psychosocial buffers

against the more harmful effects of stress. Studies Two and Three both found that state gratitude reduced the stress response, and study two also found hastened recovery. Study one connected all this together to show that trait gratitude – an index of frequency of state gratitude experiences (Wood et al., 2010) – has an indirect effect on risk of acute myocardial infarction through heart rate reactivity.

Similarly, this thesis tests the Model of Positive Psychological Well-Being (Boehm, 2021; Schache et al., 2019) by empirically investigating gratitude's role within this framework. The Model of Psychological Well-Being predicts that positive emotions will reduce stress, thus reducing deteriorative cardiovascular processes (Boehm & Kubzansky, 2012). This investigation not only validates the model's prediction that positive emotions, specifically gratitude, can mitigate stress and its deleterious effects on cardiovascular health but also extends the model by specifically adapting it to include gratitude. The studies in this thesis uniquely contribute to this literature by connecting trait gratitude to risk of acute myocardial infarction, and demonstrating under con- trolled, laboratory conditions that state gratitude and gratitude interventions can reduce cardiovascular stress responses. Taken together, this suggests that gratitude, regardless of the forms (trait, state or intervention), plays an important stress-buffering role that ultimately plays a protective role in terms of cardiovascular health, as predicted by the Model of Psychological Well-Being. This dual contribution significantly enriches our understanding of the intricate interplay between psychological well-being and physical health.

The studies in this thesis contribute to research on cardiovascular reactivity. Recent theory has highlighted that both too low and too high reactivity result in negative outcomes (Whittaker et al., 2021). This thesis builds upon existing theories by exploring the complex, potentially curvilinear relationship between cardiovascular reactivity and health outcomes. While no paper has demonstrated the existence of a curvilinear relationship between reactivity and health (such that both low and high reactivity are associated with low health, and medium reactivity is associated with higher health), Study One was able to show that despite trait gratitude being associated with increases in reactivity, it nonetheless indirectly reduced risk of heart attacks. This suggests support

for the current model of thinking. Although the neural mechanisms underlying individual differences in reactivity remain poorly understood and constitute a likely area of further research (Gianaros et al., 2017).

This research has implications for the use of gratitude interventions in clinical cardiology. In the context where preventative cardiology has set goals not simply to reduce risk of disease, but to also increase healthy longevity, gratitude interventions have an important role to play (Kubzansky et al., 2018). To improve cardiovascular health in the population, a multitude of methods are necessary, including psychological interventions from the promotion of health (Knapper et al., 2015). Gratitude interventions are typically low-cost, easy to deploy, and generally well-liked by candidates (Wood et al., 2010). Where before we had no insights into long-term benefits, Study One presents pivotal findings of increasing trait levels of gratitude on long-term outcomes of cardiovascular health.

An important finding has been that the effect sizes have typically been small. While effect sizes for indirect relationships are typically smaller (Braveman et al., 2011), taken together, this has im- plications for further research in this domain. Although more research is necessary, when considering population-level interventions, small effect interventions can have large impacts depending on rarity of the negative outcome and how broadly the intervention is implemented (Matthay et al., 2021). For example, a small effect size (standardized mean difference of 0.2) can correspond to a reduction of 18% for rare outcome (1%) like acute myocardial infarctions *if* the intervention is broadly used (Matthay et al., 2021). However, gratitude interventions may have larger effect sizes than found the studies in this thesis due to duration of treatment in Study Three (Davis et al., 2016), and measurement issues in Study One. Consistent engagement in gratitude practices is advocated for sustained benefits, underlining the need for further research to elucidate optimal durations for these interventions (Wood et al., 2010). This is not to say that all one needs are some positive psychological interventions to increase cardiovascular health. Rather, psychological interventions should form part of a comprehensive, psycho-medical combined approach to improve cardiovascular health, including modifications to government policy, public health

initiatives, workplace interventions and media campaigns among others (Knapper et al., 2015).

However, understanding gratitude intervention effectiveness in this context requires understanding how and when gratitude interventions are effective (Briner & Walshe, 2015; O'Connell et al., 2017). The Positive Activity Model suggests that person features such as personality and demographics are important for the effectiveness of positive psychology interventions as well as dosage and variety (Lyubomirsky & Layous, 2013). For example, for people to benefit from any positive activity, they need engage with it (Lyubomirsky et al., 2011), have the motivation to be happier (Lyubomirsky et al., 2011), and believe that their work will result in benefits (Layous et al., 2013). However, more research is needed in this domain, particularly with regard to implementing community-level interventions (Montiel et al., 2021). For example, are there ceiling effects and how long should interventions be to produce stable changes in trait gratitude?

Interestingly, Study Two makes a novel contribution to the Positive Activity Model. One of the suggestions of this model is that people low in positive affect or higher in depression may benefit more from positive activity as they may have more room to benefit (Lyubomirsky & Layous, 2013). However, study two in this thesis found that experiencing more positive affect than negative affect amplified the effects of state gratitude on reductions in the cardiovascular response to stress. While this was not a study using a gratitude intervention, it does suggest that there may be an amplification effect, which is in line with suggestions that positive emotions can beget upward spirals which lead to optimal functioning (Garland et al., 2010; O'Connell et al., 2016). Future research should further investigate whether gratitude leads to upward spirals in social resources as a mechanism facilitating the improvement of cardiovascular health.

The findings in this thesis are also commensurate with the effects of other positive constructs which have been found to have relationships with cardiovascular health. For example, optimism is a well-studied (Alarcon et al., 2013) positive construct defined as a disposition towards the appraisal that positive events will happen in the future (Hernandez et al., 2020). A meta-analysis of 83 studies examining the relationship between optimism found it had many significant effects on various health outcomes such as immune functioning and all-cause mortality (Rasmussen et al.,

2009). Similarly, meta-analytic evidence demonstrates that optimism is associated with lowered risk of suffering cardiovascular events like strokes or acute myocardial infarctions (Rozanski et al., 2019). Future research could examine whether combining gratitude interventions with other positive interventions results in stronger stress buffering effects on cardiovascular reactivity and recovery.

A noteworthy finding in this thesis are the different effects of trait and state gratitude on cardiovascular reactivity. Trait gratitude was found to be associated with an increase cardiovascular reactivity and state gratitude was associated with a decrease. This also supports previous research, which similarly found that state and trait gratitude have differential effects (Gallagher et al., 2020, 2021). One possible explanation is that, in Study One, use was made of an older sample which can be more susceptible to conditions where the cardiovascular system cannot muster a sufficient response commensurate with the stressor to which an individual is exposed, such as chronotropic incompetence (Brubaker & Kitzman, 2011). However, this does not account for the findings of Gallagher et al. (2021) who made use of a much younger sample and found that trait gratitude was associated with an increase in reactivity.

Similarly, Gallagher et al. (2021) suggest that effort may also act as an explanation here. For example, previous experimental research has found that happiness primes led to increased cardiovascular reactivity in a difficult task compared with an easier task (Gendolla, 2012). In line with this, trait gratitude may increase engagement, thus increasing effort and cardiovascular responses. However, Gallagher et al. (2021) concede that in their study they found no relationship between trait gratitude and engagement. It would also be unclear why state gratitude would not increase engagement in these conditions, also resulting in heightened cardiovascular reactivity.

Potentially, one explanation is that individuals with higher trait gratitude may have less opportunity to experience acutely stressful events, due to factors such as higher social support and satisfaction with life (Wood et al., 2010). Thus, acute stress may constitute a greater contrast in the lives of individuals with higher trait gratitude and subsequently a higher cardiovascular response, as the acute stress contrasts with their expectations (Wilson & Gilbert, 2005). Previous research has found that repeated exposure to stress results in a habituation effect wherein the stress response is dampened (Grissom & Bhatnagar, 2009; Peters & McEwen, 2015). For example, chronic stress predicts smaller neural acute stress responses (Giglberger et al., 2023), and trait

gratitude itself is associated with more infrequent stress (Lee et al., 2018). Similarly, higher trait gratitude is associated with effective long-term coping strategies, such as seeking social support and maintaining a positive outlook (Davis et al., 2016). However, these strategies might not always be immediately accessible or effective in the face of sudden, acute stressors, with research indicating that social support is also associated with higher reactivity without the presence of an ally in the room (Christenfeld & Gerin, 2000; Gallagher et al., 2021). While trait gratitude should index a lower threshold for the experience of state gratitude, the correlation between the two is modest (Ginty et al., 2021). Hence, during acute stress, the immediate benefits of state gratitude may not fully translate for those high in trait gratitude due to their potentially underdeveloped exposure to acute stress.

This could explain the contrary findings, where high trait gratitude is associated with increased cardiovascular reactivity, and state gratitude, being an immediate and context-specific emotional experience, provides a calming effect that reduces cardiovascular reactivity during stress. These contrary findings could suggest that the mechanisms underlying the effects of trait and state gratitude on cardiovascular reactivity are distinct. Trait gratitude may enhance overall well-being and reduce exposure to chronic stress (Cregg & Cheavens, 2021), but may lead to more noticeable reactivity during acute stress due to lessened habitation to stress. In contrast, state gratitude may provide immediate emotional regulation benefits, reducing the physiological impact of acute stress.

Similarly, it is possible that what is being indexed is a relationship between trait gratitude autonomic nervous system regulation (Behnke et al., 2022). Some studies have found relationships between gratitude and higher heart rate variability, an index of autonomic nervous system activity (Redwine et al., 2016). Individuals with higher heart rate variability potentially have greater cardiovascular fitness and as such may be more resilient to stress (Souza et al., 2021). While research on gratitude and heart rate variability is still only beginning, some recent research has found promising relationships between positive affect and higher heart rate variability (Beatton et al., 2024).

Consequently, what these increases in cardiovascular reactivity may represent is an adaptive reactivity response, meaning that individuals higher in trait gratitude may experience increased reactivity that is characterized by a healthy, efficient physiological response to stress, as Gallagher et al., (2021) point out. This may reflect the more recent claims that there is a “Goldilocks” zone

for reactivity between exaggerated reactivity and blunted reactivity (Turner et al., 2020; Whittaker et al., 2021). (Turner et al., 2020; Whittaker et al., 2021). For example, a recent ecological momentary assessment study examining psychological stress reactivity found a quadratic relationship between stress reactivity (measured psychometrically) and outcomes such as wellbeing and life satisfaction (Rush et al., 2024). As such, despite increases in reactivity, it is possible that trait gratitude is associated with increases in cardiovascular reactivity to healthy, functional levels.

A final consideration is that study one found an indirect relationship between trait gratitude and the risk of heart attack through heart rate reactivity. As such there must be something related specific to the relationship between reactivity and trait gratitude in this explanation. Heart rate reactivity declines in adults with age whereas blood pressure does not (Uchino et al., 2010). Potentially what study one finds is that this decline in heart rate reactivity is buffered by trait gratitude. Hence, for older participants, trait gratitude is associated with increases in reactivity and this results in better cardiovascular health outcomes. However, it would require further study to examine if such an indirect effect would present itself in a younger sample, and it is also worth recalling that the mechanisms underpinning the relationship between cardiovascular health outcomes and cardiovascular reactivity are not yet fully understood (Whittaker et al., 2021).

The studies in this thesis also contribute to the theoretical underpinnings of the benefits of positive emotions. Specifically, these studies offer novel evidence for the broaden-and-build theory (Fredrickson, 2004a). This posits that gratitude can both broaden cognitive awareness and build physical resources (Alkozei et al., 2018). Gratitude builds the psychological resources needed to cope with psychological stress, thus also building physical resources by reducing the effects of deteriorative processes on the cardiovascular system (Schache et al., 2019). In this case, the results of these studies showed how state gratitude reduced cardiovascular reactivity and hastened recovery, potentially leading to the cultivation of greater cardiovascular health over time. This thesis also contains the first study demonstrating an indirect relationship between trait gratitude and the risk of acute myocardial infarction through cardiovascular reactivity, suggesting that gratitude cultivates physical as well as psychological resources.

Overall, this thesis presents novel evidence regarding the stress buffering effects of gratitude

on cardiovascular responses to stress. Thus, as this thesis shows, state and trait gratitude have emerged as consistent predictors of cardiovascular reactivity across multiple studies. Its use of methodological rigour in its use of a randomised controlled trial helps overcome limitations of prior work in this area. It also offers the first novel evidence connecting gratitude to any long-term cardiovascular outcomes indirectly through reactivity. It is also offers the first novel evidence that gratitude hastens recovery from stress as well as reducing cardiovascular reactivity. Taken together, this has theoretical implications for the Transdisciplinary Model of Stress (Epel et al., 2018), as well as current models of how gratitude impacts health through the Model of Psychological Well- Being (Boehm & Kubzansky, 2012), suggesting reductions in reactivity to be a useful pathway in this regard.

8.3 Strengths and Limitations

An important criticism of positive psychology is that its results do not replicate and as a field it is not methodologically rigorous (Zyl et al., 2023). A key strength of this thesis is that in all cases the results replicated *and* extended the findings of prior studies, such as Gallagher et al. (2020), Ginty et al. (2021), and Gallagher et al. (2021). These prior studies made use of standardised stress testing protocols, which the studies in this thesis also made use of. Thus, in study one, a positive, statistically significant relationship was found between trait gratitude and cardiovascular reactivity across all examined parameters, like Gallagher et al. (2021). Studies two and three found significant, inverse relationships between cardiovascular reactivity and state gratitude, like Gallagher et al. (2020) and Ginty et al. (2021) found.

Another key strength of the studies in this thesis is its use of standardised protocols and a randomised control trial. The use of standardised stress-testing protocols followed best-practice and other research in this domain (Narvaez-Linares et al., 2020). By doing so, this research was able to control for many potential confounding variables associated with the assessment of cardiovascular parameters, such as movement, caffeine and nicotine consumption, as well as

effects associated with exercise and the consumption of food (Narvaez-Linares et al., 2020). Similarly, this thesis made use of objective biological measures, which aid the objectivity of the research and help to overcome biases of subjective reports of stress, thus delivering excellent science (Epel et al., 2018). In so doing, the studies in this thesis minimised error. Similarly, by making use of randomised con- trol trial, study three was able to leverage a powerful method to examine the impact of a gratitude intervention on cardiovascular reactivity. Finally, an important criticism of positive psychology suggest that it lacks methodological rigour and as such results do not replicate (Zyl et al., 2023). This thesis addresses this by making use of standardised stress-testing protocols throughout the three studies.

The use of mixed-effects growth curves were also a strength of this thesis in study two. It was a more parsimonious statistical method of testing the relationship between gratitude and cardiovascular reactivity and recovery (Curran et al., 2010). Utilizing these methods allowed us to test the entire stress response, which is both novel in this domain and simpler than alternate methods, allowing an insight into the entire stress process. The parsimony is *apropos* considering the alternative would be to utilize six hierarchical regressions, which would increase the error rate substantially.

Finally, the use of secondary data to connect trait gratitude to a negative cardiovascular out-come, acute myocardial infarction, through heart rate reactivity was a key contribution and strength of this work. While causal conclusions cannot be drawn, it demonstrates promising evidence for models such as the Model of Psychological Well-Being (Boehm, 2021) the Transdisciplinary Model of Stress (Epel et al., 2018).

A substantial limitation of this work was due to the impact of Covid-19. In March 2020, Ireland entered a period of lockdown that would last until September 2021 (Department of Health, 2022). Accordingly, all in-person research had to be suspended during this time and alternate plans arranged. This meant for this thesis that some projects had to be permanently suspended. Explicitly, study two had gathered 68 participants by March 2020. However, due to the lockdowns, data collection was entirely suspended by September 2021, and focus was placed on data collection

for study three.

The sample size was lower than optimal. While study one had a large overall sample, the number of participants in the group who suffered a heart attack was small. Similarly, the sample sizes for studies two and three were small. The power analyses were conducted prior to Gallagher et al. (2020) publishing their work, and it was theorised that gratitude would have a modest effect on reactivity (e.g. Davis et al., 2016). Similarly, while study one made use of MIDUS data which constitutes a representative sample of US citizens, studies two and three made use of convenience sampling, limiting the representativeness of those samples (Easterby-Smith et al., 2021).

Another limitation of this thesis was that the intervention may not have been potent enough or long enough. The Positive Activity Model (Lyubomirsky & Layous, 2013) suggests that intervention efficacy depends on dosage. This is supported by meta-analytic evidence finding that gratitude interventions have stronger effects if deployed over a longer time period (Davis et al., 2016). The intervention utilized in study three was brief, lasting only eight minutes. While the results of study three suggest that there can be benefits from a brief gratitude intervention, if completed over a longer period of time, it is likely the benefits would be greater. Similarly, the impact of gratitude interventions on stress reactivity needs to be replicated in a natural context. In this context, smart watches may represent a very useful data collection tool (Nelson et al., 2020).

8.4 Future directions

The exact mechanisms explaining how gratitude buffers against the negative effects of cardiovascular stress responses are not fully elucidated. Future research could focus on areas such as positive reframing, whereby individuals reinterpret adverse experiences more positively, thus modulating the stress experience and reducing depressive symptoms (Alkozei et al., 2018; Kurian & Thomas, 2023; Lambert et al., 2012). Future research could also focus on Fredrickson's Broaden and Build theory (Fredrickson, 2004a; Fredrickson, 2004b), which suggest that gratitude can foster cognitive adaptability and resourcefulness, which in turn can dampen the impact of stress on

health. Similarly, gratitude enhances social support, which is related to improved health behaviors and stronger social ties, thus preventing adverse interactions between stress reactivity and disease (Algoe, 2012; Alkozei et al., 2018; Harvey & Alexander, 2012; Uchino, 2006). These are potential areas for further research into *how* gratitude buffers the response to stress.

Similarly, it would be useful for research to utilize a greater variety of cardiovascular parameters; indeed, combining multiple cardiovascular assessments may lead to more stable connections between risk factors, protective factors and disease outcomes (Whittaker et al., 2021). In this vein, further research into gratitude and variables such as cardiac output and total peripheral resistance, as well as through the use of composite measures, is warranted. This is because heart rate and blood pressure index both sympathetic and parasympathetic nervous system activity (Shiota et al., 2014), whereas total peripheral resistance and cardiac output constitute more specific measures of sympathetic and parasympathetic nervous system activity (Trammel & Sapra, 2023). In this con- text, heart rate variability could also be measured, as it can be decomposed into explicit indices of nervous system activity (Thayer & Lane, 2009; Viljoen & Claassen, 2017).

One criticism of laboratory-measured reactivity is its ecological and environmental validity. Alternate methods include ecological momentary assessment, which involves repeatedly sampling participants in real-time in their natural environments (Shiffman et al., 2008). For example, ambulatory blood pressure monitors are often used in clinical settings (Turner et al., 2015), and ambulatory electrocardiographs have been used to assess allostatic load in police (Vries et al., 2022). A feasibility study of thirty-five participants concluded that for heart rate variability, ecological momentary assessment was a useful method for studying reactivity (J. Yang & Kershaw, 2022). However, as such studies are effectively field studies (Easterby-Smith et al., 2021), they cannot control for factors such as the kind of time-frame and intensity of stress experienced (Gormally & Romero, 2020). Similarly, such ecological momentary assessment studies tend to be more com- plex and expensive to run (Epel et al., 2018). Nonetheless, utilizing ambulatory methods may be a fruitful direction for future research on gratitude and cardiovascular stress responses.

Similarly, there have been suggestions that multicomponent positive psychological interventions may be more potent than single-component interventions (Schotanus-Dijkstra et al., 2015). Afterall, well-being is multi-faceted (Fancourt & Finn, 2019), and as such it may be the case that it is more efficacious to promote interventions which contain a variety of evidence-based, individual exercises (Valiente et al., 2022). Meta-analytical evidence for this approach suggests that multi-component interventions combining individual positive psychological interventions are more effective than single-intervention exercises at reducing depression (Carr et al., 2021), and demonstrated moderate effects (Hedge's $g = 0.48$) on stress reduction (Hendriks et al., 2020). Carr et al. (2021) conclude in their meta-analysis that multi-component interventions are likely to be most useful in a clinical setting to maximise their efficacy. However, there remain a number of gaps such as intervention length and number of sessions, answers to which may reveal how multi-component positive psychological interventions may be more optimally deployed (Hendriks et al., 2020). Notably, there have been very few studies exploring how gratitude interventions operate in this context (Cousin et al., 2021) or if multi-component positive psychological interventions impact cardiovascular health (Magán et al., 2022). Future research could explore how gratitude, partnered with other interventions such as optimism may yield stronger reductions in stress reactivity.

Future research could utilise longer gratitude interventions. Studies with up to six weeks of gratitude journaling have yielded large effects in prior research (Davis et al., 2016). Future research could leverage this to examine if gratitude may yielded more pronounced protective effects. Similarly, it is critical to know when effects level off or if there are boredom effects. In this vein, future research could also connect gratitude to cardiovascular outcomes such as hypertension through further longitudinal research. It would also be useful to more explicitly test the relationship between gratitude, social support, stress, and cardiovascular reactivity using a longitudinal design (Algoe, 2012; Schache et al., 2019).

Finally, future research could explore the potential curvilinear relationship between health and reactivity (Whittaker et al., 2021). As both blunted and exaggerated reactivity are associated with negative health outcomes (Balanos et al., 2010; Phillips et al., 2011; Phillips et al., 2013) it

would be useful to examine if this relationship is curvilinear, or if there are certain thresholds distinguish- ing blunted and exaggerated reactivity from healthy reactivity. While this research is currently fledgling (O' Riordan et al., 2022), it warrants additional attention.

8.5 Conclusion

While stress cannot be avoided, our responses to it have important future health implications. This thesis highlights the potential for gratitude to positively impact cardiovascular health by buffering the deleterious effects of stress on the body. In order to globally reduce the burden of cardiovascular illness, comprehensive multidisciplinary interventions will be needed. While more work is needed to study the effects of longer interventions and generate stronger evidence, this thesis constitutes contribution to this research. This thesis extends current knowledge by demonstrating how trait gratitude is longitudinally associated with a decreased risk of acute myocardial infarction, how state gratitude is associated with both reactivity and recovery, and has demonstrated support for a causal connection between state gratitude and reactivity. This thesis thus provides another stepping stone for future research on gratitude and the cardiovascular response to stress.

In closing, health psychology constitutes rigorous evidence that the human being is a psychophysical unity. It is clear that emotions, behaviours, and judgments play critical roles in the determination of physical health. In the case of this thesis, the thanks we give and the gratitude we feel not only has some moral virtue, but also plays an important health-protective role.

Appendix A

Study one supplementary material

0-1: Table A.1: Table of correlations for main variables of interest

Variable	1	2	3	4	5	6	7	8	9	10	11
1. Trait gratitude											
2. SBP reactivity	.08*										
3. DBP reactivity	.09*	.80**									
4. HR reactivity	.09**	.28**	.37**								
5. Age	.07*	.24**	.19**	-.05							
6. Sex	-.14**	.07	-.03	-.04	.02						
7. BMI	-.00	.04	-.01	-.06	-.04	.05					
8. Education	.04	-.01	-.08*	.01	-.08*	.11**	-.10**				
9. Diabetes	-.03	-.04	-.08	-.02	.07*	.03	.15**	-.04			
10. Number of chronic conditions	-.07*	-.07	-.09*	-.08*	.06	-.19**	.18**	-.04	.17**		
11. Ever smoke	-.08*	-.08*	-.05	-.10**	.10**	.06	-.01	-.12**	-.02	.08*	
12. High blood pressure	-.01	.18**	.10*	-.09*	.27**	-.02	.25**	-.00	.20**	.31**	0.2

Note. * $p < .05$. ** $p < .01$. SBP = Systolic blood pressure (mmHg), DBP = Diastolic blood pressure (mmHg), HR = Heart rate (BPM), N ranges from 660 to 912.

0-2: Table A.2: Results of supplementary single mediation models

Mediator	Summary	Coeff	SE	LLCI	ULCI
SBP reactivity	Total	-0.233	0.311	-0.748	0.536
SBP reactivity	Total indirect	-0.066	0.046	-0.199	-0.009
SBP reactivity	Direct	-0.168	0.312	-0.646	0.628
DBP reactivity	Total	-0.238	0.317	-0.722	0.556
DBP reactivity	Total indirect	-0.047	0.043	-0.181	0.005
DBP reactivity	Direct	-0.191	0.322	-0.677	0.657
HR reactivity	Total	-0.245	0.339	-0.745	0.608
HR reactivity	Total indirect	-0.105	0.077	-0.322	-0.015
HR reactivity	Direct	-0.141	0.360	-0.698	0.705

Note. SBP = Systolic blood pressure (mmHg), DBP = Diastolic blood pressure (mmHg), HR = Heart rate (BPM). LLCI: bootstrapped lower-level confidence interval; ULCI: bootstrapped upper-level confidence interval. The indirect effect is significant if the confidence interval does not contain zero.

0-3: Table A.3: Model Coefficients for the Parallel Mediation Model with Three Mediators and controlling for positive affect

	Consequent											
	Parallel mediator									Outcome		
Antecedent	SBP reactivity			DBP reactivity			HR reactivity			Myocardial infarction		
	Coeff	SE	P	Coeff	SE	P	Coeff	SE	P	Log odds	SE	P
Positive affect	0.13	0.04	0.001	0.13	0.04	0.001	0.04	0.04	0.25	0.10	0.04	0.81
Trait gratitude	0.05	0.04	0.19	0.05	0.04	0.25	0.08	0.03	0.02	-0.14	0.09	0.65
SBP reactivity										-0.10	0.09	0.02
DBP reactivity										0.18	0.43	0.05
HR reactivity										-0.21	0.30	0.03
			R^2=0.02			R^2=0.023			R^2=0.011			R^2=0.41
			P=0.047			P=0.034			P=0.125			P<.001

Note. SBP = Systolic blood pressure (mmHG), DBP = Diastolic blood pressure (mmHG), HR = Heart rate (BPM)

0-4: Table A.4: Results of mediation analysis predicting myocardial infarction: indirect relationships between trait gratitude and myocardial infarction through three reactivity measures

			BC 95% CI	
	Estimate	SE	Lower	Upper
Indirect effects		Predictor: Trait gratitude		
Total indirect effect	-0.106	0.067	-0.266	0.003
Unique effects:				
1. Systolic blood pressure reactivity	-0.064	0.059	-0.224	0.020
2. Diastolic blood pressure reactivity	0.042	0.044	-0.019	0.157
3. Heart rate reactivity	-0.084	0.060	-0.233	-0.007

Note. BC 95% CI refers to the bias-corrected 95% confidence interval using 1,000 bootstrap samples. All estimates are reported in log-odds scale; estimates with CIs that do not include zero are statistically significant and bolded.

0-5: Table A.5: Model Coefficients for the Parallel Mediation Model with Three Mediators and Covariates including positive affect and depression

	Consequent											
	Parallel mediator									Outcome		
Antecedent	SBP reactivity			DBP reactivity			HR reactivity			Myocardial infarction		
	Coeff	SSE	P	Coeff	SE	P	Coeff	SE	P	Log odds	SE	P
Depression	-0.10	0.04	0.04	-0.12	0.04	.002	-0.08	0.04	.042	0.04	.07	0.54
Trait gratitude	0.05	0.04	0.07	0.05	0.05	.24	0.07	0.03	.047	-0.04	.28	0.88
SBP reactivity										-0.09	.04	0.04
DBP reactivity										0.16	0.09	0.08
HR reactivity										-0.21	0.09	0.02
	R^2=0.017			R^2=0. 022			R^2=0.014			R^2=0.37		
	P = 0.089			P = 0.051			P = 0.086			P= 0.002		

Note. SBP = Systolic blood pressure (mmHG), DBP = Diastolic blood pressure (mmHG), HR = Heart rate (BPM).

0-6: Table A.6: Results of mediation analysis predicting myocardial infarction: indirect relationships between trait gratitude and myocardial infarction through three reactivity measures while controlling for depression.

			BC 95% CI	
	Estimate	SE	Lower	Upper
Indirect effects	Predictor: Trait gratitude			
Total indirect effect	-0.093	0.061	-0.238	0.013
Unique effects:				
4. Systolic blood pressure reactivity	-0.059	0.041	-0.199	0.030
5. Diastolic blood pressure reactivity	0.037	0.052	-0.019	0.139
6. Heart rate reactivity	-0.071	0.278	-0.214	-0.004

Note. BC 95% CI refers to the bias-corrected 95% confidence interval using 1,000 bootstrap samples. All estimates are reported in log-odds scale; estimates with CIs that do not include zero are statistically significant and bolded.

Appendix B

Psychometric measures of state gratitude, trait gratitude and affect

B.1 Gratitude adjective checklist (GAC)

Instructions: Think about how you [felt yesterday/have felt during the past few weeks]. Using a scale from 1 (not at all), 2 (a little), 3 (moderately), 4 (quite a bit), to 5 (extremely), please choose a number to indicate your level of feeling the following:

_____ 1. Grateful

_____ 2. Thankful

_____ 3. Appreciative

Scoring: Sum responses to the 3 items.

Taken from McCullough et al. (2002). \

B.2 The gratitude questionnaire (GQ6)

Instructions: Using the scale below as a guide, write a number beside each statement to indicate how much you agree with it. 1 = strongly disagree 2 = disagree 3 = slightly disagree 4 = neutral 5 = slightly agree 6 = agree 7 = strongly agree

_____ 1. I have so much in life to be thankful for.

_____ 2. If I had to list everything that I felt grateful for, it would be a very long list.

_____ 3. When I look at the world, I don't see much to be grateful for.

_____ 4. I am grateful to a wide variety of people.

_____ 5. As I get older I find myself more able to appreciate the people, events, and situations that have been part of my life history.

_____ 6. Long amounts of time can go by before I feel grateful to something or someone.

Taken from McCullough et al. (2002).

B.3 Positive and negative affect schedule (PANAS-SF)

0-7: Table B.1: Positive and Negative Affect Schedule (PANAS-SF)

	Indicate the extent you have felt this way over the past week.				
	Very slightly or not at all	A little	Moderately	Quite a bit	Extremely
PANAS 1	□	□	□	□	□
PANAS 2	□	□	□	□	□
PANAS 3	□	□	□	□	□
PANAS 4	□	□	□	□	□
PANAS 5	□	□	□	□	□
PANAS 6	□	□	□	□	□
PANAS 7	□	□	□	□	□
PANAS 8	□	□	□	□	□
PANAS 9	□	□	□	□	□
PANAS 10	□	□	□	□	□
PANAS 11	□	□	□	□	□
PANAS 12	□	□	□	□	□
PANAS 13	□	□	□	□	□
PANAS 14	□	□	□	□	□
PANAS 15	□	□	□	□	□
PANAS 16	□	□	□	□	□
PANAS 17	□	□	□	□	□
PANAS 18	□	□	□	□	□
PANAS 19	□	□	□	□	□
PANAS 20	□	□	□	□	□

Scoring: Positive Affect Score: Add the scores on items 1, 3, 5, 9, 10, 12, 14, 16, 17, and 19. Scores can range from 10 – 50, with higher scores representing higher levels of positive affect. Mean Scores: 33.3 (SD±7.2) Negative Affect Score: Add the scores on items 2, 4, 6, 7, 8, 11, 13, 15, 18, and 20. Scores can range from 10 – 50, with lower s

Taken from Watson et al. (1988).

Appendix C

Study two protocol

Protocol Overview

Participant ID number: **Date & Time:**

Dim Lights. Code all questionnaires being used.

Participant Arrival

"Hello, your name is (XXXX), is that right? My name is _____, and this is _____.

Welcome to the Health Lab! Thanks for coming in to us today."

"You can leave your things here (refer to empty table by door). Please have a seat over here. I'll just give you the Information Sheet to read through. Please let me know when you're finished."

Wait until participant has read information sheet

Do you have any questions? Would you like to attach the electrodes yourself, or will I do it"

Address questions if necessary.

"Please read this sheet as well, and if you're okay with everything, then please sign it at the end."

Provide consent form & collect once signed

"I will talk you through the blood pressure and the tasks shortly. Before we begin, I need to check a few things. Have you."

Checklist		
Consumed alcohol in the last 12 hours?	Yes	No
Participated in vigorous exercise in the last 12 hours?	Yes	No
Smoked in the last 2 hours?	Yes	No
Consumed caffeine in the last 2 hours?	Yes	No
Eaten in the last hour?	Yes	No
Also, are you right-handed or left-handed?	Right	Left

"Great, now, can I ask you to step here so that I can measure your height and weight?"

Height (cm):_____ Weight (kg):_____

"Thank you! You can take a seat again."

"Today, we are going to take some blood pressure measurements from you while you complete a task in the lab."

Answer any queries.

"Now, we will proceed. Please place your feet on the box and keep them there throughout the study. This is to control for movement which might affect the readings. During the session please do not speak unless you're asked to, as this could alter the blood pressure readings. I will explain what is expected as we go along and I can answer any questions you have at the end. Is that OK?"

"This blood pressure cuff applies pressure to the arteries in the arm for the purpose of measuring blood pressure. I am going to wrap the cuff around your upper non-dominant arm at the level of the heart. At several times throughout the session the cuff will be inflated, so that we can get the blood pressure reading. After it inflates and we have a reading it will start to deflate immediately."

"The cuff can get a little tight, but the discomfort will subside. We will run a practice measurement so that you can see how it feels. Please keep your arm straight and on the desk while measurements are being taken."

Attach sleeve to non-dominant arm, tubes placed on the upper side of arm and run practice measurement.

"Was that alright? If the cuff is quite uncomfortable, I can adjust if for you now."

Adjust cuff if necessary

"If you feel uncomfortable again please let me know. Otherwise, to make sure we have accurate measurements, it would be helpful if you could remain silent during the tasks."

[**Resting Period**]

"I have a few questionnaires for you that I'd like you to complete. You will have ten minutes to do that, but if you take longer that's no problem – you can finish them at the end. If you're done early, you can take a look at the magazine. We will take a few blood pressure measurements throughout. In about five minutes, I will ask you to remove the device; you just need to unclip it. This is so I can perform a signal test."

Provide Demographics, ERP-R, TMMS, MEQ-SA, TIPI, GQ-6, GAC.

Set timer for 10 minutes & take BP readings (five readings at 2 min intervals)

Take away all completed questionnaire.

[**Stress Task**]

[**Task description and pre-task survey**]

I'm going to now describe the tasks you will complete and then you will fill in the pre-task survey on the next page. You will complete a maths task where you will sequentially subtract numbers mentally and report your answers aloud. Please, fill in the small survey on the next page.

- When that's done, do the next part.*

"During the final six-minute math portion of this task you will be asked to sequentially subtract the number 13 from 1,022 in your head. You will verbally report your answers aloud and be asked to start over from 1,022 if a mistake is made. Your time starts now."

Record physiological measures every 2 minutes beginning at time 0. After two minutes, read the following:

"The stress task is over. You will now have a few minutes to complete the questionnaires you didn't have time to finish at the beginning of the study. Do not speak during this time. I will tell you when it is ok for you to leave and when the study is complete. In the mean-time, if you have completed the questionnaires, you can take a look at the magazine."

- Set timer for ten minutes (Five BP readings at 2 min intervals)*
- If not completed earlier, provide ERP-R, TMMS, MEQ-SA, TIPI, GQ-6 as necessary*

Collect all questionnaires

Take off blood pressure cuff and electrodes.

[**Debriefing:**]

"Thank you very much for participating!"

"I just want to let you know that the tasks you were asked to do, was unreasonably difficult and won't be showing to anybody. We didn't even record it. They do not reflect how good you are at maths or public speaking."

"This sheet contains some more information about this study. Do you have any questions?"

Provide debriefing sheet. Address questions.

Provide info sheet about hypertension if applicable.

“Thanks again for participating!”

Appendix D

Study three protocol

Protocol Overview

Participant ID number: **Date & Time:**

Dim Lights. Code all questionnaires being used.

Participant Arrival

"Hello, your name is (XXXX), is that right? My name is _____, and this is _____.

"Welcome to the Health Lab! Thanks for coming in to us today."

"You can leave your things here (refer to empty table by door). Please have a seat over here. I'll just give you the Information Sheet to read through. Please let me know when you're finished."

Wait until participant has read information sheet

Do you have any questions? Would you like to attach the electrodes yourself, or will I do it"

Address questions if necessary.

"Please read this sheet as well, and if you're okay with everything, then please sign it at the end."

Provide consent form & collect once signed

"I will talk you through the blood pressure and the tasks shortly. Before we begin, I need to check a few things. Have you….."

Checklist		
Consumed alcohol in the last 12 hours?	Yes	No
Participated in vigorous exercise in the last 12 hours?	Yes	No
Smoked in the last 2 hours?	Yes	No
Consumed caffeine in the last 2 hours?	Yes	No
Eaten in the last hour?	Yes	No
Also, are you right-handed or left-handed?	Right	Left

"Great, now, can I ask you to step here so that I can measure your height and weight?"

Height (cm):_____ Weight (kg):_____

"Thank you! You can take a seat again."

"Today, we are going to take some blood pressure measurements from you while you complete a task in the lab."

Answer any queries.

"Now, we will proceed. Please place your feet on the box and keep them there throughout the study. This is to control for movement which might affect the readings. During the session please do not speak unless you're asked to, as this could alter the blood pressure readings. I will explain what is expected as we go along and I can answer any questions you have at the end. Is that OK?"

"This blood pressure cuff applies pressure to the arteries in the arm for the purpose of measuring blood pressure. I am going to wrap the cuff around your upper non-dominant arm at the level of the heart. At several times throughout the session the cuff will be inflated, so that we can get the blood pressure reading. After it inflates and we have a reading it will start to deflate immediately."

"The cuff can get a little tight, but the discomfort will subside. We will run a practice

measurement so that you can see how it feels. Please keep your arm straight and on the desk while measurements are being taken."

Attach sleeve to non-dominant arm, tubes placed on the upper side of arm and run practice measurement.

"Was that alright? If the cuff is quite uncomfortable, I can adjust if for you now."

Adjust cuff if necessary

"If you feel uncomfortable again please let me know. Otherwise, to make sure we have accurate measurements, it would be helpful if you could remain silent during the tasks."

[**Resting Period**]

"I have a few questionnaires for you that I'd like you to complete. You will have ten minutes to do that, but if you take longer that's no problem – you can finish them at the end. If you're done early, you can take a look at the magazine. We will take a few blood pressure measurements throughout. In about five minutes, I will ask you to remove the device; you just need to unclip it. This is so I can perform a signal test."

Provide Demographics, ERP-R, TMMS, MEQ-SA, TIPI, GQ-6, GAC.

Set timer for 10 minutes & take BP readings (five readings at 2 min intervals)

Take away all completed questionnaire.

[**Intervention Period**]

"This is the writing exercise component of the task. Please read the instructions on the sheet overleaf and carry out the writing task as instructed. You have ten minutes to complete the task."

Record 5 bp measures.

[**Stress Task**]

[**Task description and pre-task survey**]

I'm going to now describe the tasks you will complete and then you will fill in the pre-task survey on the next page. In the next part of the experiment, you will be asked to prepare and give a speech on your best and worst characteristics. Following this you will complete a maths task where you will sequentially subtract numbers mentally and report your answers

aloud. Please, fill in the small survey on the next page.

When that's done, do the next part.

"This is the speech preparation portion of the task; you will prepare and give a speech. You will have two minutes to **mentally** prepare and four minutes to give the speech. The speech is about your best and worst characteristics. During the speech you will talk about three of your best and three of your worst characteristics. With each characteristic you must provide an example of a real-life situation that provides a clear illustration. It is very important that you use the entire four minutes for your speech and that you give a clearly pronounced, convincing speech as it will later be scored and judged by experts in presentations. You now have two minutes to prepare your speech.

Record physiological measures every 2 minutes beginning at time 0. After two minutes, read the following:

"This is the speech portion of the task. You are to deliver a speech describing three of your best and worst characteristics. You should speak for the entire 4-minute period. Your time starts now."

The prop camera should now be turned on. Continue to record physiological measures. If the participants stops talking during the speech, allow them to remain silent for 20 seconds. If they do not resume speaking, prompt the participant to continue speaking by instructing them: "You still have time remaining.".

At the end of the 4-minute speech performance period, read the following script to the participant: "Thank you for your speech. During the final six-minute math portion of this task you will be asked to sequentially subtract the number 13 from 1,022 in your head. You will verbally report your answers aloud and be asked to start over from 1,022 if a mistake is made. Your time starts now."

"The stress task is over. You will now have a few minutes to complete the questionnaires you didn't have time to finish at the beginning of the study. Do not speak during this time. I

will tell you when it is ok for you to leave and when the study is complete. In the meantime, if you have completed the questionnaires, you can take a look at the magazine."

Set timer for ten minutes (Five BP readings at 2 min intervals)

- If not completed earlier, provide ERP-R, TMMS, MEQ-SA, TIPI, GQ-6 as necessary*

Collect all questionnaires

Take off blood pressure cuff and electrodes.

[**Debriefing:**]

"Thank you very much for participating!"

"I just want to let you know that the tasks you were asked to do, was unreasonably difficult and won't be showing to anybody. We didn't even record it. They do not reflect how good you are at maths or public speaking."

"This sheet contains some more information about this study. Do you have any questions?"

Provide debriefing sheet. Address questions.

Provide info sheet about hypertension if applicable.

"Thanks again for participating!"

Appendix E

Study three interventions

E.1 Gratitude intervention

Written exercise instructions

Think of someone in your life who you feel like you have never fully or properly thanked for something meaningful or important that they did for you. In the space provided below and on the next page, please write a note to this person that describes why you feel like you never properly thanked them and letting them know why you feel thankful for something important that they did for you. Though this letter will not actually be sent to this person and is simply an exercise for you, please use this as an opportunity to really explore those feelings surrounding how you feel about what they have done for you and write honestly and openly from your heart. You have eight minutes.

E.2 Control intervention

Written exercise instructions

Think of the room you are in. . . . In the space below and on the next page, please provide a description of the room you are in. Try to focus on the specific elements in the room, the colours of the wall or objects in the room. Use this writing session as an opportunity to paint a detailed picture of this room and include as much specific information as you can. You have eight minutes.

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