

Socially Relevant Personality Traits and Attenuated Cardiovascular Functioning

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Abstract

Introduction. Previous research has implicated certain personality traits in the aetiology of cardiovascular disease. One common theme in such research is that traits that impinge upon social interactions and relationships (e.g., hostility, Type A) may be especially relevant. Several patterns of cardiovascular reactivity have been found to be associated with poor future health outcomes, including exaggerated and diminished reactivity, and delayed recovery after stress. The present research examines the influence of individual differences in socially oriented traits on patterns of cardiovascular functioning in different contexts.

Methods. Three empirical studies are reported. In a sample of 80 participants (60 women, 20 men), Study 1 examines the influence of broad affective personality characteristics using the NEO Five-Factor inventory, including neuroticism and extraversion subscales. Study 2 examined cardiovascular functioning during exposures to social and asocial forms of stress in a laboratory session. From a sample 94 participants (67 women, 37 men), a subsample of 44 women were included in the final analyses. Study 3 examined the effects of trait dominance on cardiovascular responses to repeated social stress exposures for 75 participants (54 women, 21 men).

Results. Study 1 found that affective personality traits (neuroticism and extraversion) were associated with diurnal patterns of cardiovascular functioning. Study 2 found that lower trait dominance was associated with exaggerated cardiovascular functioning during social, but not asocial forms of stress, relative to those higher in trait dominance. Study 3 found that blunted patterns of cardiovascular functioning for those higher in trait dominance were associated with vascular profiles of hemodynamic cardiovascular responding, indicative of future ill-health.

Conclusions. In sum, it was found that blunted patterns of cardiovascular functioning were associated with individual differences in personality traits related to interpersonal trait affectivity, including neuroticism, extraversion, and trait dominance. The research confirms some recent work that has established blunting as a negative cardiovascular trend, associated with aspects of cardiovascular functioning that are related to future disease risk. Additionally, individual differences in dominance were found to be associated with the identification of these potentially maladaptive physiological trends.

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List of Acronyms

ANOVA: Analysis of variance

ANCOVA: Analysis of Covariance

BMI: Body mass index

CAA: Coronary artery atherosclerosis

CAD: Coronary artery disease

CHD: Coronary heart disease

CO: Cardiac output

CVD: Cardiovascular disease

CVR: Cardiovascular reactivity

DBP: Diastolic blood pressure

HR: Heart rate

IBI: Inter-beat interval

IHD: Ischemic heart disease

JPRF: Jackson Personality Research Form

MAP: Mean arterial pressure

MI: Myocardial infarction

NA: Negative affectivity

NEO FFI: Neuroticism, Extraversion, Openness Five-Factor Personality Inventory

PASAT: Paced auditory serial addition task

PP: Pulse pressure

SBP: Systolic blood pressure

SV: Stroke volume

TABP: Type-A behaviour pattern

TPR: Total peripheral resistance

TSST: Trier Social Stress Task

List of Works

Below is a list of publications and conference presentations stemming directly from this thesis.

MANUSCRIPTS SUBMITTED

1. **Lee, E. M.** & Hughes, B.M. (under review). Personality differences in cardiovascular function during daytime waking hours. *Stress and Health*. (Chapter 2).
2. **Lee, E. M.** & Hughes, B.M. (under review). Trait dominance as a moderator of cardiovascular functioning during social stress. *Journal of Psychophysiology*. (Chapter 3).

PRESENTATIONS

1. **Lee, E. M.** & Hughes, B. M. (2012, July). *Cardiovascular reactivity to social and asocial stress: The association with trait dominance*. Paper presented at the 33rd International Conference of the Stress and Anxiety Research Society, Palma de Mallorca, Spain.
2. **Lee, E. M.** & Hughes, B. M. (2012, March). *Trait anxiety and normative blood pressure functioning using a self-monitoring protocol*. Paper presented at the 70th Annual meeting of American Psychosomatic Society, Athens, Greece.
3. **Lee, E. M.** & Hughes, B. M. (2011, October). *Individual differences in neuroticism: Associations with daily cardiovascular flux*. Paper presented at the Fifth International Conference on The (Non) Expression of Emotions in Health and Disease, Tilburg, The Netherlands.
4. **Lee, E. M.** & Hughes, B. M. (2011, June). *Hostility-related diurnal variations in blood pressure*. Poster session presented at the Cardiovascular Stress Reactivity: Antecedents and Consequences conference, Birmingham, UK
5. **Lee, E. M.** & Hughes, B. M. (2011, July). *Physiological consequences of neuroticism: A self-monitoring study*. Paper presented at the 32nd International Conference of the Stress and Anxiety Research Society, Münster, Germany.
6. **Lee, E. M.** & Hughes, B. M. (2011, March). *Influence of hostility on activity-related diurnal variations in blood pressure*. Poster presented at the 69th Annual meeting of the American Psychosomatic Society, San Antonio, Texas.
7. **Lee, E. M.** & Hughes, B. M. (2010, August). *Influence of hostility on work time-downtime variations in blood pressure*. Paper presented at the 31st International Conference of the Stress and Anxiety Research Society, Galway, Ireland.

Chapter 1: Introduction

Homeostasis and Physiological Functioning

First described as “milieu intérieur” by Bernard (1878), and later as “homeostasis” by Cannon (1915, 1932), homeostatic regulation refers to the ability of an organism to maintain physiological (and psychological) systems, and is encapsulated in the ability to return to a resting state once a disruptive challenge has passed. It may even refer to the necessary physiological limits of the organism, although the parameters and boundaries of such limits may change due to environmental conditions (McEwen & Wingfield, 2010). The critical relevance of the concept of homeostasis is the notion that homeostatic regulation can precipitate *both* healthful *and* health-damaging outcomes. In some circumstances, homeostasis will ensure that threats (be they toxins, environmental conditions, or other stimuli) do not exert persistently negative effects on the human body, but will instead allow the body to readjust in ways that ameliorate the dangers posed. However, in other circumstances, homeostasis will end up disrupting normal bodily function in ways that precipitate adverse *side*-effects that are themselves damaging. Whether or not homeostasis is a good or a bad thing, therefore, is largely context-dependent.

In order for a stress response to be adaptive, both psychological and physiological functioning should return the organism to a state of equilibrium following stress. This principal of balance underpins most theories of psychophysiological responses to stimuli, and has become central to our understanding of how psychological states can impact upon

physiological health and well-being. Accordingly, psychophysiological responses to stress require the exercise of homeostatic regulation of both psychological and physiological response processes followed by the engagement of some compensatory processes. In simple terms, the human body can be said to monitor stressors in the immediate environment and to attempt system maintenance through the adjustment of physiological and psychological states. A commonly used example of bodily homeostatic regulation is that of the management of bodily temperature. If sensory organs detect that the body is too cold, shivering of the skeletal muscles can attempt to produce additional body heat. In contrast, if the body is too warm, perspiration can cool the body with the secretion of moisture through the skin. In this way, the body attempts to maintain a constant optimal temperature in times of stress to the thermoregulation system, through a balancing of warming and cooling procedures. The same regulatory principle can be applied to a variety of other human physiological and psychological states.

The impact of psychological stress on physical health offers an example of how a response that was adaptive across evolutionary time serves to challenge the physical well-being of human organisms in their contemporary ecological environment. A range of significant statistical associations between psychological stress and physical health have been observed over many decades, including increased vulnerability to respiratory infection and common colds (S. Cohen, Tyrrell, & Smith, 1991) and poorer immune responses to vaccination (Vedhara et al., 1999). From such findings we can conclude that stress can increase our vulnerability to disease and reduce the effectiveness of preventative disease measures. Homeostasis plays a critical part in the pathway to disease vulnerability. Many of the body's automatic responses to stress reflect an endowment from the ancestral environment of evolutionary adaptation: while facing stressors, our heart rate (HR)

quicken, our muscle-tone increases, our digestive processes shut down, and our skin perspires (among many other responses). Each of these typifies the classical fight-or-flight response, but as famously argued by Cannon, while such responses are useful for dealing with the threats of primitive environments they are less well suited to the symbolic threats and abstractions of modern, civilised, language-speaking societies. In other words, standard stress responses are better suited to fending off sabre-tooth tigers and escaping from predators than they are to meeting project deadlines or surviving traffic jams. Nonetheless, despite the fact that they are costly and draining in the medium-term, our bodies still manifest them.

When bodily stress responses are inappropriate or inadequate there is a risk that well-being or health will be compromised, and this can function in a bi-directional manner. In relation to general human health, this can be demonstrated in terms of over- and under-reaction of stress responses. Hypersensitivity of the immune system is associated with autoimmune diseases, inflammatory diseases, and cancer (Coussens & Werb, 2001; O'Byrne & Dalgleish, 2001), while conditions such as anaphylaxis develop due to an allergic response that becomes rapidly generalised in sensitised individuals (Golden, 2007). In contrast, hyposensitivity of physiological systems can also have extremely serious health consequences (e.g., immunodeficiency through acquired conditions such as HIV/AIDS leads to significantly increased mortality risk; Grulich, van Leeuwen, Falster, & Vajdic, 2007).

One avenue of research that has perhaps revealed the most significant associations between stress and health outcomes is that relating to the cardiovascular system, which has produced a consensus that stress has predominately negative consequences for cardiovascular health (e.g., Kamarck & Lovallo, 2003; Treiber et al., 2003). The

relationship between stress and cardiovascular functioning has been popularly discussed for decades, with pervasive images of the particular vulnerability of middle-aged men working in high stress environments to cardiac incidents. Evidence suggests that clinical manifestations of cardiovascular disease (CVD) can appear sometimes decades after initial onset and asymptomatic progression (Smith & Ruiz, 2002), thus the investigation and determination of factors that might explain CVD vulnerability is crucial. When describing CVD, the full spectrum of atherosclerotic and hypertensive diseases are subsumed, including diseases of the heart or blood supply, cerebrovascular diseases affecting circulation of the brain, coronary heart disease (CHD), ischemic heart disease (IHD), coronary artery disease (CAD), heart attacks (or myocardial infarction; MI), and stroke (Labarthe, 2011). Although CVDs may be identified by the occurrence of an acute event such as MI, they are most often the result of chronic disease processes which proliferate over time.

How stress upsets homeostasis in ways that cause disease.

A number of mechanisms have been proposed to explain how stress may lead to the development of CVDs, including the build-up of physical cardiac abnormalities, such as arterial plaque deposits, and development over time of hypertension (Treiber et al., 2003). Hughes (2013) provided a succinct summary of much of the evidence describing mechanisms contributing to the development of CVD. Elevated cardiovascular responding is thought to reflect disproportionate metabolic reactivity to demands (Obrist, 1981). This disproportionality is thought, over time, to disrupt physiological homeostasis permanently, leading to the development of hypertension.

Blood pressure has been found to be a good proxy for cardiovascular health, with evidence suggesting that it has good temporal stability across long periods of time, even years (e.g., Hassellund, Flaa, Sandvik, Kjeldsen, & Rostrup, 2010). As direct assessment of arterial blood pressure is particularly invasive, for decades now blood pressure has been most commonly measured using automated sphygmomanometers that produce reliable estimates of blood pressure and HR. This allows for blood pressure to be measured easily and non-invasively, with minimal discomfort to a wide demographic range of participants. The assessment of blood pressure yields information about the regulation of cardiovascular functioning during the high and low points of a cardiac cycle, and the impact of circulating blood on the blood vessels (Hughes, 2013). The highest point of the cardiac cycle, when peak pressure occurs in the arteries is known as systole. This peak occurs near the end of the cardiac cycle when the ventricles are contracting, and the heart ejects blood intermittently resulting in what is known as systolic blood pressure (SBP; Levick, 2010). Following systole, the heart rests during a period of cardiac relaxation called the diastole (Levick, 2010). Accordingly, diastolic blood pressure (DBP) is the minimum pressure in the arteries, occurring near the beginning of the cardiac cycle when the ventricles are filled with blood.

A mechanism through which CVD is thought to originate is through long-proliferated styles of cardiovascular functioning which may lead to hypertension, and eventually CVD. The repeated elicitation of stress responses is also thought to contribute to the development of cardiac and vascular hypertrophy (a thickening of heart muscle leading to less available space for blood in chambers of the heart, potentially leading to hypertension; Lovallo & Gerin, 2003). Guidelines published by the World Health Organisation (WHO) outline criteria for the definition of hypertension, stating that SBP of

greater than 140 mmHg, and DBP of greater than 90 mmHg could be said to constitute a hypertensive state (Whitworth, 2003). Tendencies towards higher blood pressure could have physiological consequences if proliferated over time, with increased pressure on the vasculature detrimental to health. Even heightened normal levels of blood pressure have been found to be associated with increased risk of later developing CVD in large-scale epidemiological cohorts such as the Atherosclerosis Risk in Communities study and the Framingham study (Kshirsagar, Carpenter, Bang, Wyatt, & Colindres, 2006; Vasani et al., 2001). Persons who exhibit hypertensive tendencies could be at risk of developing future CVD, and this may be identified by examination of individual differences in patterns of responding to stress.

Elevated blood pressure reactivity to stress is also thought to confer disease risk by increasing serum levels of low-density lipoproteins while lowering levels of high-density lipoproteins (Raitakari et al., 1997). It is also suggested that elevated reactivity to stress could be a part of the exaggerated sympathetic reactivity through increased blood insulin concentrations, which have been shown to be associated with increased hypertension (Nazzaro et al., 2002). Elevated reactivity to stress could contribute to the development of atherosclerosis, through raising serum concentrations of proinflammatory cytokines (Georgiades, 2007). Coronary artery atherosclerosis (CAA) is a disease of the arteries which harden as a result of the deposit of plaque on the inner walls of arteries. Over time, this plaque occludes the artery making it more difficult for blood to pass through. Such restriction of arterial blood flow leads to increased risk of MI and stroke. Progression of atherosclerosis is thought to be associated with hypertension, which may aid in the build-up of fatty plaque on the arteries (Izzo, Sica, & Black, 2008). A chronic and gradual inflammatory process, CAA is the pre-cursor to many CVDs, including CHD.

Multiple risk factors have been attributed to the development of CVD, including family history, obesity, blood pressure, total cholesterol, high-density lipoprotein cholesterol, smoking, glucose intolerance, and left ventricular hypertrophy (Anderson, Odell, Wilson, & Kannel, 1991; Gregg et al., 2005; P. W. Wilson et al., 1998). However, it has been suggested that at least 25% of the variance in the development of CVDs is unaccounted for by these traditional risk factors (Beaglehole & Magnus, 2002). Referring to mortality statistics for the period to 2004, heart disease and stroke have been found to account for approximately 23% of all deaths in the United States (Lloyd-Jones et al., 2009), and by 2030, it is projected that CVDs, mainly heart disease and stroke, will be the leading cause of mortality worldwide (World Health Organisation, 2012). Attempting to understand what may constitute some of the additional variance in the development of CVD has prompted much research in recent decades, with a suggestion that psychological factors could play a role in the pathogenesis of CVD.

It is now well-established that cardiovascular trends in young, healthy people can inform us about later life cardiovascular health risk. Evaluating data from almost one million participants in 61 studies, a 2002 meta-analysis showed that high “usual” levels of blood pressure were associated with vascular mortality (Prospective Studies Collaboration, 2002). The findings refuted earlier assertions by Port, Demer, Jennrich, Walter, Garfinkel (2000) that an age- and sex-dependent threshold for hypertension (of approximately 140 – 160 mmHg for SBP) might exist, below which lower levels of blood pressure are not associated with disease. Findings from the Prospective Studies Collaboration contend that there is a continuous relationship with vascular disease risk throughout the normal ranges of blood pressure, such that usual blood pressure is positively correlated to vascular disease mortality amongst those who are normotensive, as well as those who are

hypertensive. Other evidence supporting the value of blood pressure trends in young people predicting later life cardiovascular health showed that elevations in blood pressure at baseline periods in young men (average age of 30 years), was found to be predictive of CHD, CVD, and all-cause mortality 25 years later (Miura et al., 2001). This study showed that men who had blood pressure that was high normal (SBP of 130-139 mmHg and DBP < 90 mmHg, or SBP <140 mmHg and DBP of 85-90 mmHg), or at the early stages of hypertension (SBP of 140-159 mmHg and DBP of <100 mmHg, or SBP < 160 mmHg and DBP of 90-99 mmHg) had increased cardiovascular mortality risk, which translated to an estimated shorter life expectancy of between 2.2 and 4.1 years (Miura et al., 2001). Findings such as these emphasise the value of examining blood pressure trends in not only older adults, who may already be symptomatic of CHD, but in younger, asymptomatic people too.

Cardiovascular Reactivity as a Central Disease Mechanism

Given that stress is known to have detrimental effects on cardiovascular health, interferences in the process of homeostatic regulation are posited to be one of the means through which CVD risk is increased. Examination of individual differences in cardiovascular reactivity (CVR) to (psychological) stress is one avenue of research that has been pursued in the hopes of gaining a better understanding of some of the mechanisms underlying CVD incidence and progression. The CVR hypothesis states that habitually large or exaggerated cardiovascular responses to stress are detrimental to health (Obrist, 1981), such that chronic activation of the cardiovascular system in response to stress may contribute to disease processes. Reactivity itself has been defined as the magnitude or pattern of an individual's response to behavioural stress (Treiber et al., 2003) and is

typically operationalised in research as a change score, or the difference between two periods of measurement (e.g., the difference between cardiovascular measurements during stress and baseline resting; Kelsey, Ornduff, & Alpert, 2007). Exaggerated reactivity to stress is thought to be one of the contributing factors to the development of hypertension, and later, the development of CVD (Schwartz et al., 2003). It is argued that high reactivity might pose a risk for the manifestation of cardiovascular diseases including atherosclerosis and left ventricular mass (Carroll, Lovallo, & Phillips, 2009). This implication of elevated reactivity in CVD processes has emerged from the convergence of a number of different empirical research literatures.

Animal studies of cardiovascular reactivity and health.

Research on non-human primates has shown the value of examining patterns of cardiovascular functioning as a marker of disease risk. In one early study, reactivity to a capture-and-release stressor was found to be associated with the development of CAA in male cynomolgus monkeys, such that monkeys with greatest CVR to stress had more evidence of CAA (Manuck, Kaplan, & Clarkson, 1983). A further examination of male cynomolgus monkeys showed that monkeys who experienced a social stressor (operationalised as the re-organisation of hierarchical social groups) had more extensive evidence of CVD than those monkeys who did not experience the stressor (Kaplan et al., 1983). A similar protocol to that of Manuck et al. (1983) with female monkeys confirmed that the magnitude of stress responses was associated with severity of CAA (Manuck, Kaplan, Adams, & Clarkson, 1989). Together, these results suggested that psychosocial factors were responsible for the development of CVD in both male and female primates.

The studies confirmed that both exposure to stress, and the magnitude of reactivity to stress can influence the development of CVD. Importantly, findings from animal studies such as these confirmed the role of stress in the development of pre-clinical disease states (i.e., pathogenic changes in cardiovascular structure or function that if continued could lead to the manifestation of CVD; Devereux & Alderman, 1993), and also suggested that the social factors or context could be important factors in individual vulnerability to the progression of CVD.

Reactivity and human cardiovascular health.

Epidemiological research with participants across 52 countries suggests that exposure to psychosocial stressors is associated with increased risk of acute MI (Rosengren et al., 2004). Other studies have supported associations between psychosocial conditions, stress, and CVD states. Using the Whitehall II cohort (a large on-going epidemiological study consisting of a sample of approximately 10,000 British civil servants), Marmot, Bosma, Hemingway, Brunner, and Stansfeld (1997) found that much of the difference in incidence of CVD could be attributed to psychosocial work conditions. Matthews and Gump (2002) found in a sample of men, who were already at increased risk of developing CHD, that increased work stress was associated with greater risk of all-cause mortality and of developing CVD during a nine-year follow-up period. Interestingly, in this sample a role for social factors in the observation of increased disease risk was posited. It was found that increased CVD risk was only observable for men who divorced during the course of the study, with no increased risk found for men who remained married (K. A. Matthews & Gump, 2002), suggesting that the relationship between stress and CVD could be moderated by other psychosocial factors. A comprehensive review by Treiber et

al. (2003) compiled much of the existing research on the risks of stress to human cardiovascular health. The review concluded that there is reliable evidence that CVR can predict negative health outcomes, but that many of the moderating and mediating mechanisms are as yet not fully understood (Treiber et al., 2003).

Laboratory studies of cardiovascular responses to manipulated stress.

Psychological stressors challenge the homeostasis of an organism because of their perceived threat value, regardless of potential for physical harm (Lovallo & Gerin, 2003). A wide variety of psychological stressors have been shown to induce physiological stress reactions, while individual differences in the ways in which people respond to acute stressors have been observed. Not all stressors induce the same magnitude or pattern of cardiovascular functioning. Distinctions have been made between stressors categorised as “active tasks” necessitating an active behavioural response (e.g., mental arithmetic, public speaking), or “passive tasks” that require endurance (e.g., viewing a stressful film, cold pressor task; Krantz, Manuck, & Wing, 1986). Typically, the range of tasks used in experimental stress induction procedures incorporate a wide variety of cognitive stressors (e.g., mental arithmetic, memory tasks, tracing tasks, video games), speech stressors (e.g., public speaking tasks), and physical stress tasks (e.g., the cold pressor task; Zanstra & Johnston, 2011). Variants on these types of tasks can include the manipulation of competitive challenge, or social support.

Much of the earlier work involving the manipulation of laboratory-measured CVR utilised the cold pressor task. A complex task, involving both physiological and psychological stress, the cold pressor task requires the immersion of a hand or foot in cold

water, resulting in the experience of some pain. Work by Hines and Brown (1936), was amongst the first to report a relationship between magnitude of stress reactivity and future hypertension risk using the cold pressor task. It was suggested that a period of vascular hyperactivity could precede the development of hypertension (Hines, 1940). The work of Hynes et al. prompted the widespread use of the cold pressure task in experimental CVR protocols, with many subsequent studies demonstrating positive relationships between cold pressor reactivity and the development of hypertension. A longitudinal study with an interesting period of 45 years showed that hyper-reactors to the cold pressor were more likely to have developed hypertension than normo-reactors (those with blood pressure < 140/90 mmHg; Wood, Sheps, Elveback, & Schirger, 1984). A further study showed that the effects of SBP reactivity, but not DBP or HR, of young male participants (average age of 23 years) to a cold pressor task were only observable 20 years later, and were most prominent in those who subsequently developed early hypertension (before the age of 45 years; Menkes et al., 1989). Findings from studies such as these suggest that physiological hyper-responsiveness to stress at a young age may precede the development of hypertension.

The nature of the cold pressor task is that it is both a physiological and psychological source of stress, and thus further work was need to consider the role of other (purely) psychologically demanding stressors in inducing CVR. An association between CVR to psychological challenge other than the cold pressor task and future blood pressure was found by Matthews, Allen and Woodall (1993). Cardiovascular reactivity of adults (mean age at initial testing time of approximately 43 years) and children (mean age at initial testing of 13 years) to a variety of tasks (serial subtraction, mirror-tracing and isometric hand grip) was measured, and blood pressure was assessed 6.5 years later. The

results confirmed that for adults, challenge-induced SBP and DBP reactivity was positively associated with DBP (but not SBP) at follow-up (K. A. Matthews et al., 1993). For children, male children who exhibited greater task reactivity were found to have higher follow-up blood pressure. The findings from Matthews et al. (1993) imply that people who are at high risk for elevated blood pressure have exaggerated stress-induced cardiovascular responses to psychological stress at a young age.

Bosch et al. (2001) reported differing physiological responses to acute stress manipulations such as a time-paced memory test and a stressful video showing surgical procedures. Increased systemic immunity was observed following exposure to the memory task, while decreased immunity was observed following the video stressor (Bosch et al., 2001). The results from Bosch et al. (2001) point to the power of the choice of stressor in the observation of certain stress responses. More recently, a review of over 208 empirical studies using acute psychological stressors found that stressors which incorporated both uncontrollable and socially-evaluative components were associated with the largest physiological responses, and with the longest recovery times following stress (Dickerson & Kemeny, 2004). In particular, stressors that incorporated a public speaking element were found to elicit greater cortisol stress responses than other categories of task such as mental arithmetic or Stroop task (Dickerson & Kemeny, 2004). Importantly, this socially evaluative feature of experimental tasks appeared to be the most important factor in the observation of differing stress responses, rather than ecological or methodological features such as task duration. The authors of the review suggested that self-appraisals of task performance could contribute to the generation of the observed increased stress responses during socially evaluative stress (Dickerson & Kemeny, 2004).

Further evidence for the relationship between psychological stressors and CVR found that initial CVR to a battery of standardised cognitive and psychomotor computer tasks, in a group of middle-aged Finnish men, was significantly and positively associated with carotid intima-media thickness (an important factor in the pre-clinical development of CVD) seven years later (Jennings et al., 2004). In the longitudinal CARDIA study, blood pressure reactivity of initially normo-tensive young adults to a variety of psychosocial stressors (including mirror tracing, video game, and the cold pressor task) was found to predict hypertension 13 years later (K. A. Matthews et al., 2004). The results were held constant even after adjustments for other major predictors of hypertension, including age, race, gender, BMI (body mass index), education, and resting pressure (K. A. Matthews et al., 2004), demonstrating that larger blood pressure reactivity to stress was associated with earlier development of hypertension.

A recent review of CVR observations found that there was strong evidence to support the generalisability of physiological differences observed under controlled laboratory conditions to real life settings (Zanstra & Johnston, 2011). Importantly, blood pressure was generally found to be higher during real life stress than during comparable laboratory stress induction, thereby confirming the ecological validity of laboratory stressors. Previously, consistency of cardiovascular responses to laboratory stressors over time has been found to be good across a range of stressors, including CVR to a challenging interview stressor at one instance and a mental rotations task two weeks later (Smith & O'Keeffe, 1988), and across a maths task, video game, and cold pressor task (Kelsey et al., 2007). Zanstra and Johnston's (2011) review found that stress-induced CVR in real life was reliable and subject to individual determinants and other moderating effects (e.g., environmental characteristics and personality characteristics). Particular attention to stress

was directed to CVR elicited by interpersonal stressors, due to their immediate parallels with real life stress (e.g., speech tasks; K. A. Matthews, Manuck, & Saab, 1986). The review supported the generalisability of “snapshot” physiological responses from the laboratory to real life contexts. It was argued that laboratory stressors can be effectively grouped with regards to the physiological processes that they elicit (i.e., active tasks characterised by myocardial responses; passive tasks characterised by vascular responses). With the advent of more complex ambulatory monitoring technology in recent years, findings pertaining to the examination of stress responses in real life contexts have suggested that cardiovascular functioning may be mediated by myocardial or vascular factors as a consequence of the type of stress that participants are exposed to (e.g., Zanstra, Johnston, & Rasbash, 2010). It was concluded that average cardiovascular responses to specific laboratory stressors do correlate with real life stress responses, and that the nature of the stress, as well as the reactivity of the person should be considered.

Extensions to The Cardiovascular Reactivity Hypothesis

While the claims of the CVR hypothesis (Obrist, 1981) for habitually exaggerated reactivity have been hugely influential, it is not the only dimension of cardiovascular stress responding that can be quantified, or that can be interpreted as a marker of disease risk. For example, the speed of physiological recovery after stress exposure, which can also be quantified, is likely to be linked to the impact of stress responding on the body. Similarly, the possibility that the cardiovascular system might be capable of *under*-responding to a stressor, in ways that have adverse health consequences, should also be considered (Carroll et al., 2009). Patterns of CVR other than exaggerated stress reactivity may have empirical value, given that such CVR accounts for only part of the variance seen in future blood

pressure. Carroll, Smith, Sheffield, Shipley and Marmot (1995) demonstrated that reactivity to the Raven's matrices task accounted for only 1% of the variance in follow-up blood pressure (although the age-range of participants in this study included persons unlikely to be completely disease-free at baseline, and the validity of Raven's task as an authentic psychological stressor is not well established). As suggested by Lovallo (2011), cardiovascular response deviations from normal in either direction (hyper- or hypo-responding) may signal a loss of homeostatic regulation, and therefore increase disease risk.

Delayed post-stressor recovery.

Although considerable evidence has been amassed that the magnitude of CVR is important for future cardiovascular risk (see Treiber et al., 2003 for a review), consistent with homeostatic perspectives of physiological functioning, the duration of a psychosocial stress response has also been proposed as a further mechanism that could contribute to CVD risk. Recovery can be defined as either the time required to return to baseline levels after termination of a stressor, or the degree of elevation above baseline levels within a pre-determined post-task interval (Stewart & France, 2001). A meta-analysis of 69 studies showed that recovery was most often quantified using change (delta) scores, reflecting an absolute change from baseline levels (Schuler & O'Brien, 1997). The results of this meta-analysis also found small, but reliable effects for the relationship between cardiovascular recovery and the development of hypertension. Individuals who were at higher risk of developing hypertension demonstrated less cardiovascular recovery from laboratory stress, with strongest observed predictions for cardiovascular recovery for participants who were hypertensive, or border-line hypertensive (Schuler & O'Brien, 1997). Results also showed

that more complete cardiovascular recovery was associated with decreased risk of developing hypertension (Schuler & O'Brien, 1997).

Stewart and France (2001) found that recovery from stress was associated with longitudinal levels of blood pressure (3 years later), positing that individuals who do not return to normal (lower) levels of cardiovascular functioning after exposure to stress may be at risk of developing CVD in the future. In particular, the results found that poorer SBP recovery (from a cold pressor task and a tourniquet ischemia task) was associated with elevations in baseline SBP three years after initial assessment (Stewart & France, 2001). The longitudinal predictive value of cardiovascular recovery was further demonstrated using the Whitehall II cohort. Steptoe and Marmot (2005) showed that at a 3-year interval, impaired post-stress recovery of healthy middle-aged men and women predicted increases in SBP and DBP. Attenuated SBP recovery from a battery of psychological tasks was significantly and positively associated with 3-year changes in blood pressure, specifically with higher SBP and DBP in a sample of community-dwelling adults (Stewart, Janicki, & Kamarck, 2006). Statistical significance was retained even after adjustments for traditional clinical and lifestyle risk factors.

Research on the generalisation of blood pressure recovery to more real life situations has suggested that impaired recovery has an association with blood pressure (Trivedi, Sherwood, Strauman, & Blumenthal, 2008). In a protocol involving both assessments of stressor reactivity and recovery, and ambulatory blood pressure, results showed that post-stress recovery of SBP and DBP significantly predicted daytime and night-time ambulatory blood pressure (Trivedi et al., 2008). Importantly, initial effects attributed to CVR in the prediction of ambulatory blood pressure were no longer

significant with the addition of recovery blood pressure into the statistical model (Trivedi et al., 2008).

Diminished response to stress.

The original CVR hypothesis focused on exaggerated responses as being responsible for, or at least providing a pathway for mechanisms of pre-clinical CVD states. By this logic, lesser or diminished reactivity was thought to be indicative of a healthful and adaptive profile of stress responding. However, more recently a number of paradoxical findings have emerged, leading researchers to examine whether lower stress reactivity could also indicate poor physiological stress coping. The emergence of research in this area has led to the most recent assertion that diminished as well as exaggerated physiological reactivity should be seen as non-optimal physiological functioning that can contribute to poor health outcomes (Lovallo, 2011).

Phillips, Ginty, and Hughes (in press) reviewed much of the evidence concerning blunted physiological responding to stress, and found reliable associations between blunted physiological functioning and several health outcomes. Associations with diminished stress responding were observed for a range of variables including; obesity (Carroll, Phillips, & Der, 2008), poor self-reported health (De Rooij & Roseboom, 2010), exercise dependency (Heaney, Ginty, Carroll, & Phillips, 2011), disordered eating (Ginty, Phillips, Higgs, Heaney, & Carroll, 2012), and poorer cognitive function (Ginty, Phillips, Der, Deary, & Carroll, 2011).

Individual differences in blunted stress responding has been found in some instances to be moderated by certain personality dimensions. Higher neuroticism was

associated with diminished CVR to a mental arithmetic stressor (Hughes, Howard, James, & Higgins, 2011), with blunted antibody response following vaccination, and blunted cortisol reactivity to a mental arithmetic task (Phillips, Carroll, Burns, & Drayson, 2005). Type-D personality was also found to be associated with significantly weaker myocardial responses to stress (Howard, Hughes, & James, 2011). Blood pressure reactivity to a mental arithmetic stressor was found to be *inversely* associated with depression, obesity, and poorer self-reported health in a large study of participants in the West of Scotland Study (Phillips, 2011). A further study measuring CVR to several laboratory stressors (public speaking, cold pressor, and viewing of earlier speech task video footage), found that lower CVR interacted with depression scores during tasks that had a self-relevant component (i.e., public speaking or speech viewing), such that attenuated CVR was found for individuals scoring higher in depression during these tasks (Schwerdtfeger & Rosenkaimer, 2011). Prospectively, attenuated cardiac (specifically HR) reactivity was associated with subsequent depression scores 5 years later, as measured by the Hospital Anxiety and Depression Scale (Phillips, Hunt, Der, & Carroll, 2011).

Although the mechanisms underlying blunted physiological responding to stress are not yet fully understood, several suggested theories have proposed. Phillips et al. (in press) provided an overview of several proposed explanations for observations of blunting. Firstly, diminished reactions to stress may reflect lower effort on the part of the participant. In this vein, blunted reactivity to stress could be a consequence of a dysfunction of neural systems which support motivated behaviour. Blunted reactivity could in this case potentially reflect reduced unconscious effort, which could account for observed blunted physiological responses to stress. Some support for this assertion is drawn from the work of Carroll, Batty, Mortensen, Deary and Phillips (2011), which found

that reduced motivation was associated with blunted reactions to stress. Concerning exaggerated or diminished stress responses, Carroll et al. (2009) suggest that both responses may be operating in a state of bias, at the level of the higher central nervous system (at the level of the hypothalamus and brainstem), or at the periphery. Secondly, blunting could be the result of a reduced physiological capacity to respond to stress, such that people are unable to manifest an appropriate stress reaction. Research on obesity has provided some support for this perspective. It has been found that once someone becomes obese, they are characterised by high levels of sympathetic nervous system activity, such that their sympathetic nervous system may become less responsive to stress (Tentolouris, Liatis, & Katsilambros, 2006). Thirdly, reduced responding to stress could be a consequence of reduced awareness or perception of stress. It is generally acknowledged that the processes underlying adaptation to stress are predominately dependent on cognitive aspects of stress perception, rather than on sensory receptor adaption or physiological fatigue (Thompson, 2000). As suggested by Hughes et al. (2011), styles of stress perception are likely to vary across people, but to be relatively stable within persons, resulting in a characteristic way of interpreting stressful situations, which may be moderated by personality characteristics. Blunting could be affected by a series of personality traits (such as neuroticism or depression), which dispose individuals to certain cognitive interpretations of situations. Several different studies have shown support for this position, and have been described above (e.g., Howard et al., 2011; Hughes et al., 2011; Phillips, 2011; Phillips et al., 2005). Finally, it is suggested that dampened stress reactivity may occur as a result of prior psychological or physiological events, such as childhood trauma. Associations between adverse life events experienced during childhood, and

blunted HR and cortisol response of adults to laboratory stressors have been observed, providing some support for this suggestion (Lovallo et al., 2012).

There has not been universal support for the existence of blunted physiological responding. If it is agreed that myocardial responding to stress (as indicated by increases in parameters such as SBP, HR and cardiac output; CO) is associated with exaggerated stress responding, then potentially, suboptimal cardiovascular responding could be seen as a reflection of an increased vascular response (as indicated by increased vascular resistance). Given that the normal functioning of the cardiovascular system constitutes a reciprocal interchange between vascular and myocardial forces, James et al. (2012) suggest that observed instances of (myocardial) blunted stress responses may more accurately be indicative of an increased vascular response tendency. Indeed, the nature of what may constitute adaptive cardiovascular stress responding may be dependent on the nature of the stress that is being experienced.

During exposures to active stressors (i.e., the tasks or stress responses requiring an “active” response), increases in blood pressure may be characterised by elevations in cardiac contractility and CO, as well as decreased peripheral resistance (indicating a beta-adrenergic response profile). In contrast, during passive stress (e.g., a stressor such as the cold pressor task requiring endurance or a passive behavioural response), blood pressure increases may be accompanied by diminished CO and elevated peripheral resistance (indicating an alpha-adrenergic response profile; Schwerdtfeger & Rosenkaimer, 2011). It has been suggested that the exhibition of vascular stress responses when a myocardial response might be anticipated, or the elicitation of a vascular response without an accompanying decrease in myocardial factors, could indicate a maladaptive coping response style. Some evidence suggests the assertion that increased vascular responding

may precede the development of hypertension and is associated with greater risk of CVD (Palatini & Julius, 2009).

As James et al. (2012) suggest, it may be that observations of blood pressure do not always capture the full extent of physiological functioning, as blood pressure does not reflect the complexities of hemodynamic processes. Muted CVR to acute stress may be evidence of stress dysregulation, leading to (or a result of) poor physical health (Howard et al., 2011). It is desirable that stressors are in some way acknowledged as stressful by individuals, but the associated physiological reactivity should be proportionate to the stress that is involved, and should dissipate relatively quickly. Thus, it may be that moderate levels of physiological activation might be most beneficial for health, rather than the inverse of exaggerated reactivity; attenuated reactivity. Carroll et al. (2009) characterised the relationship between physiological reactivity to stress and health as an inverted-U model. In this model, a continuous relationship between reactivity and health outcomes is conceived, whereby both exaggerated *and* diminished responses to stress are maladaptive (depending on the health outcome in question).

Cardiovascular adaptation to stress.

During repeated stressor exposures, cardiovascular parameters characterised as myocardial factors (e.g., HR) typically attenuate over subsequent stressor presentations, while other, vascular, factors (e.g., total peripheral resistance; TPR) may not decrease or may in fact increase (Kelsey et al., 1999). Although the exact mechanisms involved in the negative health outcomes associated with failed or delayed adaptation to stress remain unclear, some potential explanations for increases in vascular factors (and thus failure to adapt to stress over time) have been posited, most recently by Hughes, Howard, James,

and Higgins (2011). The authors suggest that increased vascular resistance during stress could reflect a process of auto-regulation, whereby increases in vascular factors could be prompted by the same beta-adrenergic reductions that underlie increases in myocardial factors. Alternatively, increases in parameters such as TPR which are indicative of systemic vascular restriction could be a consequence of increased alpha-adrenergic vasoconstriction, or some combination of beta-adrenergic vasodilatation, and alpha-adrenergic vasoconstriction (Carroll, Cross, & Harris, 1990; Kelsey et al., 1999). Whatever the mechanism for lack of physiological attenuation to stress, it seems that vascular responses during repeated stressor exposures are implicated in ways that are harmful to cardiovascular health.

As with other patterns of physiological responding to stress, individual styles of responding to stress have been identified in relation to adaptation to stress. Characteristic increases or decreases in cardiovascular functioning have been demonstrated in a single experimental session, with two (or more) stressor exposures (Frankish & Linden, 1991). As argued by Hughes et al. (2011), adaptation to stress has implications for the widely accepted CVR hypothesis. The authors describe how a growing literature on stress adaptation may diminish the explanatory powers of the CVR hypothesis relating to patterns of everyday physiological functioning. If people adapt to stress, then cumulative effects of reactivity to stressors, as observed in laboratory contexts, may not be as relevant as previously thought. In the same vein, the accepted generalisability of initial stress reactions, often to novel laboratory stressors, may be questionable if people are found to adapt to stress. However, research on adaptation of CVR to stress has broad implications for greater understanding of how mental stress and anxiety may influence physiological stress responses. As previously mentioned, styles of coping with stress, and stress

appraisals are relatively stable within individuals, but they vary widely between participants (Hewitt & Flett, 1996). This provides scope for examining individual stress vulnerabilities. Dispositional appraisals too could influence underlying cardiovascular responses to stress, such that active coping efforts are thought to reflect beta-adrenergic processes, while passive coping is thought to reflect alpha-adrenergic activity. Findings from Hughes et al. (2011) suggest a particular association between adaptation of cardiovascular functioning and myocardial responses to stress. When attempting to consider what particular individual difference variables may be relevant for the differentiation of cardiovascular responses, it is proposed by Hughes et al. that factors influencing immediate stress responsivity, and an individual's self-perceived capacity to adjust to stress over time should be considered. A number of personality characteristics in this vein have been proposed to be associated with cardiovascular functioning (generally), and are presented below.

Individual Differences in Cardiovascular Stress Response

Physiological responses to stress may also reflect underlying behavioural or psychological tendencies (Carroll et al., 2009). Characteristic physiological responses to stimuli or stress may be the result of pre-dispositions in personality, as well as the nature of the stimulus. Thus to understand biological responses to stress properly, attempts should be made to understand some of the individual determinants of the stress response. What we commonly refer to as personality is merely an outward expression of underlying physiological processes. Personality affects how we interpret situations, interact with people, and cope with stress, resulting in the elicitation of responses that are largely consistent within a person. Styles of physiological responding may be said to reflect

personality differences, given the widespread and reliable observation of correlations between psychometric personality variables and cardiovascular responses.

Not only have individual differences in styles of CVR been identified (i.e., a high or low stress reactor), but additionally variability in aspects of personality have been shown to be associated with differing CVD risk. One of the most prominent methods of characterising personality types is encapsulated by trait theories of personality. The trait perspective of personality assumes that certain personality characteristics or traits are relatively stable constructs that endure across situations, and that these traits influence behaviour (G. Matthews, Deary, & Whiteman, 2003). Although a person's reaction may vary between situations, there are a set of innate characteristics that colour their emotions, cognitions, and behaviour. Categorisations of people along dimensions of personality are not clinical diagnoses, but rather represent normative variations in the strength of individual dispositions. They may influence how persons perceive and react to cues in their environment, and have been implicated in the ability to adapt to psychological stress.

Historical perspectives on the biological basis of personality.

Since ancient times, attempts have been made to understand human nature by attributing behaviours and cognitions of individuals to characteristic styles of temperament. The ancient Greeks, such as Hippocrates (circa 400 B.C.), and later Galen (circa 200 A.D.) promoted the idea of an interaction between psychological and physiological states, such that differences in psychological functioning could have a biological basis. In one of the earliest incarnations of what would much later become modern personality theory, it was thought that four bodily fluids influenced aspects of

human temperament or personality. These fluids, or “humors” were thought to affect four main temperaments, known as sanguine (pleasure-seeking and sociable), choleric (ambitious and leader-like), melancholic (introverted and thoughtful), and phlegmatic (relaxed and quiet; Pervin, Cervone, & John, 2005). Individual differences in personality were thought to correspond to the predominance within an individual of one or other humor, while diseases were thought to correspond to an excess or detriment of a particular humor (Pervin et al., 2005). The concept of these four humors suggested that there were some aspects of temperament or personality that were universal, or near universal, and that there was a biological basis to personality. Galen’s humors dominated much of medical thinking until the mid-nineteenth century when advancements in disease pathogenesis and cellular pathology discredited the claims of four bodily humors. However, aspects of the description of humors persist in modern personality theories, albeit in different forms.

The most conspicuous form of “humors” in contemporary psychology is represented by the various trait theories of personality (e.g., Allport & Odbert, 1936), which consider traits (the basic units of personality) to be fundamentally biological. Traits may be described as broad descriptions of largely consistent patterns in the way individuals behave, think, and feel (Pervin et al., 2005). Several factor structures have been proposed in line with trait theory, including Allport’s (1936) triad of cardinal, central, and secondary traits; Eysenck et al.’s (1985) three bipolar dimensions of introversion-extraversion, neuroticism, and psychoticism (often referred to by the acronym PEN); and Cattell’s multivariate approach to trait theory, resulting in distinctions between ability traits, temperament traits, and dynamic traits, using a 16-item personality inventory (Cattell, Eber, & Tatsuoka, 1988).

The Five-Factor model of personality, proposed by Costa and McCrae (1992), is an empirical generalisation about the covariance of personality traits. As with some of the earlier trait theories, it is premised on the fundamental lexical hypothesis, first suggested by Francis Galton (1884). This hypothesis suggests that some aspects of personality are so pervasive throughout human societies that commonalities in the description of human behaviour and motivation will appear across languages and cultures (Pervin et al., 2005). With this in mind, the Five-Factor model organises individual differences in personality across five bipolar dimensions (or factors); neuroticism, extraversion, conscientiousness, agreeableness, and openness to new experience. The Five-Factor model integrates some previous work in trait theory, using principles of scientific rigour such as factor analysis, inherent in the work of Eysenck and Cattell, to derive personality structures describing the breadth of human personality. The Five-Factor model fundamentally integrates aspects of Eysenck's PEN theory, including the concepts of neuroticism, extraversion, and even psychoticism (as a result of low scores on both dimensions of agreeableness and conscientiousness).

As an extension to the Five-Factor model, the Five-Factor theory of personality (McCrae & Costa, 1996; McCrae & Costa, 1999) was devised as a contemporary version of trait theory, to counter some criticisms that Five-Factor traits were merely descriptive. According to the theory, the Five-Factor factors are more than mere descriptions, and should be treated as things that really exist. Traits are seen as part of a psychological structure that everyone has in varying amounts. Five-Factor theory focuses its attention on the distinction between basic tendencies (abstract psychological potentials) and characteristic adaptations (their concrete manifestation in the personality systems), assigning traits exclusively to the category of basic tendencies (McCrae & Costa, 2008). It

is argued that characteristic adaptations (e.g., attitudes, habits, roles) vary across cultures, families, and even the lifespan, while basic tendencies (or personality traits) do not (McCrae & Costa, 2008).

A bespoke cardio-active personality type: The Type-A behaviour pattern.

An association between individual differences in personality and health and well-being has long been suspected. Since the 1950s, empirical investigations have evaluated the plausibility of such relationships, with particular emphasis on cardiovascular health due to the proposal of a “coronary-prone” personality, originating with the “type-A behaviour pattern” (TABP; M. Friedman, 1996). The development of the TABP in the 1950s had a profound effect on the development of modern empirical research into the relationship between individual differences in personality and health. Stemming initially from observations from the waiting room of a San Francisco cardiology practice, TABP evolved to describe individuals who are characterised by free-floating hostility, aggressiveness or dominance, a sense of time urgency, and competitive drive (M. Friedman, 1996). It was observed that some of the waiting room chairs were in greater need of refurbishment than others, with unusual wear of the chairs on the front edges of the seats and armrests, instead of on the back areas which would have been more typical. This wear was attributed to the general impatience of certain coronary patients who often jumped up from their seats and demanded to know how much longer they would have to wait. This behaviour was later said to typify those who were characterised as type-A individuals, reflecting a feature of their general impatience.

Research findings describing increased CVD risk for type-A individuals (e.g., M. Friedman & Rosenman, 1959) prompted decades of research into the TABP. Although

TABP became hugely popularised outside of academic circles, and indeed persists to today, several large epidemiological studies have not supported TABP as a whole as a predictor of CVD (see Riska, 2000 for a review), despite the wider literature collectively demonstrating a relationship with cardiovascular risk. With the demise of support for a global TABP, attempts were made to determine what the most cardio-toxic elements of TABP might be. As a result of these investigations, consistent empirical support describing adverse cardiovascular health outcomes has widely been attributed to elements of anger and hostility (Booth-Kewley & Friedman, 1987; Everson-Rose & Lewis, 2005), but also to trait dominance (Whiteman, Deary, & Fowkes, 2000).

Negative affectivity and neuroticism as cardio-active personality dimensions.

Ample evidence for the existence of a relationship between personality variables and health outcomes has been put forward (see Smith & MacKenzie, 2006). Some of the strongest associations between personality and cardiovascular health outcomes appear to relate to a category of personality traits and behavioural dispositions termed negative affectivity (NA). Watson and Clark (1984) described this grouping of certain personality traits as a mood dispositional dimension, reflecting what has become known as NA. Included under the remit of NA are personality constructs such as neuroticism, trait anxiety, and variables derived from TABP, including hostility. All of these elements have been shown to be associated with both health outcomes and health behaviours in some respect.

Several studies have confirmed the detrimental effect of NA on cardiovascular functioning, including a comprehensive review by Suls and Bunde (2005). This review concluded that aspects of negative emotions (e.g., anger-hostility and elements subsumed

within TABP, depression, and anxiety) are related to increased risk of CHD in healthy populations (Suls & Bunde, 2005). Separately, individual differences in NA (particularly anxiety, depression and angry-hostility) were found to be associated with pre-clinical and asymptomatic CAD, as assessed by degree of coronary artery calcification (Smith et al., 2008). The results from Smith et al. (2008) found independent associations between hostility and dominance in the predication of CAD, an important distinction given the large volume of research supporting the associations between hostility and anger on human health (see Miller, Smith, Turner, Guijarro, & Hallet, 1996 for a review), and the comparatively lesser quantity of research investigating the effects of trait dominance on human cardiovascular functioning. Results from one study suggested that dominance could be accurately described as reflecting a measure of hostile dominance or aggressiveness, as it was found to predict greater CHD, even when anger was controlled for (Smith, Glazer, Ruiz, & Gallo, 2004).

A criticism of the way that personality traits are investigated in health psychology research has been that there has been a tendency for research to continually derive more narrow conceptualisations of personality, which are increasingly similar to each other (Marshall, Wortman, Vickers, Kusulas, & Hervig, 1994). In an evaluation of the convergence between health-relevant personality characteristics and wider personality domains, Marshall et al. (1994) found that many health-related personality constructs appeared to be mixtures of broad personality characteristics (as measured by the Five-Factor model), and that these broad characteristics provided adequate and valuable means for guiding research interested in the relationship between personality and health. Marshall et al. (1994) argued that a network of personality constructs, anchored by broad Five-

Factor personality characteristics, provide a rich context for the evaluation of associations between personality and health.

Neuroticism is a Five-Factor personality variable with strong links to NA. It is associated with an enduring tendency to form negative appraisals of both immediate and long-term stressors (McCrae, 1990). High neuroticism contrasts adjustment or emotional stability with maladjustment or neuroticism, and has been associated with negative health outcomes in several research studies (e.g., Shipley, Weiss, Der, Taylor, & Deary, 2007; Terracciano, Löckenhoff, Zonderman, Ferrucci, & Costa, 2008). It is important to note that variations in the neuroticism personality sub-scale are normative orientations, and do not represent clinically diagnosable conditions or disorders. Freedom from negative feelings does not mean that low scorers experience a lot of positive feelings; frequency of positive emotions is a component of the extroversion domain. The most consistently observed effects in relation to Five-Factor personality traits and cardiovascular physiological processes have related to neuroticism, and those factors relating to NA.

Neuroticism has been linked to CVD (Suls & Bunde, 2005) and CVD mortality (Shipley et al., 2007), and poorer antibody response to vaccination (Phillips et al., 2005). There is also growing evidence from large studies of representative samples that neuroticism significantly predicts longevity in the general population (Smith & MacKenzie, 2006). Neuroticism has also been found to be associated with greater CVR and less adaption to repeated stress (Hughes et al., 2011). Neuroticism was further found to be positively associated with blunted, or diminished initial stress responses, while interestingly low neuroticism was associated with higher initial stress responses, but greater decreases in stress responses (Hughes et al., 2011). While previously some physiological benefits to acute stress responding have been observed (previously acute

stress responses have been associated with improved immunity; Phillips, Carroll, Burns, & Drayson, 2009), in the long-term, the beneficial effects of such stress responses are unlikely to persist.

Although the results of studies describing the effects of neuroticism generally support a positive relationship with adverse health outcomes, some null findings have been observed. A meta-analysis by Chida and Hamer (2008) found that the assertion of a relationship between CVR and neuroticism was not supported. The review (2008) did suggest that neuroticism or negative affect could be associated with cardiovascular hypo-reactivity and poorer recovery following stress, despite not being related to reactivity. The authors further suggested that cardiovascular hyper-reactivity could be more reliably associated with individual differences in anxiety, aggression, and TABP behaviour. It should be acknowledged that the study did combine results for neuroticism with some of the wider NA variables, which in some way may have masked findings that could have been attributable to neuroticism.

Incorporating the social context: trait dominance as a cardio-active personality dimension.

Trait dominance (sometimes referred to as ‘social dominance’) is an individual difference variable encapsulating social competitiveness and the degree to which persons innately desire to exert a dominant position within their social group (Pratto, Sidanius, Stallworth, & Malle, 1994). This assertion of authority is typically aimed at individuals considered as being in the “out-group”. This conceptualisation of trait dominance is as a continuous variable, ranging from low to high dominance, with most of the distribution representing a non-clinical disposition. People who score higher on measures of trait

dominance tend to express preferences for hierarchical relationships in society, the distribution of resources based on merit, as well conservative ideologies, military programmes, and punitive justice policies (Pratto, Tatar, & Conway-Lanz, 1999). In contrast, individuals who score low on trait dominance tend to favour social equality, distribution of resources based on need, and social intervention programmes (Pratto et al., 1999). However, it should be noted that low dominance does not necessarily equate to high submissiveness; nor does low submissiveness equate to high dominance.

A dominant position might generally be associated with greater access to resources, and in evolutionary terms to the best opportunities for reproductive success. As with any evolved adaptation, trait dominance that could have been useful for prehistoric humans may not prove to be as appropriate in contemporary society, and the continued assertion of dominance may prove taxing (or otherwise counterproductive) in the long-term. Challenges to the dominant position are fraught with danger, and there is considerable stress associated with the maintenance of this dominant position. The implications of trait dominance are overtly social and inherent in the disposition, requiring social interaction in an effort to triumph or achieve dominance over other people in the environment.

Some striking results pertaining to the health-damaging consequences of dominance have been observed in relation to research with non-human primates. Some studies have shown that the effort required to sustain a dominant position can exact a huge physiological huge toll on the body. Chronic activation of such a stress response could have very damaging effects on an organism's health, with particular regard to cardiovascular health. In most social species (human included) dominance rank influences the extent to which an individual sustains physical and psychosocial stressors (Sapolsky, 2005), and over long periods this can be associated with an animal's vulnerability to

disease and disease processes. A review of the relationship between dominance and primate health found that adverse physiological outcomes are most pronounced amongst animals who are exposed to the most physical and psychological stressors (Sapolsky, 2005). Some personality explanations have been suggested for the influence of societal characteristics on stress of higher- and lower-ranking members of (a primate) society (Sapolsky, 2005). Dominant male monkeys in unstable social hierarchies were found to have significantly more advanced CAA than subordinate monkeys in stable or unstable groups (Kaplan, Manuck, Clarkson, Lusso, & Taub, 1982). For these dominant monkeys, social dominance was associated with CAA when recurrent stress was present in their immediate social environment. In contrast, it was observed that subordinate female monkeys (those that were unable to achieve dominance) had more extensive coronary artery damage than their dominant counterparts, irrespective of the conditions of the immediate social hierarchy (Kaplan et al., 1982). Results from studies such as these suggest that social characteristics of the environment may interact with dominance to produce differing health effects, with the worst effects observed for males generally who were in a position incongruent with their dominance orientation (i.e., their dominant position was consistently subject to challenge by less dominant monkeys).

A review of human cardiovascular function and trait dominance (Newton, 2009) summarised much of the research conducted on human CVR and trait dominance, finding that evidence from human subjects has largely corroborated results from animal studies. Trait dominance was found to be positively and significantly associated with adverse cardiovascular health outcomes, such as increased risk of post-MI morbidity and mortality (Helgeson, 1990), and with increased odds of combined fatal and non-fatal cardiovascular events (Siegman, Kubzansky, et al., 2000). Laboratory experiments have confirmed a role

for dominance in the aetiology of CVD. Under experimental conditions where participants were instructed to engage in social persuasion, it was found that attempts to influence outcomes in a social context produced levels of CVR that were similar to the magnitude of CVR responses generally observed to asocial tasks (e.g., mental arithmetic; Smith, Allred, Morrison, & Carlson, 1989). Cross-sectionally, hostility and dominance were found to be independently related to CHD (Siegman, Townsend, Civelek, & Blumenthal, 2000), with a suggestion that the effects of dominance for men and women could be mediated by different processes, such that indirect manifestations of antagonism confer CHD risk in women and that more overt expressions of anger confer risk in men (Siegman, Townsend, et al., 2000). Dominance and CHD in the Normative Aging Study, an ongoing cohort of older men, showed a significant prospective relationship between questionnaire assessed dominance and CHD in men (Siegman, Kubzansky, et al., 2000). This relationship remained significant even after adjusting for traditional cardiac risk factors and anger. Further, some findings have shown that greater submissiveness may exert a protective effect on the development of CHD particularly for women (Whiteman, Deary, Lee, & Fowkes, 1997).

Main Conclusions and Thesis Outline

The literature reveals that indices of cardiovascular functioning are important health risk determinants. Individual differences in patterns of reactivity and functioning have been associated with differential disease risk, and additionally, certain dimensions of personality have been shown to moderate this risk. General personality traits such as those described by Five-Factor theory have shown permeation through many cultures and societies and are good general indicators of dispositional behaviours or tendencies towards

a particular style of behaviour. More specifically, individual differences in behavioural tendencies, particularly those relating to NA have shown quite strong relationships with health outcomes and risk factors, especially cardiovascular health. These variables, including anger, hostility and anxiety are particularly relevant when the nature of the stress that is being experienced is considered as a further moderating influence.

Accordingly, the present body of research wished to investigate, in three different methodological ways, the relationship of personality traits to normative blood pressure functioning and variability. Firstly, the role of individual differences in everyday patterns of cardiovascular functioning over an extended period of monitoring was considered. Such a detailed investigation pointed to the importance of considering not only contextual (socially relevant) factors inherent in everyday life during of cardiovascular functioning, but also the temporal period during which cardiovascular functioning was assessed. The influence of a specific interpersonal personality disposition (trait dominance) on cardiovascular functioning was investigated during the manipulation of social context using two active psychosocial stressors; mental arithmetic and a speech stressor. Finally, the moderating effect of trait dominance on cardiovascular habituation to repeated social stress exposures was considered.

Chapter 2: Study 1

Personality Differences in Cardiovascular Function During Daytime Waking

Hours

Consistent with theories of homeostasis (Cannon, 1915), there is much empirical evidence to support the view that tendencies towards *either* too large *or* too small reactions to stress are harmful in to long-term physical health (Carroll et al., 2009). Delayed recovery following stress (Schuler & O'Brien, 1997; Stewart et al., 2006), disrupted habituation to stress (Hughes et al., 2011), and blunted reactivity to stress (Phillips, 2011) have all been implicated in the aetiology of CVD. To date, much of the research reporting associations among negative health outcomes, variability in cardiovascular functioning, and dimensions of personality has been gleaned from either laboratory or 24-hour ambulatory protocols. Observations of detrimental associations between blunted cardiovascular functioning, and ill-health in particular, have so far been limited to acute stress reactivity protocols. These protocols have almost exclusively been in the context of a formal laboratory setting (Phillips et al., 2005), or in informal settings arranged to mimic a laboratory (e.g., a quiet space in a domestic dwelling; Phillips, 2011; Phillips et al., 2011).

Examination of patterns of blood pressure functioning over extended periods has been limited by methodological concerns, and in particular the time-frame within which cardiovascular functioning has been examined. Ordinarily, blood pressure follows a

circadian pattern, typically with higher levels of blood pressure during daytimes falling to lower levels at night (Millar-Craig, Bishop, & Raftery, 1978). Disruptions to the circadian rhythm have been found to predict greater risk of cardiovascular morbidity when reductions in blood pressure from day to night are blunted or absent (Verdecchia et al., 1994). A large number of studies have studied variability in diurnal blood pressure by comparing daytime and night-time phases from one 24-hour period (e.g., O'Brien, Sheridan, & O'Malley, 1988). From research such as this, increased mortality and cardiovascular risk has been suggested for “non-dippers” (i.e., people whose blood pressure does not exhibit a $\geq 10\%$ drop overnight; Fagard et al., 2009). Blunted or absent reductions in cardiovascular functioning from daytime to night-time has been associated with negative cardiovascular outcomes, regardless of mean level of daytime blood pressure (Verdecchia, 2000). However, cardiovascular arousal may fluctuate within the daytime phase, such that day-night comparisons may not fully capture the nature of diurnal shifts.

Blood pressure variability implies the degree of fluctuation in blood pressure, usually expressed in terms relating to the standard deviation. Investigations of the prognostic value of increased blood pressure variability have yielded some mixed findings, including null effects when relevant cardiac risk factors were included in the analysis (e.g., Verdecchia et al., 1996). Nonetheless, a consensus has emerged that increased variability is detrimental to health (see Rothwell, 2011). In a review of the importance of variability in the prediction of hypertension, it has been reported that patients with consistently normal blood pressure, even that which is slightly elevated, are reported to have fewer vascular events, whereas it seems that higher variability implies increased risk (Rothwell, 2010). In contrast, there is some evidence that increased variability in other aspects of underlying cardiovascular functioning may be adaptive. An association between increased

risk of hypertension and *lower* heart rate variability (HRV) has been reported (Schroeder et al., 2003). Schroeder et al. (2003) reported a continuous relationship between blood pressure and HRV, with strongest observed associations at lower levels of blood pressure, suggesting that normative levels of cardiovascular variability can provide meaningful trends to examine.

It has been posited that decreased HRV could reflect an inability to respond to stress with physiological flexibility, resulting in more rigid physiological processes, thereby making individuals more vulnerable to disease through the development of hypertension or CAA (Horsten et al., 1999). The findings reported by Horsten et al. (1999) further suggested a mediating role for decreased HRV in the associations between disease vulnerability and psychosocial variables, including social isolation, low social support, and aspects of anger. Similar studies have found inverse relationships between aspects of NA and HRV (Bleil, Gianaros, Jennings, Flory, & Manuck, 2008), suggesting a role for individual differences in personality in cardiovascular variability.

The research on cardiovascular variability appears to align with principles of homeostasis, whereby maintenance of equilibrium is best for physiological states, but some response to daily life stress is still desirable. Research concerning cardiovascular variability has been predominately limited to the examination of patients with pre-existing hypertension, or of middle-aged and older participants, who by nature of their age may already be at increased risk of developing hypertension or CVD. It has been suggested that in older people, or those with long-standing hypertension, increased variability of SBP may be due to increased arterial stiffness, as a result of vascular ageing (Izzo et al., 2008, p. 128). Variability of 24-hour SBP in a sample of young, healthy participants showed that increased variability that was independently associated with arterial stiffness (Kotsis et al.,

2011). Thus, the observation of trends in younger people could be interesting if they were found to be associated with other recognised disease vulnerability characteristics, such as individual differences in personality traits related to NA. Some caution should be exercised in the direct comparison of blood pressure and HRV studies as they do monitor different aspects of cardiovascular functioning. Nonetheless, what these studies suggest is that not only can increased variability of cardiovascular functioning be detrimental to health, but diminished cardiovascular functioning too could have negative health consequences.

A large number of research studies have shown that several personality traits are related to increased risk of physical illness, with domains relating to NA consistently providing much of the evidence (Smith, Traupman, Uchino, & Berg, 2010). As described in Chapter 1, the dimension of neuroticism is a particularly influential personality trait included under the umbrella of NA. It is characteristic of persons high in neuroticism to respond with negative emotions more frequently, and with greater intensity when they experience stressful life events (Lahey, 2009), and has been associated with negative cardiovascular health outcomes (see Smith & MacKenzie, 2006; Suls & Bunde, 2005). While several studies have suggested that neuroticism represents a risk factor for CVD morbidity and mortality (Suls & Bunde, 2005), it has been suggested that other personality traits, such as extraversion, may have protective elements (R. S. Wilson et al., 2005). Some possible health-protective benefits have also been attributed to greater trait conscientiousness. Higher conscientiousness was found to be protective against all-cause mortality in men (Taylor et al., 2009), and related to greater longevity than others lower in conscientiousness (H. S. Friedman et al., 1993). Recently, it was found that greater conscientiousness predicted greater recovery from, but not reactivity to, negative emotional pictures (Javaraš et al., 2012).

While extensions to the conventional CVR hypothesis (Obrist, 1976) have suggested that both exaggerated *and* diminished patterns of cardiovascular responding may contribute to poor cardiovascular health (Lovallo, 2011), observation of blunted cardiovascular functioning has thus far been limited to laboratory-style protocols. Recently, it has suggested that it may be that blunted physiological responses are manifest in particular behaviours or personality traits (Phillips et al., in press). Several personality dispositions, particularly aspects of NA, have been shown to be associated with negative cardiovascular outcomes, included blunted reactions to stress (e.g., Phillips, 2011). Thus the following research question was tested: Over an extended period of monitoring in real life settings, can individual differences in Five-Factor personality traits moderate diurnal patterns of cardiovascular functioning in young, healthy adults?

Method

Participants

Data were collected for 80 undergraduate participants (60 women and 20 men). Consideration of possible confounding factors affecting cardiovascular functioning led to the exclusion of participants according to the following criteria: BMI greater than 30 (Dyer, Stamler, Garside, & Greenland, 2004); age above 29 years (Franklin et al., 2001); a personal history of hypertension (Sowers, Epstein, & Frohlich, 2001); and baseline blood pressure greater than 140/90 mmHg. Additionally, estimates of compliance with the protocol suggested that only those participants who had fewer than eight missing entries into the cardiovascular protocol should be included in the final sample. Participant exclusion yielded an available sample of $n = 58$ (44 women, 14 men). Gender differences

in cardiovascular functioning (e.g., Mendelsohn & Karas, 2005), so it was decided that data for men and women should be analysed separately. The sample size for men was underpowered for statistical analyses, thus the inferential investigations focused on the data available for 44 women. All reported inferential statistics refer to this sample of women, but where appropriate, descriptive statistics for men are reported.

Therefore, in summary, for inferential analyses the sample comprised 44 women, ranging in age from 17.67 to 26.67 years ($M = 19.25$, $SD = 1.58$ years), with a BMI of less than 30 ($M = 21.93$, $SD = 2.82$ kg/m²). Of these 44 women, four were self-reported smokers. These four smokers were included in the final sample as their inclusion was not found to affect the overall results.

Self-Monitoring Design

The protocols of previous research studies interested in examining cardiovascular functioning and individual differences in personality have largely been limited to laboratory-based or 24-hour ambulatory blood pressure protocols. Although enhancing methodological control, laboratory designs lack a degree of ecological validity. As such, laboratory-measured blood pressure may not always accurately reflect real life stress responding. Studies employing the use of ambulatory devices allow for the assessment of the impact of daily life experiences on blood pressure, and can observe trends that may not be appreciable with the use of laboratory designs (Steptoe, 2005). Ambulatory blood pressure measurement involves the continuous monitoring of blood pressure usually for 24 hours, typically at 15-20 minute intervals during sleep and wakefulness. It is common in clinical contexts to employ ambulatory approaches in order to glean estimates of blood pressure that are not contaminated by the effects of “white coat hypertension”. The white

coat hypertension effect refers to higher rates of observed blood pressure which may be observed when measured by a physician or nurse, or in medical environments relative to blood pressure measured in normal settings (Verdecchia & Staessen, 2002). Nonetheless, while providing a rich source of physiological data from naturalistic settings, ambulatory approaches have their own drawbacks. Monitors are attached to participants by researchers or clinicians, and remain in place (on the participant's arm most often) until removed by the researcher or clinician 24 hours later. Measurements of blood pressure are automatically taken at intervals, regardless of what the participant is doing (e.g., driving, walking, or sitting at a desk). As the monitor must remain attached to the participant during this time, they are unable to partake in certain activities (e.g., swimming or showering). In many psychological studies, this level of restriction may constitute an environmental stressor that complicates the assessment of those very associations between environmental stress and cardiovascular stress responding. Finally, the demanding and sometimes intrusive nature of ambulatory protocols mean that it is not practical to have people collect or monitor their blood pressure data for more than one consecutive 24-hour period. In summary, for research questions interested in more extended periods of time, self-monitoring designs are preferable for these practical and behavioural reasons.

Similar to ambulatory designs, self-monitoring designs use portable blood pressure monitors, but afford greater autonomy to the participant. Conveniently, the apparatus may be removed when blood pressure is not being measured. In this way, participants are not precluded from any daily activities as they might be with ambulatory monitoring, improving the experience for the participant. Throughout the self-monitoring protocol, participants may choose (within pre-determined intervals) the timing of blood pressure measurement, ensuring minimal disruption to normal routines; an essential feature of

protocols longer than 24 hours. Additionally, any anxiety or embarrassment due to ambulatory monitors activating at momentary inconvenient times is limited. In this way, reliability of measurement could be increased using the extended self-monitoring protocols for blood pressure measurement. Posture of the participant during measurement may be more consistent and uniform, as participants are able to take the time to assume the appropriate posture for the accurate measurement of blood pressure. Participants are trained in use of the portable blood pressure apparatus, and as such are required to sit quietly while using the monitors to take a reading of blood pressure. Self-monitoring designs have been employed successfully in previous research investigating the activities and responses of working adults over five consecutive days (Evans & Steptoe, 2001). A number of other studies have employed field-based methods to collect other physiological indices of stress, such as cortisol (e.g., Brant, Wetherell, Lightman, Crown, & Vedhara, 2010; Lovell, Moss, & Wetherell, 2011).

Measures

Cardiovascular responses.

OMRON 637 IT monitors (OMRON Healthcare UK, Henfield, UK) were used to measure blood pressure. These wrist-operated monitors allow participants to measure cardiovascular function accurately using the oscillometric principle (Marey, 1876). The OMRON monitors have been validated using international standardization protocols (Eckert, Gleichmann, Zagorski, & Klapp, 1997; S. Watson, Wenzel, Di Matteo, Meier, & Lüscher, 1998), and used in previous research studies (e.g., Hughes & Howard, 2009). Blood pressure is determined by recording of oscillations of blood flow in the sphygmomanometer cuff during gradual deflation of the cuff from a pressure that is in

excess of SBP to a pressure that is below that of DBP, over a period of approximately 30 seconds (Pickering et al., 2005). Estimates of SBP and DBP are derived using an empirically derived algorithm.

To correctly measure blood pressure, the monitor is attached by the participant to their non-dominant wrist while in a seated position. The monitor is activated by the participant and the non-dominant arm is positioned across the chest, such that the monitor is level with the heart. A criticism of some wrist-operated monitors has been that systematic error may be introduced when the wrist is not at heart height during measurement (e.g., Mitchell, Parlin, & Blackburn, 1964). An advantage of the OMRON monitors used in the current study is that an inbuilt sensor in the monitor only commences blood pressure measurement when it has detected that the wrist is in the correct position relative to heart height, thereby reducing the risk of introducing such systematic error.

Psychometric testing.

NEO Five-Factor Inventory. Personality was assessed using the NEO Five-Factor Inventory (NEO FFI; Costa & McCrae, 1992), a well-standardised psychometric instrument that has demonstrated good internal consistency in the past. Five possible 12-item subscales are derived from this instrument resulting in individual scores for neuroticism, extraversion, conscientiousness, agreeableness, and openness. Participants responded to items using a five-point Likert scale with responses ranging from *strongly disagree* (1) to *strongly agree* (5). Scores ranged from a possible 12 to 60 for each subscale (see Table 1 for descriptive statistics). Reliability was good for neuroticism ($\alpha = .85$), extraversion ($\alpha = .78$), and conscientiousness ($\alpha = .87$), while it was lower for the other subscales of agreeableness ($\alpha = .65$), and openness ($\alpha = .69$).

Table 1

Descriptive and Reliability Statistics for Psychometric Variables.

Variable	Women (<i>n</i> = 44)					Men (<i>n</i> = 14)				
	α	Mean	<i>SD</i>	Min	Max	α	Mean	<i>SD</i>	Min	Max
Neuroticism ^a	.85	3.12	0.64	2.00	4.42	.85	2.74	0.66	1.67	4.00
Extraversion ^a	.78	3.62	0.49	2.42	4.50	.70	3.49	0.45	2.92	4.58
Conscientiousness ^a	.87	3.28	0.62	2.08	4.75	.89	3.19	0.72	2.17	4.25
Agreeableness ^a	.65	3.63	0.47	2.58	4.50	.70	3.48	0.51	2.58	4.42
Openness ^a	.69	3.10	0.47	2.25	4.33	.75	3.27	0.54	2.33	4.33
Support number ^b	.91	5.03	0.99	1.67	6.00	.96	5.33	0.47	4.50	6.00
Support satisfaction ^c	.95	4.48	1.95	1.17	9.00	.78	4.65	2.14	1.83	8.83
Perceived stress ^d	.78	1.99	0.53	1.07	3.57	.58	1.76	0.43	1.07	2.43
Trait anxiety ^e	.88	2.14	0.44	1.40	3.30	.79	2.15	0.36	1.60	2.65
Hostility ^f	.84	0.46	0.15	0.06	0.72	.85	0.43	0.18	0.14	0.71
Cynical distrust ^f	.67	0.41	0.24	0.00	0.88	.80	0.42	0.31	0.00	0.88
Cynicism ^f	.71	0.53	0.21	0.00	0.92	.81	0.51	0.26	0.15	0.85
State anger ^g	.95	1.27	0.49	1.00	3.67	.81	1.11	0.16	1.00	1.60
Trait anger ^g	.85	2.13	0.56	1.10	3.80	.91	1.87	0.53	1.00	2.90
Anger control in ^g	.90	2.52	0.62	1.38	4.00	.91	2.51	0.72	1.00	3.63
Anger expression out ^g	.61	1.96	0.44	1.13	3.00	.43	1.86	0.36	1.25	2.50
Anger expression in ^g	.81	2.31	0.61	1.38	4.00	.79	2.24	0.58	1.50	3.25
Anger control out ^g	.89	2.69	0.61	1.63	4.00	.85	2.90	0.59	2.00	3.88
Aggression ^h	.89	2.50	0.57	1.52	3.93	.95	2.55	0.67	1.66	3.86
University support ⁱ	.77	3.24	0.38	2.60	4.00	.66	3.24	0.39	2.60	4.00

Table 1 continued

Social provisions ^l	.88	3.42	0.29	2.50	3.83	.95	3.12	0.47	2.25	3.79
Self efficacy ^k	.88	2.85	0.47	1.60	3.60	.80	2.79	0.37	2.10	3.30
Positive affect ^l	.87	3.54	0.57	2.40	4.80	.78	3.60	0.46	2.80	4.40
Negative affect ^l	.64	2.43	0.88	1.00	4.60	.24	2.16	0.69	1.40	3.80

Note. SD = standard deviation; α = Chronbach's alpha coefficient; Min = minimum; Max = maximum

^aMeasured using mean item scores from the appropriate subscale of the NEO FFI (Costa & McCrae, 1992).

^bMeasured using mean item scores from the *N* subscale of the SSQ6 (Sarason, Sarason, Shearin, & Pierce, 1987).

^cMeasured using mean item scores from the *S* subscale of the SSQ6 (Sarason et al., 1987)

^dMeasured using mean item scores from the PSS (S. Cohen, Kamarck, & Mermelstein, 1983).

^eMeasured using mean item scores from the appropriate subscale of the State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, & Lushene, 1977).

^fMeasured using mean item scores from the item Cook-Medley Hostility Inventory (Cook & Medley, 1954), or its cynical distrust (Greenglass & Julkunen, 1989), or cynicism (Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989) subscales.

^gMeasured using mean item scores from the appropriate subscale of the State-Trait Anger Expression Inventory 2 (Spielberger, 1999)

^hMeasured using mean item scores from the Buss Perry Aggression Questionnaire (Buss & Perry, 1992)

ⁱMeasured using mean item scores from the Social Support at University Scale (Hughes, 2007b).

^jMeasured using mean item scores from the Social Provisions Scale (Cutrona & Russell, 1987).

^kMeasured using mean item scores from the General Self-Efficacy Scale (Schwarzer & Jerusalem, 1995).

^lMeasured using mean item scores from the Positive and Negative Affect Scale (D. Watson, Clark, & Tellegen, 1988).

Perceived stress scale. The 14-item Perceived Stress Scale (PSS; S. Cohen et al., 1983) measures frequency of stressful thoughts and feelings experienced within a pre-defined period. Participants were asked to provide an estimate of the frequency of thoughts and feelings within the previous month that were be associated with stress. A sample item included “in the last month, how often have you felt nervous and stressed”? Responses were scored on a five-point Likert scale, from *never* (0) feeling or thinking that way, to *very often* (4) feeling or thinking that way. Higher scores indicated more perceived stress, and oppositely keyed items were reverse-scored accordingly. Reliability for the PSS was found to be good ($\alpha = .78$).

Sarason’s social support. Six two-part items assessed both the number of people providing support, and the quality of support provided using Sarason’s six-item social support questionnaire (SSQ6; Sarason et al., 1987). Sample items include “whom can you really count on to distract you from you worries when you feel under stress”, and “who accepts you totally, including both your worst and best points”? Space was available for participants to list the initials of up to nine people who provide support, or an option to state that no one provides support. Additionally, participants are asked to describe their relationship to the person providing support (e.g., mother, brother, friend, girlfriend). Participants are also asked to rate their satisfaction with the support received using a six-point Likert scale with options ranging from *very dissatisfied* (1) to *very satisfied* (6). Reliability for the number of people providing support ($\alpha = .91$), and the satisfaction with social support ($\alpha = .95$) subscales of the SSQ were excellent.

Behavioural responses.

In addition to the recording of cardiovascular responses, participants were asked to appraise their perceived levels of stress, social contact, and positive mood in the 15 minutes prior to the measurement of blood pressure (see Appendix A for a sample behavioural diary section). Social contact was subjectively rated using of three response options; low medium or high; while stress and positive mood were appraised using a four point Likert scale, ranging from *not at all* (1) to *extremely so* (4). Reliability for these behavioural questions was varied and generally quite poor (see Table 2). Participants received text messages from the experimenter to remind them to take blood pressure measurement and to return the blood pressure monitors and accompanying questionnaires. Text messages were sent automatically at 1pm on Wednesdays and at 11am on Mondays following completion of the protocol.

Procedure

Participants attended a briefing session in an on-campus laboratory building, where they were trained in the operation of the blood pressure monitors and completed some psychometric measures. Participants were asked to follow the study protocol for five consecutive days. The period encompassed three nominal weekdays (Wednesday, Thursday and Friday) and two nominal weekend days (Saturday and Sunday), employing a procedure described previously elsewhere (i.e., Evans & Steptoe, 2001). Participants were asked to take two measures of blood pressure, five times daily within pre-agreed intervals spanning approximately 90 minutes. Consistent with a previous study (Evans & Steptoe, 2001), daytime measures were defined as occurring before 18:00, including those measures taken between 08:00 and 09:30, between 12:30 and 14:00, and between 16:00

and 17.50. Two further measurement periods were defined as evening measures, taken between 19:00 and 20:50, and just before going to sleep at night.

Table 2

Descriptive and Reliability Statistics for Behavioural Diary Variables (Women, n = 44)

Day	Social contact			Stress			Positive mood		
	Mean	SD	α	Mean	SD	α	Mean	SD	α
Wednesday	1.95	0.39	.51	1.63	0.48	.64	2.81	0.59	.65
Daytime ^a	1.96	0.47	.35	1.62	0.56	.53	2.77	0.53	.36
Evening ^b	1.95	0.59	.50	1.65	0.66	.47	2.88	0.85	.72
Thursday	1.82	0.39	.47	1.66	0.52	.74	2.62	0.58	.77
Daytime ^a	1.73	0.41	.41	1.75	0.53	.52	2.57	0.67	.70
Evening ^b	1.89	0.56	.34	1.57	0.65	.58	2.63	0.70	.43
Friday	1.70	0.40	.65	1.48	0.45	.57	2.71	0.60	.75
Daytime ^a	1.64	0.46	.58	1.52	0.49	.37	2.66	0.60	.61
Evening ^b	1.77	0.48	.09	1.45	0.62	.70	2.76	0.77	.61
Saturday	1.77	0.42	.63	1.56	0.46	.69	2.59	0.59	.48
Daytime ^a	1.72	0.45	.36	1.50	0.51	.69	2.54	0.72	.34
Evening ^b	1.82	0.61	.47	1.65	0.63	.34	2.67	0.65	.35
Sunday	1.77	0.46	.70	1.55	0.54	.80	2.77	0.64	.81
Daytime ^a	1.73	0.44	.47	1.49	0.51	.76	2.67	0.69	.77
Evening ^b	1.80	0.67	.72	1.60	0.77	.83	2.86	0.76	.69

Note. SD = standard deviation; α = Chronbach's alpha coefficient.

^aDaytime measures comprised from three measurements taken at 08:00-09:30, 12:30-14:00, and 16:00-17:50.

^bEvening measures comprised from two measurements taken at 19:00-20:50 and bedtime

Overview of Analyses

Mean levels of SBP, DBP, and HR were computed for daytime and evening periods for each of the five study days (for descriptive and reliability statistics see Table 3 for women, and Table 4 for men). Reliability was found to be good for each of the cardiovascular parameters. It was determined that the first day of participation in the study (Wednesday) would serve as a practice day for all participants, so that they could familiarise themselves with the procedure and with the monitoring of cardiovascular functioning. Accordingly, data collected from this day was not used in inferential analysis, and it served as a baseline day.

In order to control for initial levels of blood pressure, an estimation of baseline values of mean SBP and DBP were obtained from the first day of the study protocol (i.e. Wednesday), and entered as a control covariate in subsequent inferential analyses. The Law of Initial Values, initially described by Wilder (1962), asserts that a phasic physiological response is dependent on the initial baseline value, such that increases in baseline blood pressure may limit or constrain further incremental responses and may enhance subsequent decreased responses (Berntson, Uchino, & Cacioppo, 1994). It was found that the current study's results were not altered by the inclusion of baseline values of blood pressure, thus initial values of SBP or DBP did not prejudice the findings.

As described previously, participants exhibiting higher than normal mean blood pressure on Wednesday (mean SBP > 140, DBP > 90mmHg) were excluded from subsequent analysis. An index of the *shift* in mean blood pressure, from daytime to evening periods, was calculated by subtracting mean daytime measures from mean evening measures. Positive values indicated increased blood pressure in evenings relative

Table 3

Descriptive and Reliability Statistics for the Cardiovascular Parameters of Women (n = 44)

Day	SBP (mmHg)			DBP (mmHg)			HR (bpm)		
	Mean	<i>SD</i>	α	Mean	<i>SD</i>	α	Mean	<i>SD</i>	α
Wednesday	114.82	7.50	.73	71.00	6.99	.83	80.42	9.67	.79
Daytime ^a	115.49	8.41	.76	71.41	7.62	.84	81.17	10.12	.80
Evening ^b	113.84	8.76	.81	70.40	7.06	.79	79.16	11.32	.86
Thursday	114.17	7.81	.80	71.02	6.99	.85	80.91	8.77	.73
Daytime ^a	114.19	8.07	.82	71.53	7.64	.84	82.93	9.60	.68
Evening ^b	114.22	9.75	.82	70.19	7.03	.80	77.95	10.03	.88
Friday	113.09	7.87	.83	69.52	5.71	.68	79.01	8.24	.70
Daytime ^a	112.58	7.59	.82	69.39	5.75	.74	80.42	8.72	.75
Evening ^b	113.68	9.88	.83	69.94	7.80	.78	77.02	10.67	.78
Saturday	112.92	7.71	.80	69.93	7.78	.83	79.41	10.41	.83
Daytime ^a	111.92	8.21	.85	69.39	7.59	.83	80.49	10.77	.81
Evening ^b	114.59	9.46	.79	70.28	9.23	.82	77.76	12.77	.88
Sunday	114.27	8.97	.91	71.50	7.29	.82	79.52	8.83	.83
Daytime ^a	112.74	9.78	.89	70.49	8.76	.79	80.29	9.68	.84
Evening ^b	116.07	10.68	.92	72.67	9.73	.82	78.32	11.29	.80

Note. *SD* = standard deviation; α = Chronbach's alpha coefficient.

^aDaytime measures derived from measurements taken at 08:00-09:30, 12:30-14:00, and 16:00-17:50 phases.

^bEvening measures derived from measurements taken at 19:00-20:50 and bedtime phases.

Table 4

Descriptive and Reliability Statistics for Cardiovascular Parameters of Men (n = 14)

Day	SBP (mmHg)			DBP (mmHg)			HR (bpm)		
	Mean	<i>SD</i>	α	Mean	<i>SD</i>	α	Mean	<i>SD</i>	α
Wednesday	122.49	6.75	.12	75.17	5.08	.70	72.33	6.23	.58
Daytime ^a	122.11	7.65	.42	75.95	6.77	.78	70.52	72.29	.61
Evening ^b	123.05	7.02	.72	73.36	4.74	.62	70.52	7.29	.69
Thursday	123.24	9.65	.74	75.83	4.94	.45	71.56	4.93	.29
Daytime ^a	121.93	7.87	.68	76.74	8.45	.81	72.58	5.46	.38
Evening ^b	125.13	13.37	.68	74.89	5.01	.10	70.00	7.30	.75
Friday	120.70	8.16	.85	74.71	5.10	.73	69.96	6.43	.58
Daytime ^a	121.27	8.92	.77	75.80	3.65	.13	69.88	5.34	.46
Evening ^b	120.34	10.25	.91	73.38	7.98	.81	69.73	12.22	.89
Saturday	120.62	7.09	.73	73.81	5.00	.61	69.47	7.61	.76
Daytime ^a	121.18	7.90	.76	74.34	5.84	.73	70.53	8.57	.72
Evening ^b	120.20	7.62	.79	73.21	5.81	.76	67.68	7.20	.76
Sunday	119.36	7.79	.72	72.58	5.51	.63	70.72	8.11	.68
Daytime ^a	118.42	7.62	.62	71.43	5.93	.48	70.45	8.41	.81
Evening ^b	120.98	9.79	.50	74.56	6.98	.73	71.29	11.83	.84

Note. *SD* = standard deviation; α = Chronbach's alpha coefficient.

^aDaytime measures comprised from three measurements taken at 08:00-09:30, 12:30-14:00, and 16:00-17:50.

^bEvening measures comprised from two measurements taken at 19:00-20:50 and bedtime.

to the corresponding daytime, while negative values indicated decreased blood pressure during evenings relative to daytime periods.

To determine the role of personality traits in the transition of blood pressure from daytime to evening periods, shift in SBP and DBP responses were each included in a 4 (day; Thursday to Sunday) \times 1 analysis of covariance (ANCOVA), with baseline blood pressure and mean personality traits entered as covariates. As the day effects in each ANCOVA were based on four within-group levels, assumptions of sphericity were tested using Mauchly's tests. Degrees of freedom were corrected using Greenhouse-Geisser corrections where assumptions of sphericity were not met. Further, given the likelihood of fluctuations in cardiovascular function across days, day effects were examined using both non-linear (e.g., quadratic or cubic) and linear within-group contrasts to assess possible polynomial trends. Significant effects were followed up with correlations to test the direction of observed interactions between continuous covariates and cardiovascular function.

A measure of blood pressure variability was computed by deriving the variability coefficient (as described previously by Kikuya et al., 2008), achieved by dividing the standard deviation of daily SBP or DBP by the corresponding day's mean SBP or DBP. This created an impression of the within-day fluctuations in blood pressure over each of the four days of the study. Coefficient of variation has shown similar prognostic value to within-subject standard deviations of blood pressure (Kikuya et al., 2008).

Separate stepwise linear regressions were used evaluate if personality traits predicted blood pressure variability on each of the four days of the study. The criterion variables entered in each (separate) stepwise regression were SBP or DBP variability on each of the four days of the study. Psychometric predictors entered simultaneously into the

regression equation were neuroticism, extraversion, and conscientiousness. Some additional psychometric predictors previously known to be associated with blood pressure were also entered into the stepwise regression model, including mean perceived stress scores from the PSS (S. Cohen et al., 1983), mean satisfaction with social support scores, from the SSQ6 (Sarason et al., 1987), and two other known correlates of blood pressure; BMI and age of participants.

Effect sizes are presented as partial η^2 for analysis of variance (ANOVA) effects, r for correlations, and β for regression coefficients. Eta-squared values of .04, .25, and .64, and r - and β -values of .1, .25, and .37 were taken to represent small, medium, and large effect sizes respectively (J. Cohen, 1988, 1992).

Results

Descriptive Statistics

Descriptive and reliability statistics for all psychometric variables measured are presented for women and men (see Table 1). Descriptives for cardiovascular parameters for women and men are also presented (See Table 4).

Behavioural Diaries

Responses to the behavioural diary items suffered from a limited range and poor internal reliability. Due to inferential interest in diurnal variations in cardiovascular functioning, paired samples t tests, adjusted for multiple comparisons using Bonferroni corrections, were conducted to assess the differences between daytime and evening ratings of stress, social contact, and positive mood. The results showed no significant differences

between diurnal subjective appraisals of social contact, stress, or positive mood for the sample of women ($N = 44$; $p > .06$). One potential difference between subjectively reported stress during daytimes and evenings on Thursday, $t(41) = 2.13$, $p = .04$, did not remain significant after the application of a Bonferroni correction to adjust for Family-wise error rate. Investigative analysis of independent samples t tests with median split dimensions of the Five-Factor model likewise did not reveal any significant differences in subjective appraisals of social contact, stress, or positive mood, following application of a Bonferroni procedure.

Daytime-Evening Shifts

Neuroticism.

SBP. The results of a 4×1 repeated measures ANCOVA with neuroticism and initial SBP entered as covariates, revealed that although a day \times neuroticism interaction was not found to be significant using within subjects effects, $F(2.60, 101.43) = 2.07$, $p = .12$, partial $\eta^2 = .10$, it was significant at the level of the quadratic function, $F(1, 39) = 5.02$, $p = .03$, partial $\eta^2 = .11$. Overall correlations between neuroticism and shift in SBP ($r = -.20$, $p = .20$) were inverse, suggesting that higher neuroticism was associated with lower shift in SBP from daytime to evening (i.e., those lower in neuroticism had greater mean increases in SBP during evening times relative to daytimes). Correlations between continuous neuroticism scores and change in SBP for individual days showed the association to be strongest on Sunday, relative to the other days, with a moderate inverse relationship ($r = -.30$, $p = .049$). Higher neuroticism was associated with low, or blunted shift in SBP from daytime to evening compared to those lower in neuroticism on Sunday (see Figure 1a

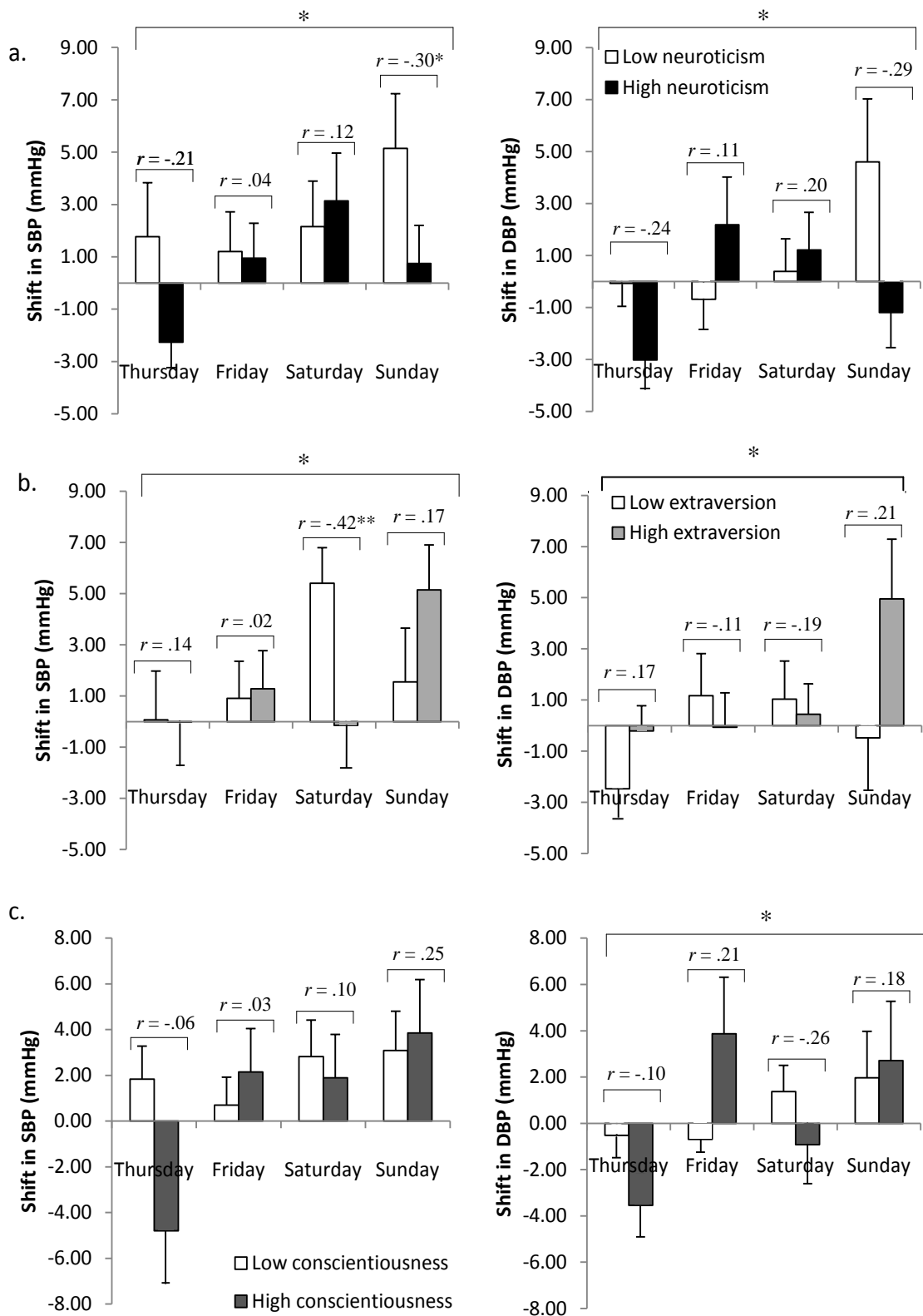


Figure 1. Interactions between shift in SBP and DBP by day with individual differences in personality. r = Pearson's correlation coefficients for personality and mean shift in blood pressure per day. Asterisks denote significance of polynomial functions for day \times

personality trait shift in blood pressure; values for above zero indicate higher blood pressure during evenings relative to daytimes, whereas values below zero indicate lower blood pressure during evenings relative to daytimes. Median splits are for illustrative purposes (higher neuroticism, $n = 25$, lower neuroticism, $n = 19$; higher extraversion, $n = 22$, lower extraversion, $n = 22$; higher conscientiousness, $n = 12$, lower conscientiousness, $n = 32$). Error bars denote standard error of the mean.

* $p < .05$. ** $p < .01$.

which uses median splits for illustrative purposes). No significant main effects were observed for day of shift, $F(2.60, 101.43) = 0.30$, $p = .79$, partial $\eta^2 = .01$; neuroticism, $F(1, 39) = 1.28$, $p = .26$, partial $\eta^2 = .03$; or initial SBP, $F(1, 39) = 0.4$, $p = .84$, partial $\eta^2 = .001$. No initial SBP \times neuroticism interaction was observed, $F(2.60, 101.43) = 0.48$, $p = .67$, partial $\eta^2 = .01$. Mean values for shift in blood pressure are contained in Table 5.

DBP. Results of a 4×1 repeated measures ANCOVA for DBP revealed a significant day \times neuroticism interaction at the level of a quadratic function was observed, $F(1, 39) = 6.84$, $p = .01$, partial $\eta^2 = .15$, despite non-significant within subjects effects, $F(2.26, 88.17) = 2.83$, $p = .06$, partial $\eta^2 = .07$. Correlations over the four days of the protocol showed an inverse relationship between neuroticism and DBP shift ($r = -.15$, $p = .33$). Correlations between shift in DBP on each day of the study and neuroticism were all relatively small and not statistically significant. The strongest correlation observed was between neuroticism and shift in Sunday DBP, such that the correlation was inverse in nature ($r = -.29$, $p = .06$). Higher neuroticism was associated with blunted shift from daytime to evening periods, especially for Sunday (see Figure 1a). No other significant main effects for day of measurement, $F(2.26, 88.17) = 0.88$, $p = .43$, partial $\eta^2 = .02$; neuroticism, $F(1, 39) = 0.79$, $p = .38$, partial $\eta^2 = .02$; or initial DBP, $F(1, 39) = 0.001$, $p = .97$, partial $\eta^2 < .001$, were found. A day \times initial DBP interaction was also found not to be significant, $F(2.26, 88.17) = 0.52$, $p = .62$, partial $\eta^2 = .01$.

Table 5

Descriptive Statistics for Shift and Variability in Blood Pressure and Heart Rate (HR)

Day	SBP (mmHg)				DBP (mmHg)				HR (bpm)			
	Shift ^a		Variability ^b		Shift ^a		Variability ^b		Shift ^a		Variability ^b	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Wednesday	-1.63	8.56	0.06	0.04	-1.01	5.67	0.07	0.04	-1.77	9.50	0.10	0.05
Thursday	+0.03	8.44	0.06	0.03	-1.34	5.06	0.08	0.04	-4.97	8.67	0.12	0.06
Friday	-1.09	6.80	0.06	0.03	+0.55	7.01	0.09	0.04	-3.40	9.23	0.11	0.05
Saturday	+2.56	8.27	0.06	0.04	+0.73	6.18	0.09	0.06	-2.66	10.51	0.11	0.06
Sunday	+3.30	9.10	0.07	0.04	-2.17	10.51	0.10	0.06	-1.97	10.42	0.10	0.06

^aShift = arithmetic difference between evening and daytime measured SBP, DBP, and HR.^bVariability = *SD* of blood pressure and heart rate divided by corresponding mean value.

HR. No significant main effects for day, $F(3, 117) = 0.30, p = .82, \text{partial } \eta^2 = .01$; neuroticism, $F(1, 39) = 0.30, p = .88, \text{partial } \eta^2 = .001$; or initial HR, $F(1, 39) = 1.59, p = .21, \text{partial } \eta^2 = .04$, were found. Additionally, no day \times neuroticism, $F(3, 117) = 0.20, p = .88, \text{partial } \eta^2 = .01$, or day \times initial DBP, $F(3, 117) = 0.19, p = .91, \text{partial } \eta^2 = .01$, interaction effects were found to reach statistical significance.

Extraversion.

SBP. A significant day \times extraversion interaction was observed for SBP, $F(3, 117) = 2.99, p = .03, \text{partial } \eta^2 = .07$, which was also found to be cubic in nature, $F(1, 39) = 5.85, p = .02, \text{partial } \eta^2 = .13$. The relationship between extraversion and shift in SBP over the four days of the study were found to be characterised by a small, inverse correlation ($r = -.03, p = .85$). Associations between shift in SBP and extraversion were particularly apparent for Saturday ($r = -.42, p = .01$), such that participants higher in extraversion had lower, or more diminished SBP during evening times relative to daytimes (see Figure 1b). No significant main effects for day for SBP, $F(3, 117) = 1.03, p = .38, \text{partial } \eta^2 = .02$; extraversion, $F(1, 39) = 0.27, p = .87, \text{partial } \eta^2 = .001$; or initial SBP, $F(1, 39) = 0.20, p = .89, \text{partial } \eta^2 = .001$, were observed. A day \times initial SBP interaction was not statistically significant, $F(3, 117) = 0.37, p = .77, \text{partial } \eta^2 = .01$.

DBP. A day \times extraversion interaction was not found to be significant using conventional within subjects effects, $F(2.24, 87.42) = 1.88, p = .15, \text{partial } \eta^2 = .05$, although a polynomial effect which was quadratic in nature was statistically significant, $F(1, 39) = 4.39, p = .04, \text{partial } \eta^2 = .10$. Correlations between extraversion and mean DBP shift over the four days showed a small positive trend ($r = .06, p = .71$), which was not found to be statistically significant for any one particular day (see Figure 1b), although the

correlation was largest on Sundays ($r = .21, p = .17$). No significant main effects for day, $F(2.24, 87.42) = 0.32, p = .75$, partial $\eta^2 = .01$; extraversion, $F(1, 39) = 0.43, p = .52$, partial $\eta^2 = .01$; or initial DBP, $F(1, 39) = 0.11, p = .74$, partial $\eta^2 = .003$, were found. A day \times initial DBP interaction was not significant, $F(2.24, 87.42) = 0.85, p = .44$, partial $\eta^2 = .02$.

HR. No significant main effects for day, $F(3, 117) = .08, p = .97$, partial $\eta^2 = .002$; extraversion, $F(1, 39) = 1.10, p = .30$, partial $\eta^2 = .03$; initial HR, $F(1, 39) = 2.00, p = .17$, partial $\eta^2 = .05$, were found. No significant day \times extraversion, $F(3, 117) = 0.28, p = .84$, partial $\eta^2 = .01$, or day \times initial HR, $F(3, 117) = 0.22, p = .88$, partial $\eta^2 = .01$, interaction effects were observed.

Conscientiousness.

SBP. For SBP, no significant day \times conscientiousness interaction was found, $F(2.54, 98.94) = 0.46, p = .71$, partial $\eta^2 = .01$. At the level of polynomial trends, a linear day \times conscientiousness interaction trended towards statistical significance, but did not reach it, $F(1, 39) = 3.84, p = .06$, partial $\eta^2 = .09$, (see Figure 1c). No significant effects for day, $F(2.54, 98.94) = 0.46, p = .68$, partial $\eta^2 = .01$; conscientiousness, $F(1, 39) = 1.36, p = .25$, partial $\eta^2 = .03$; initial SBP, $F(1, 39) = 0.21, p = .65$, partial $\eta^2 = .01$; or day \times initial SBP, $F(2.54, 98.94) = 0.69, p = .56$, partial $\eta^2 = .02$, were found to be significant.

DBP. For DBP, a significant day \times conscientiousness effect was observed, $F(1, 39) = 5.00, p = .03$, partial $\eta^2 = .11$, significant at the level of a cubic effect, despite not being significant at the level of the within-subject effects, $F(2.22, 86.52) = 2.00, p = .14$, partial $\eta^2 = .50$. Correlations between mean SBP shift over four days and conscientiousness were small and positive ($r = .09, p = .58$). Individually by day (see

Figure 1c), correlations between DBP and conscientiousness were not statistically significant, with an overall small positive trend over the four days ($r = .09$, $p = .58$), with the strongest correlation for Saturday ($r = -.26$, $p = .10$). No significant for day, $F(2.22, 86.52) = 0.85$, $p = .85$, partial $\eta^2 = .01$; conscientiousness, $F(1, 39) = 0.27$, $p = .61$, partial $\eta^2 = .01$; initial DBP, $F(1, 39) = 0.05$, $p = .83$, partial $\eta^2 = .001$; or day \times initial DBP, $F(2.22, 86.52) = 0.57$, $p = .59$, partial $\eta^2 = .01$, effects were observed.

HR. No effects for day, $F(3, 117) = 0.47$, $p = .70$, partial $\eta^2 = .01$; conscientiousness, $F(1, 39) = 2.48$, $p = .11$, partial $\eta^2 = .06$; day \times conscientiousness interaction, $F(3, 117) = 0.35$, $p = .79$, partial $\eta^2 = .01$; day \times initial DBP, $F(3, 117) = 0.21$, $p = .89$, partial $\eta^2 = .01$; or initial DBP, $F(1, 39) = 1.46$, $p = .24$, partial $\eta^2 = .04$, were found to reach statistical significance.

Agreeableness and openness.

No significant main or interaction effects were found in relation to agreeableness or openness and shift in SBP, DBP or HR over the course of the days of the study.

Blood Pressure Variability

The results of stepwise regression analyses for each day of measurement found that only variability of SBP on Sunday was significantly predicted by personality traits (see Figure 2a), such that neuroticism ($t = -2.43$, $\beta = -.35$) was the only significant predictor entered into the regression equation, $F(1, 42) = 5.90$, $p = .02$, adjusted $R^2 = .10$. The results of similar stepwise regressions showed that variability in DBP was significantly predicted by neuroticism and extraversion for two of the four days of the study (Thursday and Sunday). For Thursday (see Table 6), extraversion ($t = -2.41$, $\beta = -.35$) significantly

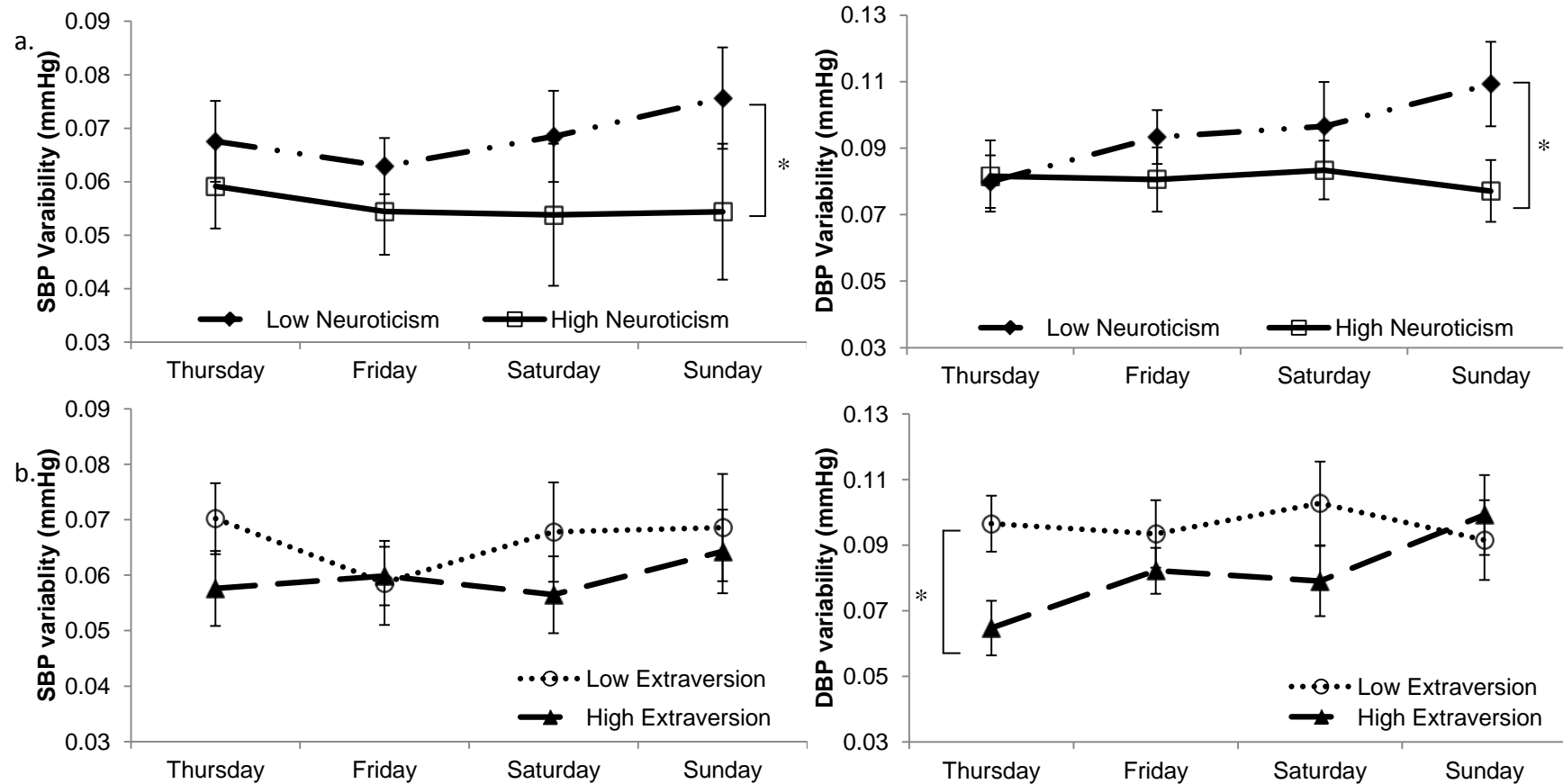


Figure 2. Personality predictors of variability in SBP and DBP. Asterisks denote significance of stepwise regression examining predictors of daily shift in cardiovascular functioning from daytime to evening. Median splits are for illustrative purposes (higher neuroticism, $n = 25$, lower neuroticism, $n = 19$; higher extraversion, $n = 22$, lower extraversion). Error bars denote standard error of the mean.

* $p < .05$.

Table 6

Predictors of Variability in Blood Pressure on Thursdays

Variable	SBP variability (mmHg)		DBP variability (mmHg)	
	<i>B</i>	95% CI	<i>B</i>	95% CI
Constant	-.01	[-.08, .06]		[.10, .28]
BMI	.003*	[<.001, .007]	-	-
Extraversion			-.30*	[-.06, -.01]
R^2	.10		.12	
F	4.40*		5.81*	

Note. $N = 44$; CI = confidence interval.

* $p < .05$.

predicted variability in DBP, $F(1, 42) = 5.81$, $p = .02$, adjusted $R^2 = .10$; and for Sunday, neuroticism ($t = -2.16$, $\beta = -.32$) remained the only significant predictor of DBP variability, $F(1, 42) = 4.65$, $p = .04$, adjusted $R^2 = .08$ (see Table 7). Mean values for blood pressure variability by day are contained in Table 5.

The findings for neuroticism showed that on Sundays, higher neuroticism was associated with attenuated SBP and DBP variability. Greater extraversion was associated with diminished DBP variability on Thursdays, (see Figure 2b). None of the other predictor variables (conscientiousness, perceived stress, or satisfaction with social support) were entered into the stepwise regression equations, and so did not contribute significantly to the models (see Tables 8 and 9 for correlations of predictor and criterion variables for SBP and DBP variability respectively). Only on Thursday was one of the known risk factors for adverse cardiovascular health (i.e., BMI) shown to significantly affect SBP

variability, $F(1, 42) = 4.40$, $p = .04$, adjusted $R^2 = .07$. The results showed that greater BMI predicted greater SBP variability on Thursday ($t = 2.10$, $\beta = .31$), but BMI did not explain additional variance in cardiovascular variability to that already accounted for by neuroticism and extraversion, on Sundays and Thursdays respectively.

Table 7

Predictors of Variability in Blood Pressure on Sundays

Variable	SBP variability (mmHg)		DBP variability (mmHg)	
	<i>B</i>	95% CI	<i>B</i>	95% CI
Constant		[.08, .19]		[.10, .27]
Neuroticism	-.02*	[-.04, -.004]	-.03*	[-.05, -.002]
R^2	.12		.10	
F	5.90*		4.65*	

Note. $N = 44$; CI = confidence interval.

* $p < .05$

Table 8

Pearson's Correlations for Mean Systolic Blood pressure (SBP), Variability, and Psychometrics in Stepwise Regressions

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Age	-														
2. BMI	-.04	-													
3. Neuroticism	.09	-.16	-												
4. Extraversion	-.02	.03	-.44**	-											
5. Consc.	.15	-.06	-.18	-.03	-										
6. Soc. Support	.35*	-.20	-.13	.06	.07	-									
7. Stress	-.06	.03	.74**	-.43**	-.04	-.21	-								
8. SBP Thurs.	-.45**	.37*	.10	.14	.15	-.17	.17	-							
9. SBP Fri.	-.24	.29	.10	.04	.29	-.13	.19	.78**	-						
10. SBP Sat.	-.32*	.27	.11	.14	.17	-.18	.12	.83**	.84**	-					
11. SBP Sun.	-.17	.26	.10	.01	.21	-.15	.14	.72**	.81**	.83**	-				
12. Var. Thurs.	-.12	.31*	-.01	-.10	-.13	-.12	.01	.01	.13	.10	.06	-			
13. Var. Fri.	-.22	.13	-.19	.14	-.10	-.17	-.20	.15	.11	.08	.10	.41**	-		
14. Var. Sat.	.02	.06	-.17	-.11	.05	-.17	-.10	-.02	.12	.01	.10	.21	.32*	-	
15. Var. Sun.	-.17	-.04	-.35*	.11	.04	.10	-.33*	-.12	-.21	-.18	-.20	.20	.36*	.39**	-

Note. Consc. = conscientiousness; Soc. Support = Soc. Support = satisfaction with social support from Sarason et al. (1987) 6-item social support questionnaire; Stress = perceived stress (S. Cohen et al., 1983); Var. = variability; Thurs. = Thursday; Fri. = Friday; Sat. = Saturday; Sun. = Sunday.

* $p < .05$. ** $p < .01$.

Table 9

Pearson's Correlations for Mean Diastolic Blood Pressure (DBP), Variability, and Psychometrics in Stepwise Regressions

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Age	-														
2. BMI	-.04	-													
3. Neuroticism	.09	-.16	-												
4. Extraversion	-.02	.03	-.44**	-											
5. Consc.	.15	-.06	-.18	-.03	-										
6. Soc. Support	.35*	-.20	-.13	.06	.07	-									
7. Stress	-.06	.03	.74**	-.43**	-.04	-.21	-								
8. DBP Thurs.	-.33*	.19	.36*	.02	-.06	-.14	.34*	-							
9. DBP Fri.	-.17	-.04	.41**	.02	.16	-.05	.36*	.71**	-						
10. DBP Sat.	-.21	.11	.23	.13	-.13	-.26	.19	.69**	.57**	-					
11. DBP Sun.	-.19	.05	.31*	.07	-.27	-.24	.18	.64**	.61**	.66**	-				
12. Var. Thurs.	-.04	.16	.07	-.35*	.25	-.06	.11	-.05	-.02	-.08	-.21	-			
13. Var. Fri.	-.28	.09	-.15	-.02	-.15	-.23	-.13	.00	-.18	.13	.08	.08	-		
14. Var. Sat.	-.03	.08	-.04	-.10	.02	-.19	.00	.12	-.06	-.08	.16	.13	.21	-	
15. Var. Sun.	-.14	.06	-.32*	.16	-.15	-.10	-.30	-.06	-.32*	-.06	-.08	.04	.33*	.33*	-

Note. Consc. = conscientiousness; Soc. Support = Soc. Support = satisfaction with social support from Sarason et al. (1987) 6-item social support questionnaire; Stress = perceived stress (S. Cohen et al., 1983); Var. = variability; Thurs. = Thursday; Fri. = Friday; Sat. = Saturday; Sun. = Sunday.

* $p < .05$. ** $p < .01$.

Discussion

Neuroticism was found to be associated with attenuated shift in SBP from daytime to evening periods, and with diminished variability in SBP and DBP. Extraversion was also associated with diminished shift in SBP, increased DBP shift, and lower DBP variability. Conscientiousness was associated with increased DBP shift, but not with SBP or blood pressure variability. The results indicated that affective traits, and in particular neuroticism, were most widely associated with lower patterns of cardiovascular functioning in normative settings, with significant effects observed only on certain days.

The well-documented health-damaging effects of higher neuroticism (see Lahey, 2009), and the posited protective effects attributed to higher extraversion (R. S. Wilson et al., 2005), imply that patterns of cardiovascular functioning of people lower in neuroticism or higher in extraversion are likely to be most adaptive. In consideration of this, the trends for lower blood pressure patterns of those higher in neuroticism could be viewed as suboptimal, such that lower neuroticism was associated with lesser shift in blood pressure, and lower blood pressure variability, relative to those higher in neuroticism. The finding that diminished patterns of blood pressure variability were associated with an individual disposition towards greater neuroticism is consistent with recent findings linking blunted CVR to neuroticism (e.g., Phillips et al., 2005; Phillips et al., in press), and the posited negative implications of diminished physiological functioning for health (e.g., Carroll et al., 2009).

For SBP, the relationship between extraversion and shift in SBP was more varied, with similar levels of shift for those higher and lower in extraversion for Thursday and

Friday. However, for Saturdays, those lower in extraversion experienced much increased SBP during evening times relative to daytimes. On Sundays, those higher in extraversion were typified by increased SBP during evening times compared to daytimes and those lower in extraversion. Participants higher and lower in extraversion had similar levels of DBP shift for the first three days of the protocol (Thursday to Saturday), but for Sunday it was observed that those higher in extraversion had much increased DBP during evening times, relative to daytimes. Typically, traits relating neuroticism have been linked to adverse health outcomes in the literature, while higher extraversion has been suggested to be protective against poor health-outcomes (R. S. Wilson et al., 2005). The current results pertaining to extraversion were a reminder that as distinct traits, neuroticism and extraversion reflect different dimensions of personality, not one polar concept. Thus, trends observed for lower extraversion should not necessarily be regarded as the same as trends observed for people with higher neuroticism. For conscientiousness, less clearly discernible trends were evident than for neuroticism or extraversion. For participants lower in conscientiousness, shift from daytime to evening remained quite low and stable, with a general association over the four days for participants lower in conscientiousness to have a lower pattern of DBP. However, conscientiousness was not found to significantly predict SBP or DBP variability.

The findings pertaining to shift in blood pressure showed that individual differences in personality (neuroticism, extraversion, and conscientiousness) were associated with shifts in blood pressure from daytime to evening periods over the four days of the study. All groups of participants had generally increased blood pressure during evening times relative to daytimes, or had blood pressure that remained at a similar level to daytime with some exceptions. With regards to variability, results complimented trends

observed for blood pressure shift. Neuroticism predicted lower SBP and DBP variability trends on Sundays, while extraversion predicted lower DBP variability on Thursdays. The literature on blood pressure variability has suggested that decreased variability is associated with more positive cardiovascular health, but interestingly in this study diminished cardiovascular functioning was associated with neuroticism, a well-documented cardiovascular risk variable.

A possible explanation of the findings could be that a disposition towards more defined extremes of personality (i.e., higher neuroticism *or* extraversion), may be associated with diminished circadian cardiovascular function, but only on certain days. Correspondingly, mid-range scores for traits may reflect a more adaptive range of both personality and physiological function. This is supported in this study by a trend for those scoring in the higher ends of the ranges for extraversion, conscientiousness, and neuroticism to have lower patterns of cardiovascular response. This reflects the theory of homeostasis that too much or too little of any construct, whether physiological or psychological, is likely to be maladaptive.

Trait theories of personality have provided effective descriptive categories for how it is that people see their world, providing a framework for all of their cognitions. It has been argued from a cognitive perspective that personality traits such as those that are derived from Five-Factor theory are the characteristics that persons *have* as opposed to the behaviours that they *do* (Cantor, 1990). Social-cognitive theories argue for an integrative view of the person and the situation, such that personality dispositions invoke patterns of behavioural responses in a variety of situations (e.g., Mischel, 2004). In the context of cardiovascular functioning, long-proliferated styles of cardiovascular responding to stress, or general patterns of cardiovascular functioning (i.e., variability) may result in increased

risk of CVD and illness due to the inherent tendency of an individual to react to situations in a given manner (e.g., those higher in neuroticism or NA). Jonassaint et al. (2009) found that neuroticism had differential effects on CVR depending on the nature of the stress task (cognitive or emotional), whereby neuroticism was negatively associated with CVR during mental arithmetic, but had a smaller, positive effect on reactivity during an anger recall task. Similarly, those lower in extraversion (introverts) showed greater DBP blood pressure reactivity, but only during anger recall. Further investigations of the relationship between aspects of NA, such as neuroticism on specific task structures would have beneficial scientific merit, particularly if the social nature of stress was considered.

Some limitations curtail the scope of interpretation for the findings. Firstly, the study used quite an arbitrary day-evening distinction for shift. Blood pressure was generally higher during evenings than during daytimes, a somewhat unexpected trend given that typical diurnal fluctuations would predict reduced blood pressure during evenings relative to days. A possible explanation for these results could be found with the results of a recent study by Shea et al. (2011). The authors showed that endogenous circadian rhythms may result in peak blood pressure being observed at a time corresponding to 21:00 hours. The current study used distinctions between daytime and evening consistent with research that previously used a similar protocol (Evans & Steptoe, 2001). The evening period included only measurements taken post-18:00 until bedtime, encompassing the period preceding and including the peak time for blood pressure as suggested by Shea and et al. (2011). This may account for some of the positive shift in blood pressure from daytimes to evenings, whereby blood pressure was higher during evening periods relative to daytime.

Secondly, the current study was limited by a lack of information on the nature of the activities being engaged with or otherwise by participants at the point of measurement, limiting the scope for interpretation of the findings. However, it is common for studies involving 24-hour ambulatory monitoring to not record activities of participants at the time of cardiovascular measurement, instead preferring participants to engage in their normal activities (e.g., Kotsis et al., 2011). Future studies could monitor variability in cardiovascular responses to specific stresses or stimuli, specifically those that are social and asocial in nature. The sample was comprised of undergraduate students who could potentially be engaged in greater or lesser social activity during evening periods or on particular days, so accounting for social context during measurements should be a feature of similar future research.

Thirdly, most of the significant findings, particularly those pertaining to neuroticism, appear to be concentrated on the cardiovascular functioning for one particular day; Sunday. As Sunday was the final day of the protocol observed by participants, it is possible that the significant trends could be an artefact of the procedure, or reflect the nature of activities engaged in on these days by participants. Participants could feasibly have been excited, or relieved in anticipation of the conclusion of the protocol, which could have resulted in more atypical patterns of blood pressure for Sunday compared to the other days of the protocol. Also, given that Sunday is the final day before the commencement of a traditional “working week”, participants could also have been engaged in effortful preparations (mental or physical) for the week ahead. All participants in the current study were undergraduate students who typically were scheduled for classes that were all within the realm of a working week – nominally Monday to Friday.

The results of Study 1 demonstrate a moderating effect for Five-Factor personality traits on fluctuations in blood pressure in real life contexts, as measured by a self-monitoring protocol. The findings suggest that there is an important role for affective components of personality in understanding blood pressure fluctuations over an extended period of time. In summary, individual differences in personality were associated with lower cardiovascular responsivity to real life stimuli during daytime waking hours, particularly for those higher in neuroticism and extraversion.

Questions Arising From Study 1

The results prompt further investigation of affective personality dispositions in relation to specific types of stress that may be experienced by individuals. Experimental manipulation of the social context of stress could prove interesting, given the demonstrated associations in Study 1 between affective personality traits and lower cardiovascular functioning. Additionally, the examination of some other behaviourally-relevant affective personality dimensions could provide further interesting insights into the relationship between lower cardiovascular functioning and personality, particularly if the social context of stress was to be manipulated experimentally.

Chapter 3: Study 2

Trait Dominance as a Moderator of Cardiovascular Functioning During Social Stress

While several associations between personality traits and cardiovascular functioning have been demonstrated in past research, much of the strongest evidence for the impact of individual differences on health outcomes relates to personality constructs that have conspicuous social (or interpersonal) dimensions. Examples include such variables as anger (Smith et al., 2004), which involves the direction of hostile thoughts towards other people; neuroticism (Côté & Moskowitz, 1998), which involves the degree to which negative affect is visible to others in the form of emotional instability; and Type D personality (Denollet, 2005), which involves the extent to which a person feels socially inhibited when experiencing negative emotions. Another important socially relevant personality variable is trait dominance, which has also been shown to associated with vulnerability to CVD risk (e.g., during social interactions; Houston, Chesney, Black, Cates, & Hecker, 1992).

As discussed in Chapter 1, psychometrically assessed trait dominance is a personality construct that assesses the degree to which persons innately desire to exert a dominant position in their social group (Pratto et al., 1994). The particular relevance of social stress to dominant personality styles could have particularly harmful implications for ill-health. Research from animal studies points to the particular importance of the

relationship between dominance and social context in CVD outcomes (Kaplan et al., 1982). Dominant male monkeys were found to have significantly more advanced CAA during conditions of instability in their social hierarchies (Kaplan et al., 1982). In contrast, the same study observed that subordinate female monkeys (those that were unable to achieve dominance) had more extensive coronary artery damage than their dominant counterparts, irrespective of the conditions of the immediate social hierarchy.

Summarising much of the research conducted on human cardiovascular function and trait dominance, Newton (2009) found that evidence from human subjects has largely corroborated results from animal studies.

In humans too, trait dominance was found to be positively and significantly associated with adverse cardiovascular health outcomes, such as increased risk of post-MI morbidity and mortality (Helgeson, 1990), and with increased odds of combined fatal and non-fatal cardiovascular events (Siegman, Kubzansky, et al., 2000). In some instances, submissive traits were found to be protective against ill-health. This was especially true for women, for whom higher submissiveness was associated with decreased odds of first onset cardiovascular events (Whiteman et al., 1997). For men too, some health benefits were apparent for greater trait submissiveness, where it was related to slower progression of peripheral arterial disease (Whiteman et al., 2000).

Previous laboratory-based experimental work involving trait dominance has primarily used behavioural rather than psychometric conceptualisations of dominance, and dyadic behavioural components of stress. For example, Smith, Limon, Gallo, and Ngu (1996), used tasks that were designed to elicit dominant or submissive behaviour from their participants. As suggested by Hughes and Callinan (2007), previous research in the area of trait dominance and experimental reactivity has suffered from this randomisation of

participants to dominant groups, rather than using a psychometric measure to ascertain a personality disposition to dominance, and accordingly categorise participants along an dominance dimension. Thus, some of the previous experimental findings pertaining to dominance could be confounded by participant individual differences in dominance.

Dominance is a core component of TABP (M. Friedman, 1996), which has traditionally been discussed as if it were primarily a masculine phenomenon (Riska, 2000). As Riska (2000) described, the construct of TABP (originating in the 1950s), serves to “medicalise” masculinity. However, elements of TABP such as dominance are not the universal preserve of men; conspicuous gender differences in the way cardiovascular functioning and dominance are intertwined have been observed, some of which suggest that the variable may even be more clinically relevant to women. Hughes and Callinan (2007) found that a positive association between trait dominance and CVR during social stress was only observed for women, while an inverse relationship was observed for men, with no significant differences or interactions with trait dominance observed for men or women in relation to a mental arithmetic stressor (Hughes & Callinan, 2007). In contrast, Gramer (2003), in a sample of men, found that compared to lower trait dominant participants, men higher in dominance had increased SBP and pulse pressure (PP), and lower DBP. Indeed, the effects were found to be most pronounced during mental arithmetic stress (Gramer, 2003). However, the mental arithmetic task in Gramer’s (2003) study was performed in front of an observer, and thus had a socially relevant component which may have influenced the results. Gramer and Berner (2005) later found that trait dominance influenced CVR to an interpersonal stressor, whereby during attempts to dominate a dyadic discussion, high-dominant participants had enhanced SBP and PP reactivity to the stress task, accompanied by lower DBP. In this study, effects of

dominance were found to be largely similar across gender groups (Gramer & Berner, 2005), with only differences in DBP accounted for by more pronounced elevations in dominant women.

A recent review (Newton, 2009) concluded that in the main, studies involving socially relevant stressors show significant positive associations between trait dominance and acute cardiovascular responses, with more uniform patterns of responses evident for women than for men. Analysis of the relationship between dominance and asocial stressors found no significant effects on cardiovascular functioning for women, but some indication of a positive trend for systolic SBP reactivity for men (Gramer, 2003; Gramer & Huber, 1997). As such, no reliable indicators of trends between trait dominance and asocial forms of stress have been shown. This is not wholly surprising, as trait dominance is a socially oriented personality construct with roots in an evolutionary need to assert one's social position.

A number of empirical studies have shown that there are some maladaptive patterns of cardiovascular functioning during stress that can have long-term health-damaging effects, including both exaggerated and diminished responses to stress (Lovallo, 2011). Acute stress testing in laboratory settings allows for the standardised assessment of psychophysiological responses to challenge, and for associations with individual differences to be attributed under controlled conditions (Chida & Hamer, 2008). Although psychophysiological stress responses may not have clinical importance in themselves, it is argued that they provide information about the ways that individuals respond to stress, such that patterns of maladaptive stress responding that proliferate over years may accumulate and result in disease processes (Chida & Hamer, 2008). Chapter 1 introduced the idea that CVR to various psychological stressors is associated with future

cardiovascular ill-health, and that these trends can originate in normal blood pressure of young health adults (e.g., K. A. Matthews et al., 2004; Treiber et al., 2003). Acute physiological stress manipulations in the laboratory have used a variety of psychological and physiological tasks to elicit stress responses. Distinctions may be made between stressors that are passive or active in nature, and further active stressors which have a social component, or are asocial.

Tasks such as the cold pressor task may elicit both physiological and psychological stress, characterised by passive coping attempts. In contrast to the cold pressor task, adaptive responses to active stress tasks (e.g., cognitive tasks including mental arithmetic, mirror tracing, computer games, or speech tasks) require a behavioural response, such as problem-solving. Individual differences in stress vulnerability may in part be due to an interaction of the nature of stress that is experienced, and innate personality differences which dispose someone to react to such stress in a particular manner (e.g., through exaggerated or diminished responding). Active and passive stressors have been associated with the activation of different physiological mechanisms. Passive stress, and associated passive coping, has been shown to be associated with alpha-adrenergic patterns of physiological (cardiovascular) functioning, while active stress responses (active coping) are associated with beta-adrenergic stress responses (e.g., Sherwood, Allen, Obrist, & Langer, 1986; Sherwood, Royal, & Light, 1993).

Prominent amongst acute stress laboratory paradigms is the use of mental arithmetic tasks to elicit cognitive stress, and associated CVR. Usefully, they may be delivered devoid of any emotional valence or social implications. Mental arithmetic stressors have been widely used and demonstrated to produce reliable stress responses and changes in patterns of cardiovascular functioning in many studies (e.g., Howard et al.,

2011; Hughes, 2007a; Jonassaint et al., 2009). Variations of mental arithmetic stressors include the paced auditory serial addition test (PASAT; Gronwall, 1977), which involves working memory and attention in addition to simple addition, and has been used widely in published work (e.g., Ginty et al., 2012; Willemsen et al., 1998). Other forms of mental arithmetic stressors involve requesting participants to count backwards from a large number in large multiples, which has been found to be significantly stressful (e.g., Aubert, Verheyden, d'Ydewalle, Beckers, & Van den Bergh, 2010).

While mental arithmetic stressors are challenging, it is argued that for some individuals other types of stressors may produce more striking stress responses. Social stressors have been shown to elicit greater CVR than non-social stressors (e.g., al'Absi et al., 1997; Gramer, 2003). Socially-salient stressors have been shown to elicit considerable and reliable stress responses, and may have particular salience for individuals who have stronger tendencies towards NA or neuroticism (e.g., Schneider, 2004), and individual differences in other personality dimensions with strong interpersonal components, such the trait dominance. Socially relevant stressors are generally acknowledged to elicit more powerful physiological stress responses than tasks without a socially relevant component (Dickerson & Kemeny, 2004). It has been suggested that a possible explanation for the relative effectiveness of socially relevant stressors in inducing physiological stress is that it manipulates what has been termed the human social self-preservation system (Dickerson & Kemeny, 2004). Physiological responsiveness to (social) stressors are evaluated in terms of their threat to an individual's self esteem, social status, and psychological, physiological and behavioural responses to a threat or stressor are coordinated in response to such threats (Dickerson & Kemeny, 2004). It has been argued that the magnitude of an individual stress

response is regulated by vulnerability and protective factors in the environment (Dickerson & Kemeny, 2004).

The value of social tasks in inducing stress has been well-documented. The Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993) protocol has provided a framework for the investigation of acute stress responses for 20 years. Widely used in the literature, the TSST consists of a combination of socially oriented tasks designed to induce human stress, including socially evaluative stress in the form of speech preparation and delivery in the presence of a experimental confederates, and a mental arithmetic task where participants are required to count backwards from a large number, with prompts from a confederate if incorrect answers are given. Variants on the TSST have been used to elicit physiological stress separately with success, whereby elements of the procedure may be presented in isolation, such as either the speech preparation task or the mental arithmetic task are presented. As used in the TSST, mental arithmetic stressors may encompass a degree of social stress when participants are asked to count backwards from a number in front of an experimenter. However, it is possible to isolate the cognitive elements of the mental arithmetic stressor by removing the social element and having participants complete the mental arithmetic stressor on a computer. This allows for the active, cognitive stress elements of the task to be investigated, devoid of the social stress element.

From a review of the research literature, Kamarck and Lovallo (2003) concluded that while there is insufficient evidence to suggest that social tasks have more ecological relevance than asocial tasks in predicting responsiveness to real life stress, further research is needed to clarify the role of social stress. The relevance of socially-salient stress could be increased depending on individual differences in aspects of personality that have

specific interpersonal or behavioural resonance. Kamarck and Lovallo (2003) argue for a focus on tasks which involve psychological effort or behavioural preparation as important research concerns for CVR research.

Study 1 showed that cardiovascular functioning varied across daytime and evening periods, and was moderated by affective dimensions of personality. The findings posed questions as to the impact of contextual factors on such observed differences in physiological differences, with particular interest in social and interpersonal factors, due to the inherent interpersonal relevance of affective personality characteristics. Meanwhile, results from laboratory studies (e.g., Gramer, 2003; Gramer & Berner, 2005; Hughes & Callinan, 2007) point to the particular value of assessing formal stress tasks that have social components, and suggest that women as well as men display significant cardiovascular stress responses moderated by trait dominance. Thus, it seems that further investigation of the social context under which stress is experienced may be particularly important for understanding the interaction between trait dominance and physiological functioning, given that trait dominance has overt social preservation characteristics, proposed by Dickerson and Kemeny (2004) to be especially relevant in the induction of physiological responses during social stress. Individual differences in trait dominance Accordingly, the present study aims to investigate if dominance, an individual differences variable with overt social elements, can be seen to moderate patterns of responding to active stress that is social or asocial in nature.

Method

Participants

Data were collected for 94 undergraduate participants (67 women and 27 men). A filter was applied to the data in an attempt to control for some demographic characteristics that have been known to effect blood pressure, such as age, BMI, and pre-existing hypertension. The final study sample was confined to participants aged less than 35 years of age, without a personal history of hypertension, with normal levels of blood pressure at baseline ($< 140/90$ mmHg), and with BMI within normal ranges (< 30). As a result of this, six women and three men were excluded from the final sample. Due to known gender differences in blood pressure, it was determined that cardiovascular data for women and men should be analysed separately. However, the small sample of men was underpowered for statistical analyses, so it was decided that only women would be included in the final sample for inferential analyses. All presented inferential statistics presented below for this study refer to data collected for women, unless otherwise stated. Some descriptive statistics pertaining to cardiovascular and psychometric data collected for men are reported where appropriate.

The final sample of women ($n = 61$) had a mean age of 20.98 ($SD = 3.93$), with normal BMI ($M = 21.82$, $SD = 2.45$). The sample included eight smokers, who reported smoking between one and thirteen and a half cigarettes daily ($M = 1.01$, $SD = 3.05$). Smokers were included in the final sample, as they were found not to affect the trend of the results. A manipulation check (independent t -test) showed no significant differences between participants assigned to social or asocial stress conditions with regard to age or BMI.

Measures

Cardiovascular responses.

Cardiovascular parameters of SBP, DBP, and HR were assessed using a Dinamap Pro 100 Vital Signs Monitor (GE Medical Systems, Tampa, Florida). This sphygmomanometer uses the oscillometric method to return blood pressure readings from a cuff attached to the participant's upper non-dominant arm over a period of approximately 30 seconds. The Dinamap apparatus has been found to provide accurate readings of cardiovascular functioning in healthy adults (Ni et al., 2006), and has been used widely in previously published scientific studies (e.g., Hughes & Callinan, 2007; O'Donovan & Hughes, 2008).

Psychometric testing.

Trait dominance was measured using the Jackson Personality Research Form (JPRF) social dominance subscale (Jackson, 1999). Sixteen items assessed trait dominance, with scores ranging from 0-16. Sample items include “I feel confident when directing the activities of others”, and “I would make a poor military leader” (reverse scored). High scorers on the trait dominance subscale of the JPRF attempt to control their environments and influence or direct other people; they are forceful, decisive, authoritative, and domineering (Jackson, 1965). Reliability was good in the current study ($\alpha = .80$; see Table 10 for descriptive and reliability statistics). Mean trait dominance scores were calculated by determining the mean response to the 16 items, and this mean was used primarily as a covariate in subsequent analyses.

Subjective task ratings.

Participants provided subjective post-stress ratings for three separate items measuring perceived stress, interest, and difficulty of the task (see Table 11 for descriptive statistics). Participants were asked to rate “how stressful/ interesting/ difficult did you find the task” following their completion of the stress task. Answers were obtained using a five-point Likert response scale. Possible scores ranged from 0 (not at all; stressful, interesting or difficult) to 4 (extremely; stressful, interesting or difficult). Mean scores for each item were derived.

Stress Tasks

Participants were randomised to one of two stress conditions. During the social stress task, participants were presented with a set of cards, each of which had a different word printed on it. Participants were instructed to speak for as long as they could about the word on the top card, saying whatever came to mind. If they could not think of anything more to say, they could move on to the next card, repeating the procedure. The words were

Table 10

Descriptive Statistics for Psychometric Variables (By Item) Including Estimates of Scale Reliability for Women (n = 61) and Men (n = 24)

Variable	Women					Men				
	α	Mean	<i>SD</i>	Min.	Max.	α	Mean	<i>SD</i>	Min.	Max.
Dominance ^a	.80	0.51	0.24	0.00	1.00	0.67	0.26	0.14	1.00	0.67
Cynical Distrust ^b	.74	0.95	0.45	0.00	2.00	1.17	0.36	0.25	1.88	1.17
Cynicism ^c	.83	1.10	0.45	0.15	2.00	1.36	0.40	0.29	2.15	1.36
Trait Anger ^d	.74	0.73	0.36	0.10	1.50	1.07	0.58	0.20	2.80	1.07
Aggression ^e	.83	2.14	0.43	1.28	3.10	2.68	0.57	1.54	3.62	2.68
Sig. other support ^f	.96	5.87	1.50	1.50	7.00	5.77	1.23	2.25	7.00	5.77
Family support ^f	.95	5.75	1.58	1.00	7.00	5.59	1.10	2.75	7.00	5.59
Friends support ^f	.95	5.81	1.42	1.00	7.00	5.92	0.72	3.75	7.00	5.92
Total support ^f	.96	5.81	1.38	1.36	7.00	5.76	0.79	3.33	6.92	5.76
Support number ^g	.90	4.72	1.94	1.17	9.00	3.65	2.11	1.00	7.67	3.65
Support satisfaction ^h	.94	5.09	0.98	1.17	6.00	5.08	0.71	3.17	6.00	5.08

Table 10 continued

Active ⁱ	.75	5.31	1.64	2.00	8.00	5.42	1.84	2.00	8.00	5.42
Planning ⁱ	.84	5.51	1.77	2.00	8.00	5.67	1.66	2.00	8.00	5.67
Emotional ⁱ	.87	5.38	1.89	2.00	8.00	4.58	1.74	2.00	8.00	4.58
Instrumental ⁱ	.83	5.25	1.73	2.00	8.00	5.04	1.63	2.00	8.00	5.04

Note. α = Chronbach's alpha coefficient; SD = standard deviation; Min. = minimum; Max. = maximum.

^aMeasured using the trait dominance subscale of the Jackson Personality Form (Jackson, 1999).

^bMeasured using cynical distrust (Greenglass & Julkunen, 1989) subscale of Cook-Medley hostility inventory (Cook & Medley, 1954).

^cMeasured using the cynicism (Barefoot et al., 1989) subscale of Cook-Medley hostility inventory (Cook & Medley, 1954).

^dMeasured using the trait anger subscale of the State-Trait Anger Expression Inventory 2 (Spielberger, 1999).

^eMeasured using the Buss Perry Aggression Questionnaire (Buss & Perry, 1992).

^fMeasured using appropriate subscales of the multidimensional scale of perceived social support, (Zimet, Dahlem, Zimet, & Farley, 1988).

^gMeasured using the *N* subscale of the SSQ6 (Sarason et al., 1987).

^hMeasured using the *S* subscale of the SSQ6 (Sarason et al., 1987).

ⁱMeasured appropriate subscales of the brief ways of coping scale (Carver, 1997).

Table 11

Mean Subjective Task Ratings for Experimental Condition

Item	Women ^a				Men ^b			
	Social		Asocial		Social		Asocial	
	Mean	<i>SD</i>	Mean	<i>SD</i>	Mean	<i>SD</i>	Mean	<i>SD</i>
Stressful?	2.03	0.89	2.10	0.87	1.90	0.88	2.00	1.21
Interesting?	1.73	1.08	1.42	0.92	1.70	0.82	1.19	1.11
Difficult?	2.37	0.96	2.71	0.90	1.70	1.25	2.63	1.02

^aSocial task, $n = 32$; Asocial task, $n = 27$.

^bSocial task, $n = 10$; Asocial task, $n = 16$.

chosen from the MRC Psycholinguistic database

(http://www.psych.rl.ac.uk/MRC_Psych_Db.html; M. Wilson, 1988), and were printed on individual cards. The number of letters per word ranged from five to nine, and the number of syllables ranged from two to four. Similar to a previous study (Hughes & Callinan, 2007), the words chosen were intended to serve as prompts for wide-ranging commentary, and so consisted of relatively high-frequency generic nouns (see Appendix B). Participants were instructed to speak clearly into the microphone provided, and their attention was drawn to a television screen where they remained visible to themselves. They were informed that their speeches were being video-recorded and would later be evaluated by the research team for overall content, clarity, and delivery style.

The asocial task chosen was a mental arithmetic task, performed via a computer program. Subtraction problems appeared on-screen, and participants were required to input

the correct solution within 10 seconds. The task (see Hughes, 2001) was designed to include principles of “standardised flexibility” (Turner et al., 1986), such that difficulty was contingent on accuracy; accurate responses led to more difficult problems (i.e. problems involving larger numbers), whereas incorrect answers led to less difficult problems.

Procedure

The research took place in an on-campus laboratory. Upon arrival at the laboratory, participants were invited to be seated and relax for 10 minutes while completing some psychometric measures. The blood pressure apparatus was attached to their non-dominant upper arm after these 10 minutes had elapsed, and participants subsequently were invited to relax for a further 10 minutes while reading a nature magazine. Measurement of cardiovascular parameters commenced during this reading period, with assessments of blood pressure and HR taken at 1, 4, 6, and 8 minutes into the 10-minute period. Participants were randomised to one of two experimental conditions, whereby they completed a six-minute stress task that was either social or asocial in nature. During the six-minute stress task and six-minute resting period, blood pressure and HR were assessed at 30 seconds, 2 minutes 30 seconds, and 4 minutes 30 seconds intervals. Post-stress, participants rested for a further six minutes. During the post-task resting phase, participants were asked to revert to reading the nature magazine they had previously been provided with during baseline measurement. Participants rated their subjective appraisals of the experimental task as stressful, interesting, and difficult at the end of the procedure.

Overview of Analyses

Epochs of cardiovascular measurement were averaged to produce mean SBP, DBP, or HR responses for each of the three experimental phases (baseline, task, and post-task resting; see Table 12 for descriptive statistics). Chronbach's alpha reliability coefficients indicated excellent reliability across experimental phases for all cardiovascular parameters ($\alpha > .91$).

Several 3 by 2 mixed ANCOVAs were conducted to evaluate mean cardiovascular functioning during the two stress conditions. The within subjects factor was the relevant cardiovascular parameter (mean SBP, DBP, or HR) with three levels; baseline, stress task, and resting phases. The between subjects factor was stress condition with two levels; social and asocial stress conditions. Trait dominance was entered as a continuous covariate in all 3 \times 2 mixed ANCOVAs. Where necessary, adjustments to degrees of freedom for violations of assumptions of sphericity were made using Greenhouse-Geisser epsilon corrections. In these instances, only the correctly adjusted degrees of freedom are reported. In the 3 \times 2 mixed ANCOVAs, significant main effects for phase were taken to indicate that cardiovascular functioning varied across experimental phases. Given the likelihood of polynomial trends for the within-subject variable (phase), quadratic effects were investigated in addition to linear effects. Paired samples *t* tests were conducted when a main effect for phase was significant to determine where the differences between experimental phases lay. Significant main effects for condition were understood to suggest that mean cardiovascular functioning differed between the stress task conditions. Mean cardiovascular functioning values during social or asocial stress conditions were scrutinised in this case to confirm the nature of trends. Significant phase \times condition interactions suggested that cardiovascular

Table 12

Descriptive Statistics for Cardiovascular Functioning over Successive Experimental Phases for Women and Men

Variable	Baseline				Task				Resting			
	Social ^a		Asocial ^b		Social ^a		Asocial ^b		Social ^a		Asocial ^b	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Women												
SBP ^a	107.88	9.53	106.88	9.50	122.42	16.42	112.85	11.92	108.67	10.20	105.61	10.38
DBP ^a	64.36	6.79	63.94	8.26	74.03	9.27	68.71	8.47	65.70	7.26	64.10	8.11
HR ^b	75.19	9.63	73.53	9.97	89.01	10.47	83.40	13.24	75.88	9.95	73.70	10.74
Men												
SBP ^a	109.29	9.92	117.52	9.83	125.37	18.74	124.18	12.04	110.22	9.96	119.00	9.40
DBP ^a	62.18	5.96	62.01	6.31	73.07	9.09	67.83	8.23	62.78	6.95	63.23	5.43
HR ^b	65.25	18.26	69.88	12.20	77.37	21.19	75.64	11.12	64.90	16.40	70.35	10.74

^aSocial task; Women $n = 32$, Men; $n = 13$.

^bAsocial task; Women, $n = 27$; Men, $n = 12$.

functioning varied across time differentially for those in social and asocial stress conditions. Independent samples *t* tests investigated the trends for such observed effects, with Bonferroni corrections applied where necessary to control for multiple comparisons.

Significant three-way phase \times condition \times dominance interactions were taken to indicate that patterns of cardiovascular functioning over experimental phases were contingent on experimental condition that participants were assigned to, and also individual differences in trait dominance. To further investigate instances of this observed interaction, a new between subjects grouping variable was computed with four levels. It was constructed using median-split trait dominance scores (median item score for dominance was 0.57 for women), to create higher and lower trait dominance categories. These dominance categories were further organised by condition; social or asocial stress (i.e., low-dominant participant assigned to social stress condition, $n = 13$; high-dominant participants assigned to the social stress condition, $n = 15$; low-dominant participants assigned to the asocial stress condition, $n = 16$; and high-dominant participants assigned to the asocial stress condition, $n = 15$). One way independent ANOVA with the new four-level grouping variable as the between subjects factor was conducted on each experimental phase (baseline, task, and resting) for the cardiovascular parameters if the three-way interaction was found to be significant. The new grouping variable was also used for illustrative purposes in figures.

Effect sizes are presented as partial η^2 for ANOVA effects, and *r* for correlations. Eta-squared values of .04, .25, and .64, and *r*- values of .1, .25, and .37 were taken to represent small, medium, and large effect sizes respectively (J. Cohen, 1988, 1992).

Results

Baseline Equivalence

Independent samples *t* tests revealed no significant differences in cardiovascular functioning at baseline were identified for women assigned to social ($n = 30$) or asocial ($n = 31$) stress task conditions for SBP, $t(59) = 0.41$, $p = .68$; DBP, $t(59) = 0.22$, $p = .83$; or HR $t(59) = 0.66$, $p = .51$, or for those higher or lower in trait dominance ($p > .13$).

Independent samples *t* tests revealed no significant differences in mean levels of trait dominance between participants assigned to social or asocial stress conditions ($p = .66$).

Subjective Task Ratings of Stress, Difficulty, and Interest

The results of a series of separate 2 (task type; social or asocial) \times 1 (subjective ratings of task one) with trait dominance ANCOVAs for ratings of stress, interest or difficulty, revealed only one significant effect. During analysis of subjective ratings of task interest, a significant main effect for the covariate, trait dominance, was revealed, $F(1, 57) = 5.46$, $p = .02$, partial $\eta^2 = .09$. A Pearson's correlation (see Table 13 for descriptive statistics) showed a significant inverse correlation between trait dominance and perceptions of how interesting the task was ($r = -.29$, $p = .03$, $n = 61$). Regardless of the type of task participants completed, those lower in trait dominance rated the tasks as more interesting, $M = 1.69$, $SD = 0.89$, than participants higher in trait dominance, $M = 1.47$, $SD = 1.11$. No significant differences in subjective ratings of stressfulness ($p = .78$), interest ($p = .23$), or difficulty ($p = .16$) were found between participants assigned to social or asocial stress tasks. Despite differing physiological responses (higher for the social stress task), participants did not report differing subjective appraisals of the tasks.

Table 13

Table of Descriptive Statistics for Subjective Task Ratings

Task ratings	Low Dominance ($n = 13$)				High Dominance ($n = 17$)			
	Social		Asocial		Social		Asocial	
	Mean	<i>SD</i>	Mean	<i>SD</i>	Mean	<i>SD</i>	Mean	<i>SD</i>
Stressful?	2.23	0.60	2.00	0.89	1.88	1.05	2.20	0.86
Interesting?	1.69	1.03	1.69	0.79	1.76	1.15	1.13	0.99
Difficult?	2.62	1.04	2.63	1.02	2.18	0.88	2.80	0.77

Note. *SD* = standard deviation. Trait dominance measured using the trait dominance subscale of the Jackson Personality Research Form (Jackson, 1999).

Mean SBP

The results of a 3×2 mixed ANCOVA with trait dominance revealed a significant main effect for phase, $F(1.63, 89.85) = 16.81, p < .001$, partial $\eta^2 = .23$. Phase was also found to have a significant quadratic trend, $F(1, 55) = 22.50, p < .001$, partial $\eta^2 = .29$. *Post hoc* paired samples *t* tests showed that mean SBP was significantly higher during stress compared to resting, $t(58) = 8.16, p < .001$, or baseline periods, $t(58) = -6.98, p < .001$. No significant difference between SBP during baseline and resting phases was observed, $t(60) = 0.31, p = .76$. A significant main effect for type of task, $F(1, 55) = 5.47, p = .02$, partial $\eta^2 = .09$, showed that SBP was higher in the social stress condition than in the asocial condition, regardless of phase or dominance (see Table 12). No significant

effects for trait dominance, $F(1, 55) = .03, p = .87$, partial $\eta^2 < .001$; task \times trait dominance interaction, $F(1, 55) = 3.02, p = .09$, partial $\eta^2 = .05$; or phase \times trait dominance interaction, $F(1.63, 89.81) = 1.08, p = .33$, partial $\eta^2 = .02$, were found.

A significant phase \times task type interaction, $F(1.63, 89.85) = 12.84, p < .001$, partial $\eta^2 = .19$, which was also significant at the level of a quadratic function, $F(1, 55) = 13.69, p = .001$, partial $\eta^2 = .20$, was found. Investigation of the interaction using three *post hoc* independent samples *t* tests, with an appropriate Bonferroni correction, showed a significant difference between participants in the social and asocial stress conditions during the stress task, $t(48.87) = 2.54, p = .01$, whereby those in the social stress condition had higher mean SBP. No significant differences were observed between groups during baseline ($p = .68$) or recovery ($p = .25$) phases.

A significant phase \times task type \times trait dominance interaction, $F(1.63, 89.85) = 7.27, p = .002$, partial $\eta^2 = .12$, was observed. The three-way interaction was also significant at the level of the quadratic function, $F(1, 55) = 6.78, p = .01$, partial $\eta^2 = .11$. To investigate this interaction further, a new grouping variable with four levels was created to identify those participants in the social or asocial conditions who were higher and lower in trait dominance (split at the median; median = 0.57). This was then used as a between subjects variable in three one way independent ANOVAs with SBP measured during baseline, task, or resting as the dependent variable. Results of these ANOVAs showed no significant group differences during baseline, $F(3, 57) = 0.53, p = .67$, partial $\eta^2 = .03$, or resting phases, $F(3, 57) = 1.92, p = .14$, partial $\eta^2 = .09$, but statistically significant differences were found for SBP measured during the task phase, $F(3, 55) = 3.47, p = .02$, partial $\eta^2 = .16$ (see Figure 3a). *Post hoc* Tukey test procedures showed significant differences ($p = .02$) between low-dominant participants, such that higher SBP was observed for low-

dominant participants in the social stress condition, $M = 125.32$, $SD = 14.07$, compared to low-dominant participants in the asocial stress condition, $M = 108.97$, $SD = 11.76$. No significant differences in mean SBP were found between high-dominant participants in the social or asocial stress conditions ($p = .94$), between high and low-dominant participants in the social ($p = .74$) or asocial ($p = .39$) conditions, between low-dominant participants in the social compared to high dominants in the asocial ($p = .41$), or low dominants in the asocial compared to the social stress responses of high-dominant participants ($p = .15$).

Mean DBP

ANCOVA analysis revealed a significant main effect for phase, $F(1.68, 92.47) = 29.40$, $p < .001$, partial $\eta^2 = .35$. A significant quadratic-level effect was also observed for DBP, $F(1, 55) = 40.96$, $p < .001$, partial $\eta^2 = .43$. Similar to SBP, it was observed that DBP during the stress task was significantly different to DBP observed at baseline, $t(58) = -8.28$, $p < .001$, and at resting, $t(58) = 8.99$, $p < .001$, but that baseline and resting DBP did not significantly vary from each other, $t(60) = -1.50$, $p = .14$.

A significant phase \times task type interaction, $F(1.68, 92.47) = 11.38$, $p < .001$, partial $\eta^2 = .17$, was significant at the level of the quadratic function, $F(1, 55) = 15.89$, $p < .001$, partial $\eta^2 = .22$. Three *post hoc* independent samples *t* tests examined the differences, with condition as the grouping variable. An effect for task DBP was found not to be significant following an appropriate Bonferroni correction for multiple comparisons, $t(57) = 2.30$, $p = .03$. It was found that DBP trended towards higher mean levels during the task phase for those in the social task condition. No significant differences were observed for baseline ($p = .83$) or resting ($p = .42$) phases of DBP.

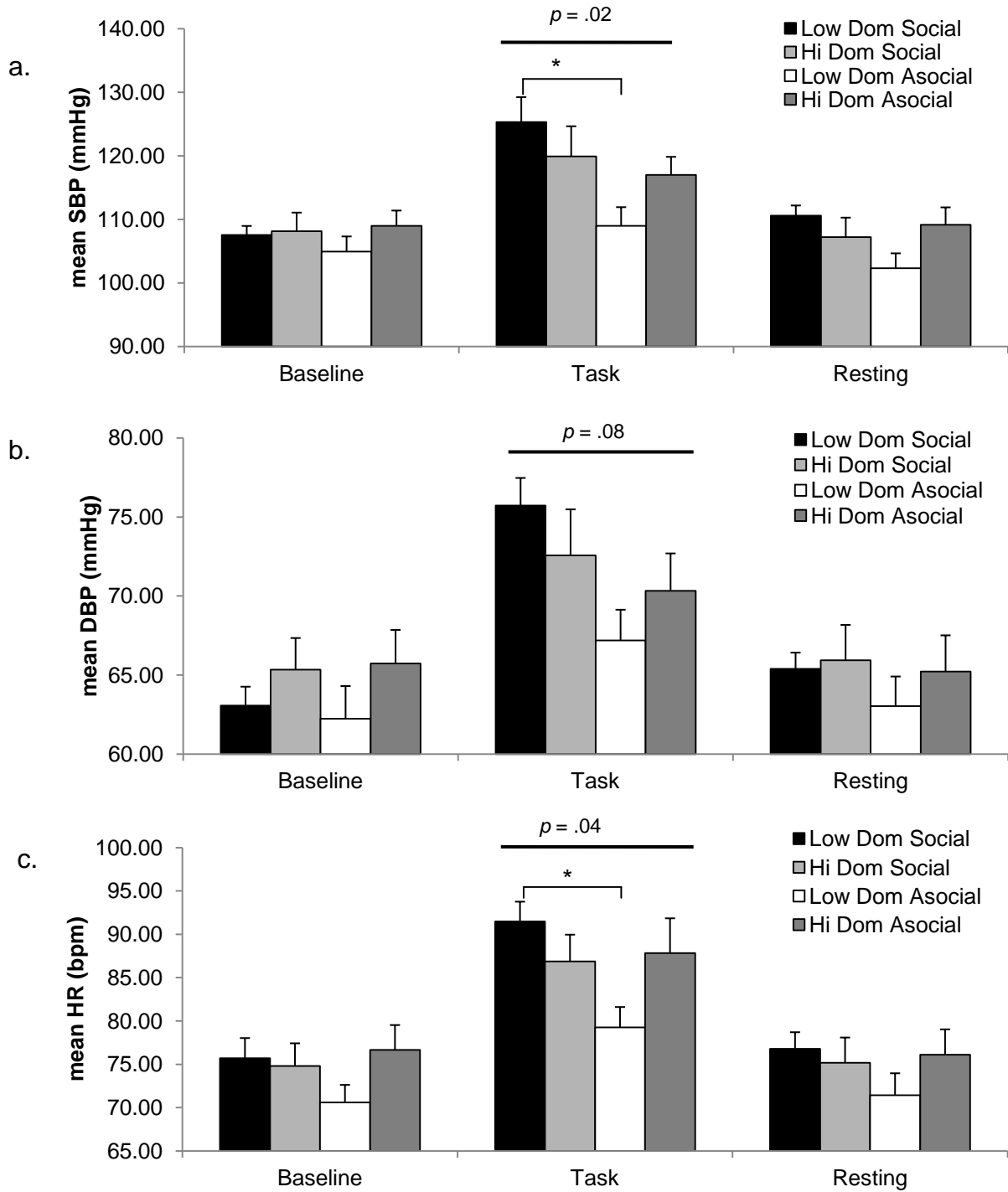


Figure 3. Mean cardiovascular functioning over three experimental phases for social and asocial tasks with median split trait dominance for illustration; Low-dominant social task ($n = 13$), high-dominant social task ($n = 17$), low-dominant asocial task ($n = 16$), high-dominant asocial task ($n = 15$). p values indicate significant of 4×1 ANOVA for dominance and stress condition groupings by cardiovascular functioning during the task phase. Asterisks denote post-hoc Tukey test significance between levels of the between subjects variable. Error bars denote standard error.

* $p < .05$.

A significant phase \times trait dominance, $F(1.68, 92.47) = 3.62, p = .04$, partial $\eta^2 = .06$, interaction was observed. *Post hoc* independent samples *t* tests did not clarify the interaction further, such that no clear significant differences were identified between those higher and lower in trait dominance during successive phases of the experiment (baseline, $p = .13$; task, $p = .86$; or resting, $p = .44$). No significant between-subject effects were observed for trait dominance, $F(1, 55) < .001, p = .99$, partial $\eta^2 < .001$; task type, $F(1, 55) = 1.85, p = .18$, partial $\eta^2 = .03$; or trait dominance \times task type interaction, $F(1, 55) = .81, p = .37$, partial $\eta^2 = .02$.

A significant phase \times task \times trait dominance interaction effect was observed, $F(1.68, 92.47) = 5.75, p = .01$, partial $\eta^2 = .10$. Similar to analysis of effects for SBP, three one way independent ANOVAs using a composite variable (describing the median-split level of dominance groups exposed to social or asocial stress) as the between subjects factor were conducted to examine whether experimental condition and stress condition were associated with DBP during the three phases of the study. The results did not clarify the interaction further (see Figure 3b). No significant differences were observed in DBP between the four groups during the task, $F(3, 55) = 2.38, p = .08$, partial $\eta^2 = .12$; baseline, $F(3, 57) = 0.79, p = .51$, partial $\eta^2 = .04$; or resting phases, $F(3, 57) = 0.43, p = .73$, partial $\eta^2 = .02$.

Mean HR

A significant main effect for phase was observed, $F(1.64, 89.96) = 35.55, p < .001$, partial $\eta^2 = .39$, which was found to be characterised by a quadratic function, $F(1, 55) = 48.07, p < .001$, partial $\eta^2 = .47$. Paired samples *t* tests confirmed that mean HR was higher during task phases than during baseline, $t(58) = -13.04, p < .001$, or resting phases, $t(58) =$

12.15, $p < .001$. No significant difference was observed between mean HR during baseline and resting phases, $t(60) = -0.76, p = .45$.

Although there was no significant phase \times trait dominance interaction, $F(1.64, 89.96) = 1.02, p = .35$, partial $\eta^2 = .02$, a significant phase \times task type interaction was found, $F(1.64, 89.96) = 6.47, p = .004$, partial $\eta^2 = .11$, which was also significant at the quadratic level, $F(1, 55) = 8.82, p = .004$, partial $\eta^2 = .14$. Three *post hoc* independent samples t tests with a Bonferroni correction examining differences in HR across phases for those in social or asocial task conditions revealed no statistically significant differences, $ps > .08$. Mean HR was elevated for those in social stress group, $M = 89.01, SD = 10.47$, relative to those in the asocial stress group, $M = 83.40, SD = 13.24$, but the trend was not significant, $t(57) = 1.79, p = .08$.

A significant main effect was apparent for task type, $F(1, 55) = 5.34, p = .03$, partial $\eta^2 = .09$, such that HR was higher for those in the social stress task group, $M = 82.07, SD = 10.41$, compared to those in the asocial stress task group, $M = 80.07, SD = 11.94$. No significant between subjects effects were observed for a task type \times trait dominance interaction, $F(1, 55) = 3.90, p = .05$, partial $\eta^2 = .07$, or for trait dominance, $F(1, 55) = .11, p = .74$, partial $\eta^2 = .002$.

A significant phase \times task type \times trait dominance interaction was observed for HR, $F(1.64, 89.96) = 3.80, p = .03$, partial $\eta^2 = .07$. One way independent ANOVAs with the composite dominance-stressor variable as the between subjects factor examined the differences between the four groups and HR during experimental phases. No significant differences between levels of the between subjects variable during baseline, $F(3, 57) = 1.18, p = .33$, partial $\eta^2 = .06$, or resting, $F(3, 57) = 0.81, p = .50$, partial $\eta^2 = .04$, were identified. It was found that only during the task phase were there significant differences

between the levels of the between subjects variable, $F(3, 55) = 2.88$, $p = .04$, partial $\eta^2 = .14$. *Post hoc* Tukey tests demonstrated that the only statistically significant differences between groups were for participants lower in trait dominance: between those assigned to the social and asocial stress conditions, $p = .03$ (see Figure 3c). During the stress tasks, mean HR was higher for participants lower in trait dominance in the social stress condition, $M = 91.49$, $SD = 8.18$, compared to those in the asocial stress condition, $M = 79.26$, $SD = 9.34$. No other group comparisons were significant.

Discussion

The present findings are consistent with the suggestion that trait dominance serves to moderate cardiovascular functioning during stress, and as such may influence long-term risk of adverse CVD outcomes. The interaction between cardiovascular functioning and dominance was further found to be dependent on the nature of the stressor being faced. However, the direction of the findings was not as expected. Specifically, women with lower trait dominance were found to have higher mean SBP and HR during social stress compared to equivalent low trait dominant women facing asocial stress. Interestingly, no significant findings were observed in relation to women higher in trait dominance, such that cardiovascular functioning during social or asocial stress was not significantly different to that observed for women lower in dominance.

Previous research found that dominance was positively associated with CVR to stress, and in particular social stress. The current results showed in contrast, greater mean cardiovascular functioning for *low*-dominant participants under conditions of social stress, relative to low-dominant participants under asocial stress conditions. Lower dominant

participants reported greater post-task interest in the stress tasks generally. As a result, the increased cardiovascular functioning during social stress for low-dominant participants could be attributed to greater (effortful) task engagement. However, significantly higher SBP and HR were observed in relation to the social task, while mean SBP and HR remained quite low for the asocial task. No significant differences between self-reported interest in social and asocial tasks were found, suggesting that interest in the stress tasks may not have wholly accounted for the increased cardiovascular functioning observed during the social task, as there was no significant difference in engagement between social and asocial tasks. Additionally, no significant differences among participant's subjective ratings of the stressfulness or difficulty of the tasks performed were observed.

While an emphasis might be given to the apparent supernormal elevation in cardiovascular responses among low-dominant participants, an alternative explanation is that the responses seen for high-dominant women were in fact, subnormal. In other words, the non-significant differences between social and asocial tasks for women higher in trait dominance could reflect diminished or inadequate stress responses during the tasks, such that blood pressure and HR during the socially oriented stress task reflect a pattern of attenuated cardiovascular functioning during stress. As discussed previously, a number of recent studies have found blunted cardiovascular responses to be associated with maladaptive mood and mental health statuses (e.g., Neumann et al., 2011; Phillips et al., 2011). Moreover, it has been suggested that diminished stress responding could be as harmful to long-term health as exaggerated stress responding (see Lovallo, 2011). Attempts to exert a dominant position have been associated with increased risk of atherosclerosis, primarily thought to be mediated through patterns of exaggerated cardiovascular functioning and reactivity to stress. However, blunted patterns of

cardiovascular functioning could be another potential mechanism through which dominance is related to adverse cardiovascular outcomes.

For women higher in dominance, mean levels of SBP and HR were equivalent for those exposed to either social or asocial tasks, suggesting no significant differences in cardiovascular functioning between stressors. Typically, social stressors have been shown to elicit greater stress reactions than asocial stressors, while individual differences in personality may moderate the magnitude of different stress reactions. Indeed, previously dominance was found to be positively associated with cardiovascular functioning during social stress (e.g., Gramer & Berner, 2005; Hughes & Callinan, 2007). The current results showed that those lower in dominance displayed statistically significant trends that would be expected; greater cardiovascular functioning (SBP and HR) during social stress compared to asocial stress (e.g., al'Absi et al., 1997). The findings may suggest that there may have been no differentiation between social or asocial stress for higher dominant women, as physiological responses between conditions were not significantly different from each other. It could be that higher dominant participants may have been trying to exercise a characteristically dominant style of responding to social *and* asocial tasks, resulting in similar levels of cardiovascular stress functioning. In the long term, a failure to differentiate between types of stressors could be harmful in the long-term if people are unable to appreciate stressor differences, and to evaluate whether their individual characteristics are sufficient to meet the demands posed by the nature of the task.

Although CVR has primarily been found to be positively associated with trait dominance (e.g., Hughes & Callinan, 2007), an association between diminished cardiovascular functioning and dominance during socially relevant stress has also been observed. Smith et al. (1996) observed significantly greater CVR for women who were

motivated to behave in a style consistent with submissiveness or friendliness, than for women who were motivated to display dominant or hostile behaviour. These results support the current findings that trait dominance was associated with diminished cardiovascular functioning during social stress. While the results from Smith et al. (1996) parallel the current findings, the experimental protocol manipulated dominant *behaviour*, rather than the current study's interest in the dimension of dominance as a personality dimension, and thus could have been confounded. Smith et al. (1996) simulated interview questions for job vacancies in fields that were designed to be consistent with submissive or dominant behaviour (i.e., a position in customer service or in sales management respectively). Nonetheless, the findings do provide some support for the observation of diminished physiological responding to social stress by women higher in trait dominance observed in the present study. While clinically the magnitude of the differences in cardiovascular functioning may appear to be relatively small, results from the Prospective Studies Collaboration (2002) have shown that even small differences in cardiovascular parameters in young, healthy adults could have long-term cardiovascular health implications.

The diminished cardiovascular functioning effects for higher dominant participants were observed in spite of the fact that the stressors chosen in the current study were successful in eliciting stress responses from participants (as evidenced by significant quadratic trends for cardiovascular parameters, and increased blood pressure and HR during stress relative to baseline). The stressors chosen have been shown in previous studies to elicit differential cardiovascular effects that interacted with psychometrically-measured dominance (Hughes & Callinan, 2007), and thus were a good choice for the current study. Mean levels of trait dominance were found to be similarly moderate,

although marginally lower, in the current study ($M = 7.07$, $SD = 3.74$), relative to levels observed by Hughes and Callinan (2007) for women using the same instrument (JPRF; $M = 7.83$, $SD = 3.29$). This suggested that that the personality characteristics of the current sample were not entirely different from other studies, despite the observation of blunted effects.

The results point to the value of pursuing an investigation of the consequences of exposure to socially oriented stress in particular, in line with much of the previous research in the area (see Newton, 2009). Further work is needed to extend the results obtained by the present study to investigate the implications of repeated exposures to social stressors. Do less dominant individuals habituate to social stress, or does their elevated cardiovascular functioning persist or increase? Additionally, do the blunted effects observed for higher dominant women dissipate? The current study extends prior knowledge that dominance is related to cardiovascular responding to social forms of stress. However, significant trends were only identified for participants lower in dominance, suggesting that the relationship between trait dominance and stress is not linear, but multi-factorial. The relationship is contingent on the intensity of observed dominance, and the nature of the stress that is experienced.

Questions Arising From Study 2

Given the observed blunted reactions to social stress for high-dominant participants in the current study, the question arises as to what the contributing hemodynamic mechanisms underlying blunted cardiovascular functioning are? It has been suggested that blunted styles of cardiovascular functioning may not reflect a lack of cardiovascular responding *per se*, but instead reflect a different hemodynamic process underlying

cardiovascular regulation. Thus, further investigations into underlying hemodynamic regulation of blood pressure during social stress are warranted. Further, do the observed effects in the current study persist with repeated stress exposures?

Chapter 4: Study 3

Cardiovascular Habituation and the Relationship with Trait Dominance

Homeostasis refers to the ability to maintain physiological (and psychological) systems, encapsulated in the ability of an organism to return to a resting state once a challenge has passed (Cannon, 1932). Adaptation or habituation to stress is one mechanism by which homeostasis is maintained. Distinguishable from motor or sensory fatigue, according to the dual process theory (Groves & Thompson, 1970), habituation is demonstrated by attenuated responses to repeated stress. In contrast, incremental responses to stress are termed sensitisation (Groves & Thompson, 1970). Failure to accurately assess the magnitude of a stress response required by a task or situation could lead to long-term cardiovascular consequences if maladaptive patterns of stress responding persist. In laboratory examinations of habituation, a minimum of two stressor exposures has been shown to provide enough opportunity for individuals to demonstrate reduced physiological responses to stress (Frankish & Linden, 1991).

Although blood pressure reactivity is a useful index of the extent of physiological functioning prompted by stress, the complexities of the underlying cardiovascular mechanisms are not always apparent when blood pressure by itself is examined. Regulation of blood pressure involves a dynamic and compensatory interaction between processes of CO and TPR in order to achieve homeostasis. The inter-connected nature of these variables within the circulatory system is well-documented in medical physiology.

The operation of this reciprocal relationship between CO and TPR can be conceptualised as a process of flow and resistance (Levick, 2010). Increased CO results in greater ejection of blood from the ventricles of the heart to surrounding arteries, reflecting a physiological preparedness of the body to execute an action. In contrast, increased TPR reflects increased vascular constriction, causing an opposition or resistance in the blood vessels that serves to regulate blood flow (Levick, 2010). The term hemodynamic profile has been advocated as a means of describing this specific compensatory relationship. The complexity of the cardiovascular system is such that it is possible for two different people to have the same mean level of blood pressure, despite their individual blood pressure having different hemodynamic determinants, as noted by Brod, Fencel, Hejl, and Jirka (1959). Sherwood, Royal, and Light (1993) noticed anomalies between blood pressure and the hemodynamic profiles of two healthy young men. Despite having equivalent levels of blood pressure during baseline and during a mental arithmetic task, striking individual differences were observable between the two participant's CO and TPR during the mental arithmetic task. One participant displayed a large increase in CO coupled with a small decrease in TPR, while the second participant's response was characterised by a simultaneously modest increases in CO and a decrease in TPR (Sherwood et al., 1993). While blood pressure alone would have suggested that the two men were equivalent in their response to the mental arithmetic stressor, there were in fact differences in their cardiovascular responding.

Referring to the nature of the predominant hemodynamic mechanism underlying the blood pressure response, descriptions of the nature of CO-oriented and TPR-driven responses have been termed myocardial and vascular respectively (Sherwood & Turner, 1995). As mentioned earlier, blood pressure that is characterised by an increase in CO,

accompanied by decreased TPR is known as a myocardial or beta-adrenergic pattern. In contrast, blood pressure typified by increased TPR with a concurrent reduction in CO is termed a vascular or alpha-adrenergic response. Additionally, what is termed a mixed response, or blood pressure that is characterised by a dual increase (or decrease) in CO and TPR, is also possible. Thus, changes in blood pressure can be determined by either alpha- or beta-adrenergic factors, or indeed a combination of these factors.

Conforming to explanations of physiological activation under conditions of stress espoused by fight or flight theory (Cannon, 1915), the mobilisation of the cardiovascular system for a fight or flight response has been termed “active coping” (Obrist, 1976). The precise nature of a psychological stress task has been known to stimulate differing hemodynamic mechanisms. Active tasks (like public speaking or mental arithmetic stressors) have been shown to be associated with myocardial responses. It is thought that myocardial patterns of stress responding are adaptive initially, as they demonstrate an increased active physiological response to something that is stressful. Subsequent exposures to the same stressor have shown reduced beta-adrenergic stress responses in several studies (al'Absi et al., 1997; Frankish & Linden, 1991; Kelsey et al., 1999), with the suggestion that attenuation of myocardial responses to repeated stressor exposures are an almost universal occurrence (Kelsey, Soderlund, & Arthur, 2004). In contrast to myocardial processes, vascular profiles of stress reactivity are typically evoked by tasks requiring passive coping, such as the cold pressor task. Prolonged vascular responses to stress have been found to be associated with negative cardiovascular health outcomes (Palatini & Julius, 2009), whereby elevated vascular activity, and a return to normal levels of CO may lead to increased vascular resistance, and excessive vascular reactivity (Palatini & Julius, 2009). Unlike findings relating to myocardial processes, alpha-adrenergic

parameters have been shown to remain relatively stable across stress exposures, or to increase upon subsequent stressor exposures (al'Absi et al., 1997; Kelsey et al., 1999).

Individual variations in the magnitude, pattern, and duration of stress-induced hemodynamic responses could have implications for the nature and extent of pathophysiological change leading to CVD (James et al., 2012). Individual response stereotypy suggests that some individuals may respond to different types of stress with increased physiological activation in a particular physiological system (Lacey & Lacey, 1958). Therefore, people may have a characteristic way in which they respond to stress, regardless of the type of stress that they are exposed to (i.e. someone may be characterised by a myocardial or vascular pattern of responding). Individual differences in hemodynamic stress responsivity have been shown to have some temporal stability. For example, blood pressure has been shown to be affected by similar underlying hemodynamic mechanisms during competitive reaction time-tasks on two occasions (Sherwood, Turner, Light, & Blumenthal, 1990).

While many empirical studies have examined cardiovascular adaptation to active, yet asocial forms of stress (e.g., mental arithmetic tasks), fewer studies have looked at adaptation to more socially oriented stress tasks. Socially relevant experimental tasks such as public speaking tasks, are widely used in laboratory protocols for studying stress reactivity, have been found to elicit relatively high, stable, and homogeneous responses (al'Absi et al., 1997). Greater levels of CVR, and slower adaptation to the stressor have been found when repeated public speaking tasks were used, compared to repeated mental arithmetic stressors (al'Absi et al., 1997). In an era where tasks such as public speaking are rated as very stressful by many people, and there is considerable anxiety associated with

these types of challenges, it is of interest to examine the physiological consequences of such tasks experimentally.

The ability to habituate to subsequent presentations of similar stress is an adaptive skill, and one with great interpersonal variation. Some strong evidence has emerged for individual differences in stress responding for several aspects of personality, such as tendencies towards NA (e.g., emotional instability, anger, and anxiety) and socially relevant trait dominance. It has been found that people with greater dispositions towards neuroticism, anger, and hostility, and a desire for social dominance have shown profiles of CVR that are associated with negative future cardiovascular outcomes, such as increased incidence of CHD and atherosclerosis (see Booth-Kewley & Friedman, 1987; Everson-Rose & Lewis, 2005; Suls & Bunde, 2005). Specifically, several studies utilising animal models have shown individual differences in trait dominance to be associated with increased atherosclerosis and CVD progression in primates (e.g., Kaplan et al., 1982; Manuck, Marsland, Kaplan, & Williams, 1995). In humans too, trait dominance has been found to be positively associated with CVR to socially relevant forms of stress, but not asocial forms of stress (Gramer & Berner, 2005; Hughes & Callinan, 2007; Newton, 2009). Trait dominance has also been linked to some adverse health outcomes, including increased odds of both fatal and non-fatal cardiovascular events (Siegman, Kubzansky, et al., 2000), while greater submissiveness, has been found in some instances to be protective against ill-health (Siegman, Kubzansky, et al., 2000; Whiteman et al., 2000). A more dominant disposition means that individuals are more likely to actively compete for in-group success in a social hierarchy, at the expense of some others. It is argued that socially oriented tasks may be more relevant for people with higher trait dominance as they provide scope for dominance-related responding. Whereas, the people lower in dominance may

find the social stress tasks to be more threatening or challenging, because they do not have the same desire for hierarchy.

Study 2 showed that cardiovascular functioning during socially relevant stressors was significantly moderated by trait dominance. Questions arise about whether the observed associations between dominance and cardiovascular functioning persist across stressors, and also what the cardiovascular mechanisms underlying observed effects of trait dominance might be. Investigation of individual differences in personality, coupled with a consideration of the diversity of hemodynamic responding to repeated stress could elucidate variations in CVR more clearly. Thus, the following research questions are posed; during repeated exposures to socially oriented stressors, is trait dominance associated with CVR and habituation? Further, can individual differences in hemodynamic activity be identified as one mechanism through which increased cardiovascular vulnerability could proliferate?

Method

Participants

Participants were 75 undergraduate students (54 women and 21 men). Due to known gender differences in cardiovascular functioning, as well as considerations of statistical power, the sample was confined to women only for the purposes of statistical analysis ($n = 54$). The convention in research involving cardiovascular variables has reliably been to analyse data for men and women separately, due to known increased risks of CHD development for men compared to women (Lloyd-Jones, Larson, Beiser, & Levy, 1999). Although there is some suggestion that gender differences in CHD risk are

decreasing, with some pointing to the greater participation of women in the workforce, and the increased stress associated with this as one possible explanation for this (Frankenhaeuser, 1991).

For the purposes of this study, only women under the age of 35 years (ages ranged from 17 to 34; $M = 20.98$, $SD = 3.42$), who did not report a personal history of hypertension, with blood pressure within normal ranges at baseline (blood pressure < 140/90 mmHg), and who had a BMI of less than 30 ($M = 22.92$, $SD = 2.98$) were included in the final sample ($N = 46$). This final sample included nine smokers, who reported mean of 1.18 ($SD = 4.69$) cigarettes were smoked daily. As it was determined that the inclusion of smokers did not alter the trend of the results compared to when they were excluded from analyses, it was decided to include them in the final sample for reasons of increased power.

Measures

Cardiovascular responses.

Cardiovascular functioning was assessed using a Finometer hemodynamic cardiovascular monitor (Finapres Medical Systems BV, BT Arnhem, The Netherlands). The Finometer measures beat-to-beat cardiovascular functioning non-invasively and is the successor of previous cardiovascular monitoring devices, the TNO Finapres-model-5 and the Ohmeda Finapres 2300e, which have been used in the previous research (e.g., Beckham et al., 2002; Gregg, Matyas, & James, 2002; Philippsen et al., 2007; Van Rooyen et al., 2004).

Determination of cardiovascular functioning using the Finometer is premised on the volume-clamp method, first developed by Penáz (1973). With this method, finger

arterial pressure is measured using an appropriately-sized finger cuff (attached to the middle finger of the non-dominant hand) and an inflatable bladder cuff (attached to the upper arm). This apparatus is used in combination with an in-built infrared photoplethysmograph (attached to the wrist), which consists of an infrared light source and detector. The emitted infrared light in the finger cuff is absorbed by blood in the arteries, and pulsation of arterial diameter during a heart beat causes a pulsation in the light detector signal.

To assess cardiovascular function, the unloaded diameter of the finger arteries is found (i.e., the point at which equal pressure inside and outside of the artery is achieved), and maintained by clamping the artery at this volume. When the volume clamp is active at the proper unloaded diameter, intra-arterial pressure is equal to finger cuff pressure. The artery is kept at this unloaded diameter by varying the pressure at the finger cuff (through a pulsation of the cuff). The individualised determination of the volume at which the artery should be clamped is automatically calculated using PhysioCal (Finapres Medical Systems, Amsterdam). The original volume clamp method (Peñáz, 1973) required operator expertise in the determination of the correct clamping volume, but the development of the PhysioCal algorithm (Wesseling, De Wit, Van der Hoeven, Van Goudoever, & Settels, 1995) simplified this process. PhysioCal makes continuous tracking of the correct clamping volume possible, allowing for periodic adjustments to volume to be made without interrupting the continuous measurement. Continuous measures of arterial CO, stroke volume (SV), and TPR are estimated from arterial blood pressure waveforms using the validated Modelflow modelling method (Wesseling et al., 1995; Wesseling, Jansen, Settels, & Schreuder, 1993). Modelflow uses a non-linear, three-element model of aortic input impedance to describe left ventricular functioning (Wesseling et al., 1993). The

model includes the three major properties of the aorta and the arterial system; aortic impedance, aortic compliance, and systemic vascular resistance. Age, sex, height, and weight are required for the computation of Modelflow (as they are involved in the determination of the individual aortic pressure–area relationship), and so were entered into the Finometer prior to the commencement of measurement.

The Finometer has been shown to provide accurate blood pressure measurement in young populations (Schutte, Huisman, van Rooyen, Oosthuizen, & Jerling, 2003) and in cardiac populations (Guelen et al., 2003). These studies report that the validation criteria of the Association for the Advancement of Medical Instrumentation are satisfied by the Finometer, and it is therefore recommended for use in research and in clinical settings.

Psychometric measurement.

Trait dominance was measured using the JPRF social dominance subscale (Jackson, 1999). Sixteen items assessed trait dominance, ($M = 5.24$, $SD = 4.10$; possible scores ranged 0-16). High scorers on the trait dominance subscale of the JPRF attempt to control their environments and influence or direct other people; they are forceful, decisive, authoritative, and domineering (Pratto et al., 1994). Reliability in the current study for trait dominance was good ($\alpha = .88$). Data for several other psychometric variables were collected, but are not the subject of present analysis (see Table 14 for descriptive statistics).

Subjective task ratings.

Participants provided subjective ratings for three separate items measuring task engagement; perceived stress, interest, and difficulty. Participants were asked *how*

Table 14

Descriptive Statistics for Psychometric Variables (by item) Including Estimates of Reliability

Variable	Women ($n = 46$)					Men ($n = 18$)				
	α	Mean	SD	Min.	Max.	α	Mean	SD	Min.	Max.
Neuroticism ^a	.86	3.22	0.66	1.50	4.58	.79	2.83	0.57	2.08	3.83
Extraversion ^a	.77	3.49	0.50	2.25	4.33	.89	3.23	0.62	1.42	3.92
Openness ^a	.81	3.41	0.57	1.92	4.42	.84	3.52	0.60	2.42	4.50
Conscientiousness ^a	.84	3.38	0.60	1.75	4.58	.64	3.25	0.41	2.42	3.83
Agreeableness ^a	.68	3.81	0.41	3.00	4.75	.89	3.49	0.62	1.67	4.50
Trait dominance ^b	.88	0.38	0.29	0.00	1.00	.82	0.58	0.26	0.07	0.93
Social Desirability ^c	.68	0.45	0.13	0.15	0.88	.79	0.45	0.16	0.15	0.70
Trait Anxiety ^d	.85	2.23	0.41	1.30	3.00	.82	2.12	0.35	1.65	2.85
State Anger ^e	.86	0.06	0.00	0.00	0.80	.47	0.07	0.08	0.00	0.27
Trait Anger ^e	.79	1.76	0.40	1.20	3.00	.89	1.77	0.47	1.10	2.90
CMHo ^f	.89	1.17	0.31	0.30	1.82	.87	1.33	0.26	0.80	1.78

Table 14 Continued

Cynical Distrust ^f	.69	1.07	0.42	0.13	1.88	.75	1.22	0.41	0.50	2.00
Cynicism ^f	.78	1.22	0.39	0.15	2.00	.73	1.36	0.32	0.92	2.15
BPAQ ^g	.90	2.22	0.54	1.21	3.69	.91	2.37	0.54	1.55	3.48
SAM threat ^h	.89	1.43	0.96	0.00	3.75	.83	0.97	0.69	0.00	2.50
SAM Challenge ^h	.77	1.19	0.69	0.00	2.75	.73	1.75	0.69	0.25	2.75
PSS ⁱ	.83	1.99	0.51	0.86	3.07	.78	1.65	0.40	0.79	2.36
MSPSS Sig Other ^j	.89	6.10	1.10	2.25	7.00	.95	5.46	1.13	3.50	7.00
MSPSS Family ^j	.89	5.88	0.96	3.50	7.00	.84	5.40	1.25	2.50	7.00
MSPSS Friends ^j	.93	5.84	1.18	2.00	7.00	.93	5.26	1.20	2.50	7.00
MSPSS Total ^j	.93	5.94	0.91	2.75	7.00	.92	5.38	0.98	3.58	7.00
SSQ6 Number ^k	.87	4.17	1.63	0.00	9.00	.94	3.27	1.84	0.83	8.33
SSQ6 Satisfaction ^l	.28	5.27	1.79	1.00	15.17	.17	5.12	2.19	3.17	13.17
Active ^m	.85	1.62	0.94	0.00	3.00	.71	1.61	0.68	0.50	3.00
Planning ^m	.86	1.54	0.95	0.00	3.00	.66	1.72	0.65	0.50	2.50

Table 14 Continued

Emotional ^m	.73	1.64	0.89	0.00	3.00	.73	1.19	0.77	0.00	3.00
Instrumental ^m	.91	1.48	1.02	0.00	3.00	.70	1.08	0.86	0.00	2.00

Note. *SD* = standard deviation; α = Chronbach's alpha coefficient; Min. = minimum; Max. = maximum; CMHo = Cook-Medley hostility inventory; BPAQ = Buss-Perry aggression questionnaire; SAM = stress appraisal measure; PSS = perceived stress scale; MSPSS = multidimensional scale of perceived social support; SSQ6 = Sarason's six-item social support questionnaire; COPE = coping inventory.

^aMeasured using mean item scores from the appropriate subscale of the NEO FFI (Costa & McCrae, 1992).

^bMeasured using the trait dominance subscale of the Jackson Personality Form (Jackson, 1999).

^cMeasured using the social desirability scale (Crowne & Marlowe, 1960)

^dMeasured using mean item scores from the appropriate subscale of the State-Trait Anxiety Inventory (STAI; Spielberger et al., 1977).

^eMeasured using the trait or state anger subscale of the State-Trait Anger Expression Inventory 2 (Spielberger, 1999).

^fMeasured using mean item scores from the item Cook-Medley Hostility Inventory (Cook & Medley, 1954), or its cynical distrust (Greenglass & Julkunen, 1989), or cynicism (Barefoot et al., 1989) subscales.

^gMeasured using the Buss Perry Aggression Questionnaire (Buss & Perry, 1992).

^hMeasured using the Stress Appraisal Measure (Peacock & Wong, 1990)

ⁱMeasured using mean item scores from the PSS (S. Cohen et al., 1983).

^jMeasured using appropriate subscales of the Multidimensional Scale of Perceived Social Support, (Zimet et al., 1988).

^kMeasured using the *N* subscale of the SSQ6 (Sarason et al., 1987).

^lMeasured using the *S* subscale of the SSQ6 (Sarason et al., 1987).

^mMeasured appropriate subscales of the brief ways of coping scale (Carver, 1997).

“stressful/ interesting/ difficult did you find the task” following their completion of the stress tasks. Answers were obtained using a five-point Likert response scale, with possible scores ranging from 0 (not at all; stressful, interesting or difficult) to 4 (extremely; stressful, interesting or difficult).

Speech Task.

Participants were asked to perform two of three possible speech task scenarios. The speech tasks chosen were similar in format and content to procedures previously employed in published research (e.g., Bostock, Hamer, Wawrzyniak, Mitchell, & Steptoe, 2011; Hamer, Tanaka, Okamura, Tsuda, & Steptoe, 2007; Hamer et al., 2006). The content of the scenarios were chosen for their relevance to the largely young, undergraduate sample. The researcher described the scenario, participants were given two minutes to prepare their speech, and following this they were asked to deliver their speech to a video camera for two minutes. Some paper and pens were provided for participants to use if they wished during the preparation phase. Participants were told that their speeches would be recorded, and that the tape would later be evaluated by the researcher for overall content, clarity and delivery. The text of the scenario was available to participants throughout the preparation and speech phases. The instructions delivered to participants were as follows:

I'm going to present you with a hypothetical scenario, and you will be asked to give a speech about this scenario. The scenario is randomly generated, so any resemblance to previous experiences of yours is completely coincidental. I will briefly describe this scenario to you, and you will be asked to then spend two minutes briefly preparing a speech

in response to the scenario. When you are preparing your speech, you should try to imagine how you might feel, and how you might react if you were faced with this same situation. After the two minutes of preparation time, I will ask you to begin to deliver your two minute speech. Please try to continue speaking for the full two minutes of the task, I will tell you when the time is up.

Participants were asked to speak about two of these three scenarios:

1. You are falsely accused of shoplifting by a security guard as you are leaving a shop. Please prepare the speech that you might give in defence of yourself.
2. You are falsely accused of cheating on an exam by an invigilator. Please prepare the speech that you might give in defence of yourself.
3. Your boss for your part-time job falsely believes that you are not performing your duties well-enough, and threatens to fire you. Please prepare the speech that you might give in defence of yourself.

Procedure

Testing took place in a small university psychophysiological laboratory. Participants were invited to read an information sheet, and to sign a consent form if they agreed to participate. Age, gender, height, and weight were recorded for the correct calibration of the Finometer. Participants were seated in a comfortable chair and

familiarised with the Finometer apparatus. It was attached to their non-dominant hand, adjusted for participant comfort, but remained switched off. For 10 minutes, participants were asked to relax while completing some psychometric questionnaires. After 10 minutes, participants were asked to put aside the questionnaires and to relax for a further 10 minutes. A National Geographic magazine was provided to assist with this relaxation (Jennings, Kamarck, Stewart, Eddy, & Johnson, 1992). Continuous blood pressure monitoring began at this time. After 10 minutes spent relaxing and reading the magazine, participants were given instructions for the first stress task and asked to spend two minutes preparing for this task. The two-minute speech stressor was followed by a three-minute resting period, and the second speech stressor instructions, preparation, and task time. Finally participants were detached from the Finometer and completed the remainder of the psychometric questionnaires. All testing took place in the same laboratory. The researcher remained in the room throughout testing, but was separated from the participant by an opaque screen.

Overview of Analyses

To assess whether there were any differences in task engagement between participants over the two exposures to the stressors, a series of *t* tests were conducted to first assess whether there were differences in participants higher and lower in trait dominance, and secondly whether there were difference in task engagement between Exposure 1 and 2. Pearson's correlation analyses were used to confirm these group-based analyses.

Beat-to-beat cardiovascular data for SBP, DBP, HR, mean arterial pressure (MAP), CO, TPR, SV, and inter-beat interval (IBI) were first reduced to 30 second intervals. Mean

values were then computed for each experimental phase; namely baseline, Exposure 1, resting post-Exposure 1, and Exposure 2 phases.

In order to examine whether the stressor was successful in eliciting CVR, one way within subjects ANOVA, with *post hoc* comparisons of means, was conducted for each cardiovascular parameter (SBP, DBP, HR, MAP, CO, TPR, SV, and IBI) separately, with phase (baseline, Exposure 1, resting, and Exposure 2) values entered as the within subjects factor. Due to the likelihood of the relationship between the variables being best identified as a polynomial function, cubic and quadratic within-subject contrasts were considered. Significant main effects were taken to indicate a change in cardiovascular functioning over the phases of the experiment. With the inclusion of more than two levels to the within subjects factor, estimates of Sphericity were corrected using Greenhouse-Geisser epsilon corrections (ϵ) where assumptions of Sphericity were not met.

To examine cardiovascular adaptation, it was first considered if cardiovascular parameters had returned to their baseline states during the inter-stressor resting period by conducting paired samples *t* tests (baseline versus resting phases) for each cardiovascular parameter. Cardiovascular functioning during the inter-stressor resting phase that was equivalent to or lower than baseline levels was taken to be indicative of the occurrence of adaptation. For those cardiovascular parameters that were found to return to baseline levels following the cessation of the stressor, adaptation was calculated as the arithmetic difference between stressor phase and the immediately preceding baseline (Exposure 1), or resting period (Exposure 2). For cardiovascular parameters failing to return to baseline levels during the resting phase, adaptation was computed as the arithmetic difference between stressor levels of the cardiovascular parameter and baseline levels of that parameter.

For those parameters that did not achieve adaptation, 2×1 repeated measures ANCOVAs were conducted, with reactivity to Exposures 1 and 2 entered as the two levels of the within subjects factor, and trait dominance as the continuous covariate to determine the effects of trait dominance on CVR. Baseline levels of each cardiovascular parameter were entered in these ANCOVAs as an additional covariate, given that baseline values are likely to be systematically associated with subsequent stressor levels of cardiovascular functioning (Benjamin, 1967). Significant main effects for phase indicated reactivity of cardiovascular parameters to the experimental stressors, while significant main effects for trait dominance were taken to indicate associations between trait dominance and cardiovascular functioning, regardless of experimental phase. Significant phase by trait dominance interactions suggested an effect of trait dominance on CVR.

To examine the effect of trait dominance on CVR for parameters that reached adaptation (i.e., those that had a non-significant difference between baseline and resting levels of functioning), 4×1 repeated measures ANCOVA were conducted for each cardiovascular parameter. Experimental phase (baseline, Exposure 1, post- Exposure 1 resting, and Exposure 2) was entered as the within subjects factor, and trait dominance as the continuous covariate. In these ANCOVA analyses, patterns of cardiovascular functioning were assessed using cubic-level and quadratic-level within-subject contrasts to examine change over the four experimental phases. For analyses with more than two levels to the within-subject factor (i.e., phase), significant effects involving the within-subject factor were explored using *post hoc* comparison of responses. As appropriate, corrections to violations of the assumption of Sphericity were made using Greenhouse-Geisser epsilon corrections (ϵ) when violations of the assumption of Sphericity were indicated by a significant Mauchly's test.

Effect sizes are presented as partial η^2 for ANOVA effects, and r for correlation coefficients. Eta-squared values of .04, .25, and .64, and r -values of .1, .25, and .37 were taken to represent small, medium, and large effect sizes respectively (J. Cohen, 1988, 1992).

Results

Subjective Task Ratings

Independent samples t tests revealed no significant differences between women higher and lower in trait dominance (based on a median split) for ratings of either Exposures 1 or 2 as stressful, interesting or difficult ($ps > .46$). Paired samples t tests to examine trends for each group over time found that there were some significant differences for participants lower in trait dominance, such that these participants rated the Exposure 2 to be significantly less stressful ($p = .001$), and significantly less difficult ($p = .01$) than Exposure 1. No such significant differences were found for participants higher in trait dominance, although there was a trend toward lower levels of both stressfulness and difficulty ($ps = .07$; see Table 15 for means and standard deviation of task engagement variables). Pearson's correlations showed small inverse correlations (ranging from $r = -.08$ to $r = -.16$) between trait dominance and each aspect of task engagement, but none reached statistical significance ($ps > .31$). Additionally, no significant differences were observed between those higher and lower in trait dominance and their subjective ratings of tasks as stressful, interesting, or difficult ($ps > .46$), suggesting that irrespective of dispositional dominance, similar levels of stress, difficulty, interest during Exposures 1 and 2 were experienced by all participants.

Table 15

Paired Samples t tests Showing Differences Between Task Engagement for Higher and Lower Dominant Participants Across Exposures 1 and 2

Task ratings	Low Dominance ($n = 21$)						High Dominance ($n = 25$)					
	Exposure 1		Exposure 2		<i>t</i> -test		Exposure 1		Exposure 2		<i>t</i> -test	
	Mean	<i>SD</i>	Mean	<i>SD</i>	<i>t</i> (20)	<i>p</i>	Mean	<i>SD</i>	Mean	<i>SD</i>	<i>t</i> (24)	<i>p</i>
Stressful?	2.48	1.08	2.10	1.09	2.94	.001	2.28	0.94	2.00	1.19	1.90	.07
Interesting?	1.67	0.73	1.67	0.73	.000	1.0	1.60	1.04	1.64	1.15	-0.33	.75
Difficult?	2.48	0.98	2.14	1.11	3.16	.01	2.24	1.13	1.92	1.32	1.88	.07

Note. *SD* = standard deviation.

Confirmation of Reactivity

Excellent internal consistency was observed for all cardiovascular parameters (Chronbach's $\alpha > .96$; see Table 16 for descriptive statistics for women and Table 17 for men). One way ANOVA using mean values of the cardiovascular parameters revealed significant linear and within-subject contrasts effects (significant at the cubic level of within-subject contrasts) for all parameters ($p < .001$), except TPR.

In the case of TPR, trends were found to be significant at the level of both the linear and quadratic function ($p = .001$). Effect sizes ranged from partial $\eta^2 = .81$ for DBP, to partial $\eta^2 = .24$ for TPR. The polynomial effects were supportive of the premise that there was a significant change in cardiovascular parameters over successive phases of the experiment (see Table 18 for paired samples t tests demonstrating overall change over the experimental phases for women).

Cardiovascular Adaptation

To investigate adaptation of each cardiovascular parameter after Exposure 1, paired samples t tests investigated potential differences between baseline and resting periods. The results of these paired samples t tests showed that only HR, $t(45) = -1.75, p = .09$; TPR, $t(45) = .21, p = .83$; and IBI, $t(45) = 1.27, p = .21$, were found to return to baseline levels during the post-Exposure 1 resting period, with non-significant differences between baseline and resting cardiovascular functioning. For SBP, $t(45) = -13.24, p < .001$; DBP, $t(45) = -14.81, p < .001$; MAP, $t(45) = -15.90, p < .001$; CO, $t(45) = -5.83, p < .001$; and SV, $t(45) = -5.61, p < .001$, mean cardiovascular functioning during the resting period was found to be significantly higher than at baseline (see Table 16). For those parameters that failed to return to baseline levels (i.e., SBP, DBP, MAP, CO, and SV), the method of

Table 16

Descriptives of Cardiovascular Parameters by Phase for Women (N = 46) with Reliability Statistics

Variable	Baseline			Exposure 1			Resting			Exposure 2			Cubic trend	
	α	Mean	<i>SD</i>	α	Mean	<i>SD</i>	α	Mean	<i>SD</i>	α	Mean	<i>SD</i>	<i>p</i>	η^2
SBP ^a	.98	117.21	9.98	.96	137.84	14.66	.96	129.71***	11.18	.97	139.48	15.35	< .001	.72
DBP ^a	.99	72.32	7.76	.96	86.65	10.41	.97	79.25***	8.21	.97	88.92	9.94	< .001	.81
HR ^b	.99	78.60	11.37	.97	92.46	14.17	.98	79.52	11.84	.98	89.70	14.11	< .001	.72
MAP ^a	.99	90.62	8.62	.96	108.81	12.09	.96	100.43***	9.02	.97	110.72	11.54	< .001	.80
CO ^c	.99	5.18	1.18	.98	6.31	1.65	.98	5.73***	1.33	.99	5.90	1.62	< .001	.50
TPR ^d	.99	1.17	0.50	.97	1.15	0.44	.99	1.16	0.47	.98	1.27	0.05	.89	.24
SV ^e	.99	66.63	13.90	.98	68.76	15.25	.99	73.14***	16.24	.99	66.44	16.17	< .001	.33
IBI ^f	.98	0.79	0.13	.96	0.67	0.11	.98	0.78	0.13	.96	0.70	0.11	< .001	.70

Note. Asterisks refer to statistical significance of paired samples *t* tests conducted to compare baseline and resting values for cardiovascular parameters.

^ammHg. ^bbpm. ^clpm. ^dpru. ^eunits. ^fms.

*** *p* < .001.

Table 17

Descriptives of Cardiovascular Parameters by Phase for Men (n = 18) with Reliability Statistics

Variable	Baseline			Exposure 1			Resting			Exposure 2		
	α	Mean	SD	α	Mean	SD	α	Mean	SD	α	Mean	SD
SBP ^a	.96	118.90	8.98	.96	145.73	14.42	.94	135.12	10.76	.96	145.77	14.24
DBP ^a	.98	74.39	7.22	.97	90.93	9.75	.96	83.17	7.54	.96	91.60	9.09
HR ^b	.98	77.81	9.97	.95	93.28	13.28	.98	79.01	12.03	.98	90.51	14.87
MAP ^a	.97	91.00	7.00	.96	112.83	11.31	.95	103.42	8.37	.96	113.26	10.58
CO ^c	.99	6.58	1.64	.98	8.12	2.13	.99	7.26	2.00	.99	7.69	2.31
TPR ^d	.99	0.90	0.25	.96	0.90	0.25	.97	0.93	0.26	.98	0.97	0.28
SV ^e	.99	84.73	16.29	.98	87.46	17.62	.99	92.10	17.59	.98	84.91	17.77
IBI ^f	.98	0.79	0.12	.94	0.67	0.10	.98	0.79	0.13	.98	0.69	0.11

^ammHg. ^bbpm. ^clpm. ^dpru. ^eunits. ^fms.

Table 18

Exposure-Elicited Cardiovascular Responding over Experimental Phases (Paired Samples t tests)

Parameter	Baseline-Exposure 1		Exposure 1-Resting		Resting-Exposure 2	
	<i>t</i> (45)	<i>p</i>	<i>t</i> (45)	<i>p</i>	<i>t</i> (45)	<i>p</i>
SBP ^a	-14.52	<.001	7.37	<.001	-7.57	<.001
DBP ^a	-16.82	<.001	10.22	<.001	-13.78	<.001
HR ^b	-9.84	<.001	10.63	<.001	-9.89	<.001
MAP ^a	-16.82	<.001	9.92	<.001	-11.96	<.001
CO ^c	-7.73	<.001	6.78	<.001	-1.98	.05
TPR ^d	0.41	.69	-0.41	.69	-5.42	<.001
SV ^e	-1.89	.07	-5.75	<.001	8.18	<.001
IBI ^f	9.63	<.001	-9.96	<.001	9.69	<.001

^ammHg. ^bbpm. ^clpm. ^dpru. ^eunits. ^fms.

analysing CVR of these variables to Exposures 1 and 2 differed from those variables deemed to have demonstrated adaptation (i.e., HR, TPR, and IBI), as described above.

Cardiovascular Reactivity

Blood pressure (SBP, DBP, MAP). In order to examine the primary agents of cardiovascular functioning, 2×1 ANCOVAs were conducted to analyse (separately) the effects of SBP, DBP, and MAP reactivity to stress. Trait dominance and initial baseline

values were entered as covariates in the appropriate analyses. For SBP, no significant main effects for reactivity, $F(1, 42) = 2.47, p = .12$, partial $\eta^2 = .06$; trait dominance, $F(1, 42) = .04, p = .84$, partial $\eta^2 = .001$; or baseline SBP values $F(1, 42) = 0.06, p = .81$, partial $\eta^2 = .001$, were found to be significant. No significant reactivity \times dominance, $F(1, 42) = 0.47, p = .50$, partial $\eta^2 = .01$; reactivity \times baseline, $F(1, 42) = 2.15, p = .15$, partial $\eta^2 = .05$; baseline \times dominance, $F(1, 42) = 0.01, p = .93$, partial $\eta^2 < .001$; or reactivity \times dominance \times baseline, $F(1, 42) = 0.40, p = .53$, partial $\eta^2 = .01$, interactions were found to be significant. Thus, it seemed that SBP reactivity did not significantly differ over stressor exposures, and SBP reactivity did not interact significantly with trait dominance (see Figure 4a).

For DBP a significant main effect for reactivity, $F(1, 42) = 8.04, p = .01$, partial $\eta^2 = .16$; and a significant reactivity \times baseline interaction, $F(1, 42) = 6.11, p = .02$, partial $\eta^2 = .13$, were found. No significant main effects for dominance, $F(1, 42) = .08, p = .78$, partial $\eta^2 = .002$; or baseline DBP, $F(1, 42) = .12, p = .74$, partial $\eta^2 = .003$, were observed. Additionally, no significant reactivity \times dominance, $F(1, 42) = 2.67, p = .11$, partial $\eta^2 = .06$; or reactivity \times baseline \times dominance, $F(1, 42) = 2.44, p = .13$, partial $\eta^2 = .06$, interactions were revealed. This suggested that DBP reactivity differed over task exposures, with greater reactivity found for Exposure 2, but did not significantly interact with dominance (see Figure 4b).

For MAP, similar to findings were observed as those relating to DBP, such that significant main effects for reactivity, $F(1, 42) = 7.96, p = .01$, partial $\eta^2 = .16$; and a significant reactivity \times baseline interaction, $F(1, 42) = 6.73, p = .01$, partial $\eta^2 = .14$, were found. No other main or interaction effects involving MAP were found to be statistically significant ($ps > .19$).

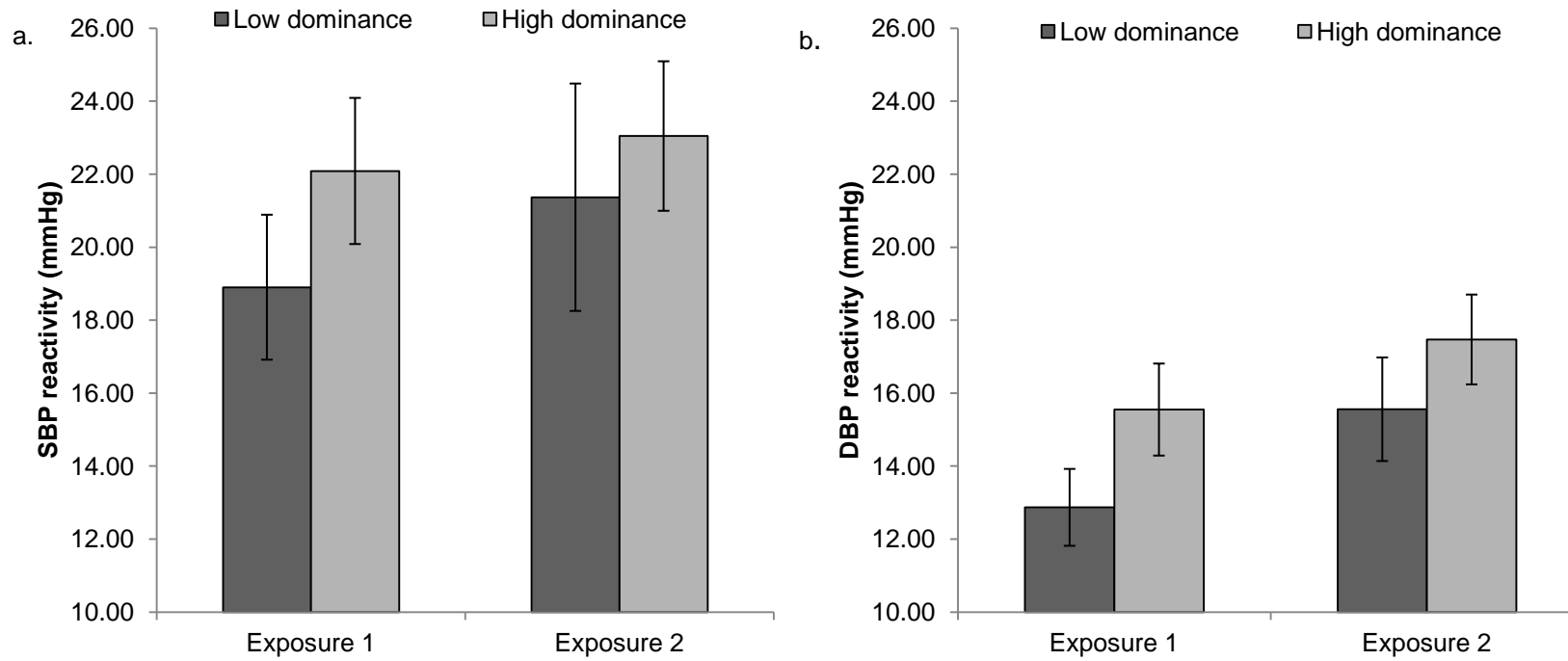


Figure 4. Reactivity of SBP (a) and DBP (b) to Exposures 1 and 2 with illustrative median splits for low ($n = 21$) and high ($n = 25$) trait dominance. Error bars denote standard error.

Although DBP and MAP were observed to change significantly over the course of the experimental phases, with increased DBP and MAP reactivity during Exposure 2 (DBP; $M = 16.60$, $SD = 6.32$, MAP; $M = 20.10$ $SD = 8.31$) compared to Exposure 1 (DBP; $M = 14.32$, $SD = 5.78$, MAP; $M = 18.18$, $SD = 7.33$), it appeared that the covariate of trait dominance was not associated with these changes in cardiovascular functioning. Blood pressure, as determined by SBP and DBP (and by MAP, an estimate of average blood pressure which is approximated from SBP and DBP), remained at similar levels for those higher and lower in trait dominance across successive stressor exposures (see Figure 4b for DBP).

CO and TPR. A of 2×1 ANCOVA for the analysis of CO, with trait dominance and baseline CO entered as covariates revealed that for CO, no significant main effects for reactivity, $F(1, 42) = 2.22$, $p = .14$, partial $\eta^2 = .05$; trait dominance $F(1, 42) = .03$, $p = .96$, partial $\eta^2 < .001$; or baseline CO, $F(1, 42) = 0.25$, $p = .62$, partial $\eta^2 = .01$ were found. Additionally, no significant reactivity \times dominance, $F(1, 42) = 1.70$, $p = .20$, partial $\eta^2 = .04$; reactivity \times baseline $F(1, 42) = 0.50$, $p = .48$, partial $\eta^2 = .01$; baseline \times dominance, $F(1, 42) = 0.22$, $p = .64$, partial $\eta^2 = .01$; or reactivity \times dominance \times baseline, $F(1, 42) = 1.32$, $p = .26$, partial $\eta^2 = .03$, interactions were found. Although a significant inverse correlation between dominance and CO reactivity to Exposure 1 was observed ($r = -.35$), the results of the ANCOVA showed that CO reactivity did not vary significantly for Exposure 1 and 2, and this stability of CO was irrespective of participant's disposition towards trait dominance (see Figure 5a).

The results of 4×1 ANCOVA for the analysis of TPR, with trait dominance as a covariate, showed significant main effects for TPR functioning at the level of a quadratic

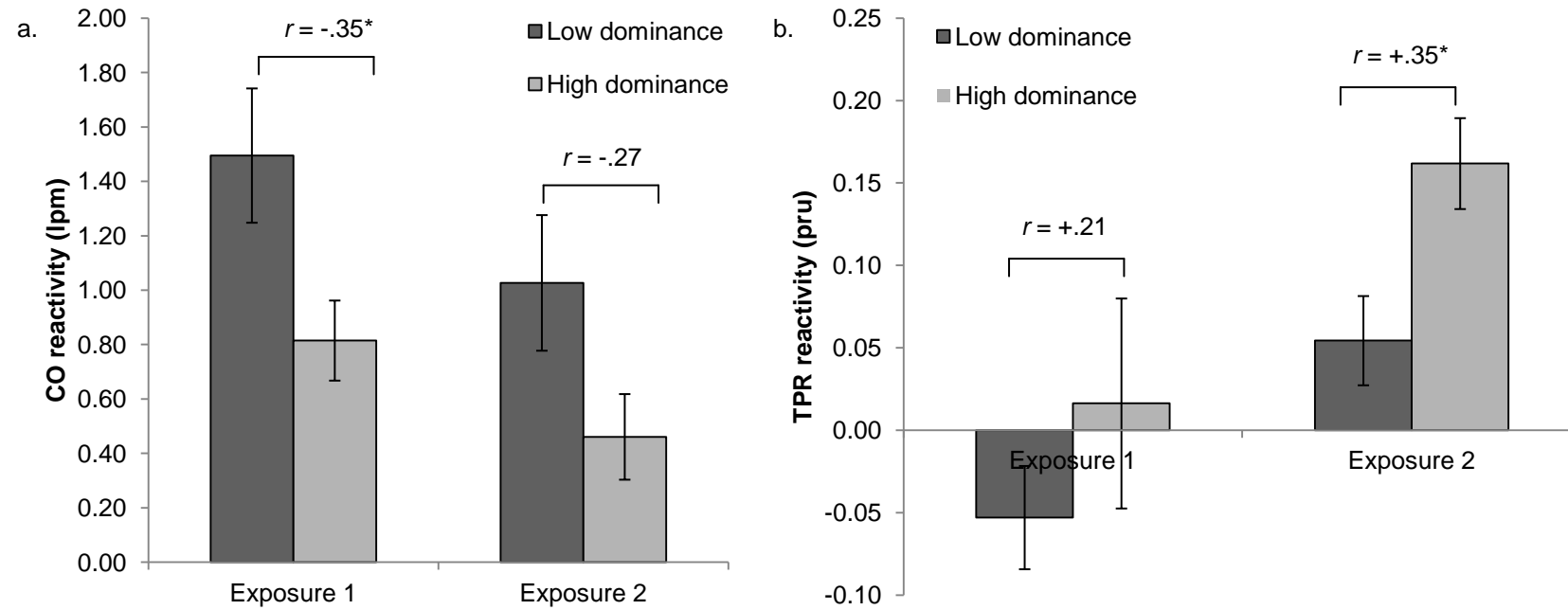


Figure 5. Reactivity of CO (a) and TPR (b) to Exposures 1 and 2 for participants low ($n = 21$) and high ($n = 25$) in trait dominance. Error bars denote standard error of the mean. Values for r refer to the Pearson's correlation between stressor exposure reactivity and trait dominance.

* $p < .05$

function, $F(1, 44) = 5.49, p = .02$, partial $\eta^2 = .11$; and a significant main effect for dominance, $F(1, 44) = 5.70, p = .02$, partial $\eta^2 = .12$ (see Figure 6b). A significant phase \times trait dominance interaction that was cubic in nature was also observed, $F(1, 44) = 6.79, p = .01$, partial $\eta^2 = .13$. Further analysis of the findings using a mixed ANOVA procedure, with median splits for dominance, qualified the utility of using median splits to illustrate the trends for TPR, as significance was retained for the phase \times trait dominance interaction at the cubic level, $F(1, 44) = 6.09, p = .02$, partial $\eta^2 = .12$. Pearson's correlation analyses between the continuous trait dominance scores and indices of CVR to stressor exposures demonstrated significant positive relationships with TPR during Exposures 1 ($r = .41, p = .01$) and 2 ($r = .37, p = .01$), confirming that greater TPR reactivity was associated with greater trait dominance.

The cubic nature of the phase \times trait dominance interaction observed for TPR suggested that further examination of the effects of trait dominance on TPR reactivity to experimental phase should be considered. A *post hoc* 2×1 repeated measures ANCOVA was conducted with TPR reactivity (calculated as the arithmetic difference between the stress exposure level of TPR and the TPR of the immediately preceding baseline or resting phase) to Exposure 1 and Exposure 2 entered as the two levels of the within-subject factor (see Figure 5b). Continuous trait dominance scores were entered as the covariate. In order to control for initial values, baseline TPR was added as an additional covariate.

The results of the 2×1 ANCOVA revealed significant main effects for reactivity, $F(1, 42) = 18.87, p < .001$, partial $\eta^2 = .31$; and for baseline TPR, $F(1, 42) = 5.67, p = .02$, partial $\eta^2 = .12$. Significant interaction effects for reactivity by baseline TPR, $F(1, 42) = 27.48, p < .001$, partial $\eta^2 = .40$; and reactivity by baseline by trait dominance, $F(1, 42) =$

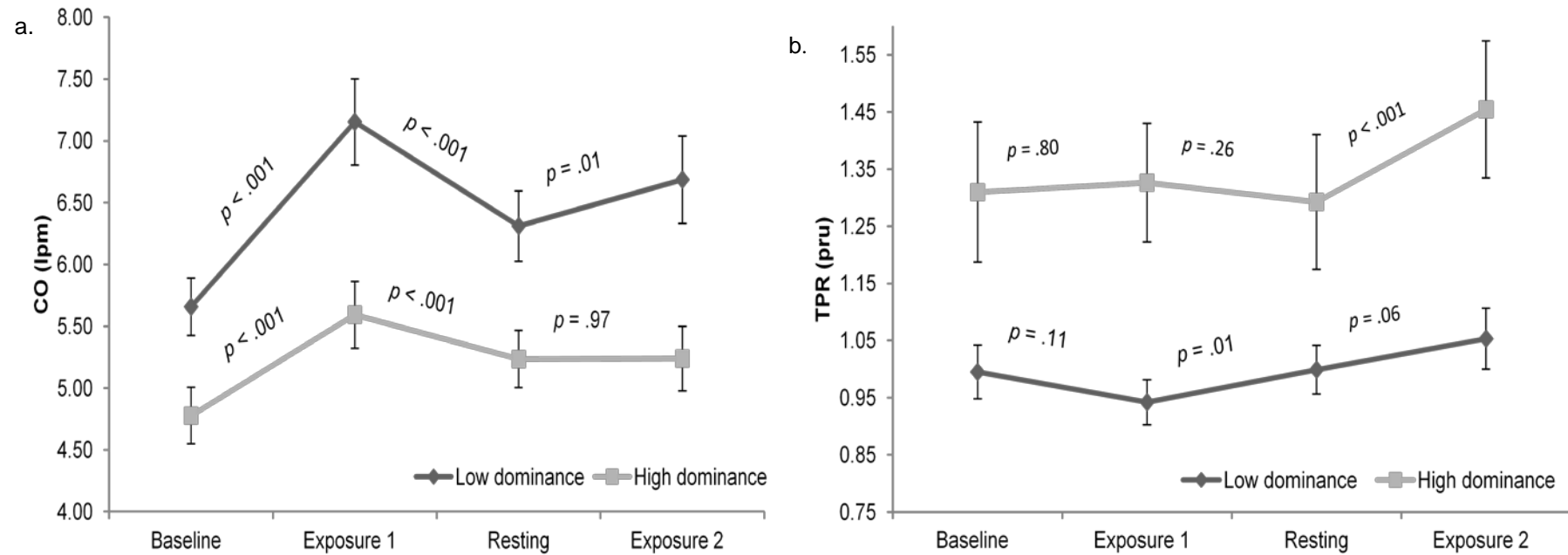


Figure 6. Patterns of CO (a) and TPR (b) across successive stressor exposures with median split low ($n = 21$) and high trait dominance ($n = 25$). p values represent significance of the changes from each experimental change to the next. Error bars denote standard error of the mean.

15.40, $p < .001$, partial $\eta^2 = .27$, were also observed. No significant main effect for dominance was revealed, $F(1, 42) = .21$, $p = .65$, partial $\eta^2 = .01$. However, a reactivity by trait dominance was found to be significant, $F(1, 42) = 11.24$, $p = .002$, partial $\eta^2 = .21$, suggesting that independent of the effect of baseline TPR, trait dominance was associated with TPR reactivity to Exposures 1 and 2. Pearson's correlations showed significant positive correlations between mean social dominance scores and TPR reactivity to Exposure 2 ($r = .35$, $p = .02$), but not Exposure 1 ($r = .21$, $p = .16$), confirming the direction of the findings. For Exposure 1, mean TPR reactivity was quite low for all participants. However, low trait dominant participants displayed attenuated TPR during Exposure 1 compared to TPR observed at baseline, which can be viewed as an inverse pattern of reactivity (see Figure 6b). For Exposure 2, participants higher and lower in trait dominance exhibited reactivity to the stressor, relative to the preceding resting period, with greatest TPR reactivity exhibited by those scoring higher on the measure of trait dominance.

Other cardiovascular parameters. The results of a 2×1 ANCOVA for SV showed a significant main effect for phase, $F(1, 42) = 9.00$, $p = .01$, partial $\eta^2 = .18$; a significant phase \times baseline SV interaction, $F(1, 42) = 6.67$, $p = .01$, partial $\eta^2 = .14$; and a significant phase \times baseline SV \times trait dominance interaction, $F(1, 42) = 5.63$, $p = .02$, partial $\eta^2 = .12$. Additionally, a phase \times trait dominance interaction was also found to be statistically significant, $F(1, 42) = 6.14$, $p = .02$, partial $\eta^2 = .13$, suggesting that trait dominance was significantly associated with reactivity of SV successive stressor exposures independent of baseline SV (see Figure 7). Pearson's correlations showed inverse, but non-significant associations between SV reactivity to Exposures 1 ($r = -.15$, $p = .31$) and 2 ($r = -.11$, $p = .48$), and trait dominance. For those lower in trait dominance, a trend for greater

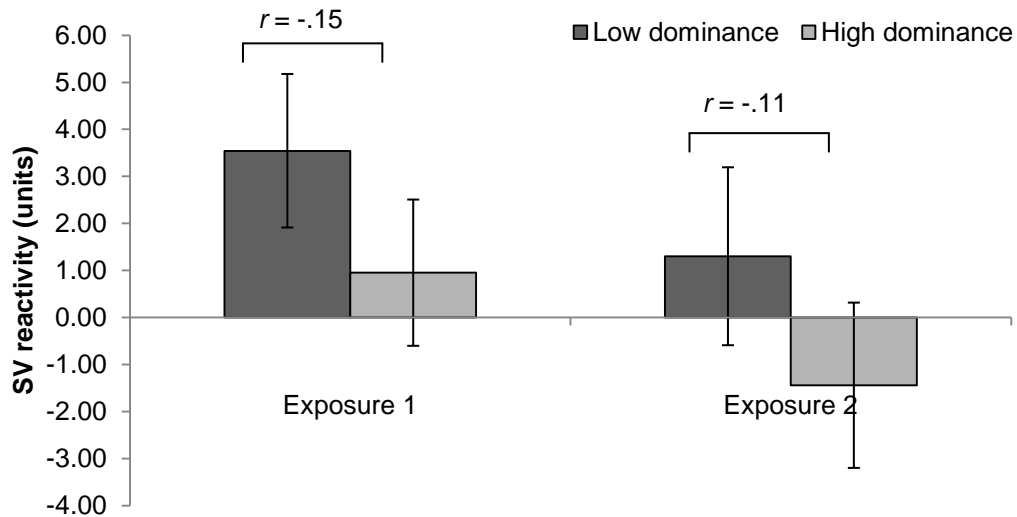


Figure 7. Reactivity of SV across successive stressor exposures with median split low ($n = 21$) and high trait dominance ($n = 25$). Values for r refer to the Pearson's correlation between reactivity to stressor exposure and trait dominance. Error bars denote standard error or the mean.

SV reactivity to Exposures 1 and 2 was observed compared to those higher in dominance, while SV during Exposures 1 and 2 was higher than SV observed at baseline. Higher trait dominant participants showed a profile of SV reactivity that was characterised by higher mean SV during Exposure 1, relative to baseline, while during Exposure 2 mean SV was found to be lower than during the baseline period.

The results of 4×1 ANCOVAs showed significant main effects for phase for HR, $F(1, 44) = 74.39, p < .001$, partial $\eta^2 = .63$; and IBI, $F(1, 44) = 61.56, p < .001$, partial $\eta^2 = .58$, significant at the cubic level (see Figure 8). A phase \times trait dominance interaction, significant at the level of the cubic function, was observed for HR, $F(1, 44) = 5.11, p = .03$, partial $\eta^2 = .10$, while a corresponding phase \times trait dominance interaction for IBI

failed to reach conventional levels of statistical significance, $F(1, 44) = 3.45, p = .07$, partial $\eta^2 = .07$. The pattern of results showed that mean levels of HR were similar for participants higher and lower in trait dominance, but during Exposures 1 and 2, mean HR was lower for those lower in trait dominance. No significant main effects for dominance were found for either HR or IBI. Similar to *post hoc* analyses conducted for TPR, a 2×1 repeated measures ANCOVA was conducted for HR reactivity. Reactivity to Exposures 1 and 2 were the two levels of the within subjects factor, and trait dominance and baseline HR entered as covariates in the analysis. The results revealed no further significant main or interaction effects involving HR reactivity. Inverse, but non-significant relationships between trait dominance and HR reactivity to Exposures 1 ($r = -.18, p = .25$) and 2 ($r = -.13, p = .37$) were found. These results demonstrated that although trait dominance did influence HR when the four phases of the experimental protocol were considered, it was not associated with HR reactivity to the stressors.

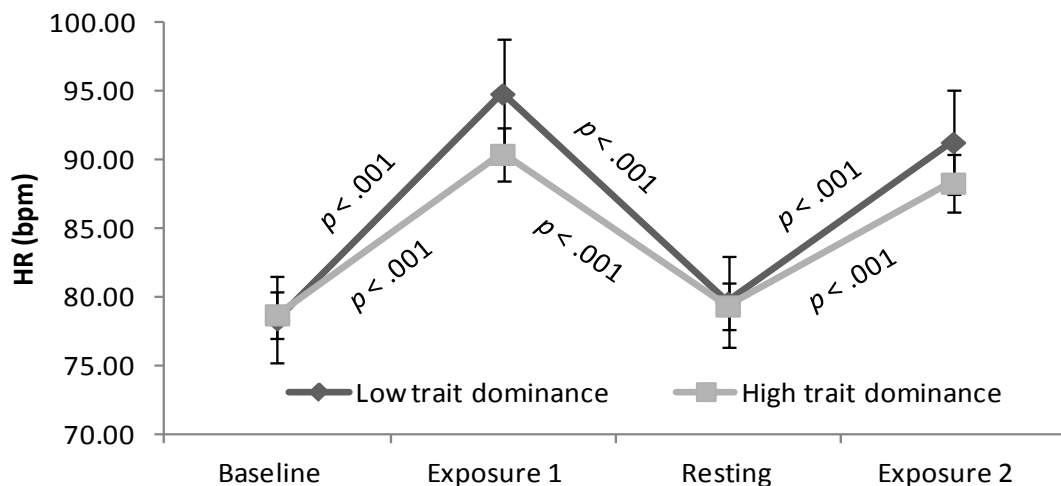


Figure 8. Reactivity of HR across successive stressor exposures with median split low ($n = 21$) and high trait dominance ($n = 25$). Error bars denote standard error of the mean.

Discussion

Over successive exposures to social stress, higher trait dominance was associated with diminished physiological habituation to social stress. Higher trait dominant participants were characterised by an increased vascular stress response (i.e., greater TPR) to Exposure 2, compared to participants lower in trait dominance. Cardiovascular profiles characterised by an initial myocardial response to stress, followed by a habituation of that response on subsequent exposures to similar stress are generally regarded as healthful. In contrast, sustained vascular profiles of cardiovascular responding are associated with CVD risk (Julius, Harburg, Cottington, & Johnson, 1986; Palatini & Julius, 2009). The results are consistent with evidence that points to an association between maladaptive patterns of CVR and trait dominance.

The findings relating to hemodynamic profile showed that while levels of CO were similar for those higher and lower in dominance over successive experimental phases, concurrent values for TPR were found to have changed significantly. As mentioned previously, optimal regulation of the hemodynamic profile is characterised by simultaneous increases in either CO or TPR, with parallel decreases in the other parameter. The current results showed that while levels of CO were held constant for Exposure 2 for those higher and lower in dominance (i.e., there were no significant interactions between dominance and CO), significant changes were found to have occurred for the complimentary parameter of TPR (as demonstrated by a significant dominance by TPR interaction). This implied that the nature of the cardiovascular processes at work during Exposure 2, subject to the strong influence of TPR, reflected the operation of a vascular

profile of hemodynamic functioning. Participants higher and lower in trait dominance displayed increased TPR reactivity to Exposure 2, relative to Exposure 1, but the greatest increase was observed for those higher in trait dominance. During Exposure 1, while CO was greater than at baseline for those lower in dominance, TPR was *lower* than at baseline, suggestive of an active coping orientation (Obrist, 1976). Although TPR increased during Exposure 2 for lower trait dominant participants, a consistently high level of corresponding CO was maintained.

Although CO was not found to be significant, another characteristically myocardial factor; SV, was significantly associated with trait dominance. Reactivity of SV to social stress exposures was found to be inversely associated with trait dominance. An inverse (but non-significant) correlation between SV and trait dominance was observed, such that lower trait dominance was associated with greater SV reactivity to Exposures 1 and 2. During Exposure 2, participants higher in dominance demonstrated a profile of SV that was lower than that which observed at baseline, while corresponding TPR reactivity increased. Significant effects observed for HR appeared to be more general (an inverse association with trait dominance), and could not be reduced further to the levels of effects observed for either TPR or SV.

The findings support evidence that dominance is associated with maladaptive patterns of cardiovascular responding (e.g., Newton, 2009), and shed further light on some of the physiological processes that may underlie increased cardiovascular risk associated with trait dominance. The study shows that repeated social stress exposures can prompt potentially maladaptive cardiovascular responses in women with a greater dominant disposition. The results support the findings of previous studies that employed repeated stressor exposure protocols to other active stressors (e.g., mental arithmetic; Kelsey et al.,

1999; Kelsey et al., 2004), which reported general attenuations in myocardial, but not vascular reactivity with repeated stress exposures.

The innate desire of people higher in trait dominance to exert a controlling influence to the attainment of social hierarchy may be a feature of the increased vascular profile of responding. Higher trait dominant individuals persisted in the exhibition of underlying hemodynamic responses to stress, even when it appears that blood pressure is stable. Interestingly, only those lower in social dominance reported Exposure 2 as significantly less stressful and less difficult, compared to Exposure 1. Some small declines in subjective appraisals of stress and difficulty between the two stressors for those higher in dominance were not statistically significant (see Table 15). Despite no observed significant between subjects effects for trait dominance relating to subjective task appraisals, the fact that lower trait dominant participants found Exposure 2 to be less stressful and less difficult reflects their attenuated physiological functioning during Exposure 2. In contrast, even the task appraisals of those higher in dominance did not demonstrate habituation of perceived stressfulness or difficulty of the tasks, mirroring their physiological responding.

The results of the current study add to the debate surrounding blunting of cardiovascular functioning. As argued by James et al. (2012), examination of hemodynamic processes can reveal added value to the interpretation of findings that may at first appear to reflect blunted physiological function. James et al. (2012) suggested that blunted blood pressure responses could be the result of a particular personality's tendency towards a characteristic vascular or myocardial response pattern (i.e., high-dominant individuals may trend toward more vascular-oriented cardiovascular response patterns). Therefore, as suggested by James et al. (2012), an alternative explanation to cardiovascular

blunting (i.e., less pronounced blood pressure) could be that it is indicative of augmented vascular tendencies. A more vascular reactivity orientation, and the negative health consequences associated with this, could potentially moderate the increased CVD risk that has been attributed to greater dominance (Kaplan et al., 1982; Manuck et al., 1995; Newton, 2009). In contrast, lower trait dominance could be protective against some of the ill-effects of vascular profiles of responding (Whiteman et al., 1997), if reactivity to stress is characterised by an initial myocardial response, and attenuated subsequent responses.

Limitations of the study could be addressed by including an extended protocol with further stressor exposures to demonstrate whether the current trends could be replicated using a protocol with other socially relevant components. It has been suggested previously that stressor tasks that include both a socially evaluative component and a cognitive demand may provide the one of the best laboratory models to examine stress reactivity, and by extension, stress habituation (Dickerson & Kemeny, 2004). Variations of the speech task could be interesting to examine, such as conditions where an independent observer is present during the task, rating participant's speech rather than using a video camera. The findings extend previous research with repeated mental arithmetic tasks (Howard et al., 2011; Hughes et al., 2011) and suggest that individual differences in dominance may moderate profiles of hemodynamic responding.

Chapter 5: Overall Discussion

Integrated Summary of Studies

The possibility that some personality traits render the individual prone to the onset of physical disease has been studied for many years in psychology. However, as befits the biopsychosocial approach, it would appear that the role of such traits is heavily intertwined with that of the social context (including the ways in which that context changes over time). Across three studies, the present thesis examined the impact of personality, social context, and social dominance on specific indices of cardiovascular functioning known to be relevant to disease pathogenesis. Firstly, a self-monitoring protocol examined patterns of cardiovascular functioning in a field setting over an extended period of time. Secondly, cardiovascular functioning during exposure to social or asocial forms of stress was assessed in the laboratory. Finally, hemodynamic parameters of cardiovascular functioning during repeated exposures to social stress were also assessed in a laboratory setting. Overall, the findings confirm that considering personality along with social context succeeds in elucidating a number of likely psychosomatic pathways to disease.

Overview of Study 1

Results from Study 1 suggested that certain affective personality traits, particularly neuroticism and extraversion, were associated with blunted cardiovascular functioning over an extended period of monitoring. While Study 1 was interested in diurnal shifts in blood pressure from daytime to evening periods, it also assessed blood pressure variability

during each day of measurement to examine more continuous diurnal fluctuations during this period. The results of ANCOVAs showed that socially relevant personality traits (i.e., higher neuroticism, extraversion, and conscientiousness) were associated with diminished shift in blood pressure from daytime to evening periods. Regression analyses showed that neuroticism and extraversion were also associated with blunted blood pressure variability.

The novel finding was the observation of an association between personality traits and diminished diurnal cardiovascular shift and variability. Previously, research which has observed blunted patterns of cardiovascular activity has largely been confined to laboratory-type settings (e.g., Hughes et al., 2011; Phillips, 2011; Phillips et al., 2005). Thus, the current findings from Study 1 are among the first to report significant associations between personality and blunted cardiovascular functioning in *field* settings, and the first to observe such *over an extended period of monitoring*. The fact that the affective characteristics involved each had strong social aspects pointed to a potential role for interpersonal factors in the observation of blunted cardiovascular trends.

Overview of Study 2

Study 2 investigated specific contextual factors. Despite the ecological validity advantages of the Study 1 protocol, it needed to be examined if cardiovascular functioning under specific environmental stress was moderated by certain cardio-active personality traits. The impact of a specific interpersonal cardio-active personality trait—namely trait dominance—on cardiovascular functioning during social or asocial laboratory stress was investigated.

The findings from Study 2 showed that only amongst women lower in dominance was differential physiological responding evident between social and asocial stress,

whereby blood pressure was significantly greater during social forms of stress.

Interestingly, for high-dominant women a diminished ability to differentiate between two types of stress, or indeed blunted cardiovascular functioning during social stress was observed. Previously, research has shown that social stress in particular elicits higher physiological stress reactions than asocial forms of stress (al'Absi et al., 1997), and higher dominant women have been shown to exhibit greater CVR to social stress compared to asocial stress (Hughes & Callinan, 2007). Observations from Study 2 suggested blunted cardiovascular during social stress for higher dominant individuals. Consistent with observations of cardiovascular blunting in field settings from Study 1, Study 2 expanded the understanding of factors influencing stress responses, by considering contextual factors and a specific interpersonal personality dimension.

Overview of Study 3

Study 3 attempted to synthesise findings from Studies 1 and 2 which suggested that certain socially relevant traits moderated patterns of diminished cardiovascular functioning over extended periods of time, and during social stress respectively. Thus in order to extend these findings, Study 3 investigated whether socially relevant personality dimensions with strong interpersonal connotations moderated cardiovascular functioning over repeated (social) stressor exposures, and whether variations in the pattern of underlying hemodynamic variables could be observed.

Dominance was found to be associated with attenuated habituation to repeated social stress, such that significant vascular-dominated CVR was observed for women higher in dominance, compared to women lower in dominance during Exposure 2.

Vascular-oriented activity was only observed during repeated exposures to social stress, but not during initial stressor exposure. These findings suggested that maladaptive patterns of stress responding may become particularly pronounced during repeated exposures to social stress for higher dominant individuals. The literature on extensions to the CVR hypothesis would suggest that attenuated habituation to stress could be harmful if such patterns of responding persisted over time in a chronic fashion. Vascular styles of CVR to stress have been suggested to be more adaptive for tasks requiring passive coping efforts, compared to more actively-oriented tasks such as speech tasks used in Study 3. Therefore, Study 3 showed that dominance was associated with vascular patterns of cardiovascular functioning during repeated social stress that are consistent with long-term CVD risk, such that reduced habituation to stress was found for those higher in dominance.

Therefore, despite non-significant differences in indices of blood pressure for individuals higher and lower in trait dominance, differences in the vascular processes were found, consistent with increased vascular activity during Exposure 2, reflecting a potentially maladaptive response to an active stressor, and disrupted habituation to social stress, moderated by trait dominance. The findings implicate a role for dominance in the aetiology of human CVD mechanisms, reflecting findings from animal studies which showed that dominance was positively associated with chronic disease processes (e.g., CAA; Kaplan et al., 1983).

Overall Implications of Findings

Homeostasis and Cardiovascular Functioning: Diminished Responding to Stress

Over the three studies, diminished cardiovascular functioning was found to be associated with socially relevant personality traits. These personality dimensions have been widely observed previously to be associated with CVD morbidity and mortality, with particular evidence for dominance and neuroticism (Helgeson, 1990; Smith & MacKenzie, 2006). The current findings support proposed extensions to the CVR hypothesis, consistent with homeostatic regulation, that exaggerated physiological responding in either direction (i.e., exaggerated *or* diminished functioning) are potentially harmful to good health outcomes (Lovallo, 2011). A recent review paper also highlighted the growing recognition of the importance of considering blunted cardiovascular functioning when examining health outcomes (Phillips et al., in press). Results from the current thesis are consistent with other recent empirical findings that have emerged, which also posit that blunted cardiovascular responding during experimentally-manipulated stress may have implications for ill-health (e.g., Carroll, Phillips, & Lovallo, 2011; Ginty, Gianaros, Derbyshire, Phillips, & Carroll, 2013). As James et al. (2012) suggest, blunted styles of CVR could reflect activation of different hemodynamic mechanisms that result in similar blood pressure patterns being observed for different individuals, but which are underscored by differing hemodynamic pattern as observed by Sherwood et al. (1993).

While blunted physiological functioning has been previously observed to be moderated by Five-Factor affective personality traits such as neuroticism (e.g., positively associated with blunted cortisol, immune and cardiovascular functioning; Phillips et al., 2005), less consideration has been given to other socially relevant dimensions, such as trait

dominance. Like some affective traits, dominance has been associated with exaggerated cardiovascular functioning and subsequent CVD risk (e.g., primate studies have observed associations between significantly advanced CAA and dominance; Kaplan et al., 1983). As such, and consistent with principles of homeostasis, associations between blunted patterns of physiological functioning and dominance too could be harmful for cardiovascular health. Indeed, the current results reflect such an association, showing that not only were affective dimensions of personality (i.e., neuroticism and extraversion) associated with blunted cardiovascular functioning, but also that a specific interpersonal dimension of personality (i.e., dominance), was also associated with diminished cardiovascular functioning during times of social stress.

Interpersonal Personality Traits

By their nature, individual differences in personality traits offer a framework for cognitions and behaviours (Mischel, 2004). The observation of diminished cardiovascular functioning for individuals with greater dispositions towards several personality dimensions may be linked by aspects of interpersonal traits. The current thesis argues that interpersonal features of these traits are important to consider. It has been shown that the social nature of environmental stress may account for the observation of diminished physiological activity for certain interpersonal personality styles. The interpersonal or social aspect of personality and the nature of the stress may account for why pronounced ill-effects of CVD are observed for personality traits with strong interpersonal components (i.e., neuroticism, dominance and general NA).

The experience of socially oriented forms of stress may almost certainly be influenced by processes of affective regulation, either by expression or inhibition of

affective states, or by attributing an affective valence to particular challenges or interactions (John & Gross, 2004). Neuroticism, characterised by greater emotional lability and reactions of greater intensity when faced with stressful experiences (Lahey, 2009), which make social stress particularly physiological stressful, given efforts to exert affective regulation. Generally, social stress for individuals higher in neuroticism may have particular salience (Schneider, Rensch, Lyons, & Riffle, 2012). Although individuals higher in extraversion actively seek social interactions, and are energised by such interactions (Costa & McCrae, 1992), seeking social situations may not always be associated with positive affective experiences, in the same way that neuroticism is not universally a negative affective orientation in every situation. Like extraversion, trait dominance has more overt social consequences than neuroticism, characterised by a desire to establish and maintain social hierarchical structures (Pratto et al., 1994). Social forms of stress have strong implications for any difficulties of emotional regulation, such that an inability to hide negative affective orientations may cause situations to be stressful for any party involved in the social interaction.

Contextual Stress Factors

The current thesis has shown some socially relevant personality traits to be associated with diminished patterns of physiological functioning. The social nature of stress in particular was found to illustrate differential cardiovascular responses effectively, given that interpersonally-oriented personality traits were associated with blunted cardiovascular shift and blood pressure variability during everyday stress (Study 1), with diminished blood pressure during social stress (Study 2), and with attenuated habituation to repeated social stress, characterised by vascular hemodynamic factors (Study 3).

Previously, research has shown socially oriented stress to be a particularly effective in eliciting physiological stress (e.g., using the TSST; Kirschbaum et al., 1993).

Social stresses are likely to comprise many of the most challenging or threatening tensions in modern life (e.g., whether it is public speaking or interactions with colleagues). The degree to which individuals find such situations or interactions tasks stressful is not only determined by environmental or contextual factors, but also by psychological factors. Consistent with homeostasis, the ability to cope effectively with such stress may not require a diminished response to stress, but rather an ability to experience stress as stressful, while in a manner that is proportionate to the stressful nature of the task. Further, an ability to return to normal levels of arousal following the cessation of stress is beneficial for health (Steptoe & Marmot, 2005). In this way homeostasis is maintained by effective regulation of physiological responses. Cardio-active personality traits, known to be associated with CVD risk, and typically associated with *over*-responding to stress, could too be implicated in *under*-responding to stress (as shown in the current thesis), itself potentially harmful to cardiovascular health in the long-term (see Phillips et al., in press).

Stress reactivity may correspond to congruence, or lack thereof, between a personality disposition to strive for success and specific contextual factors (e.g., social stress). Of course, distinctions need to be drawn between situations that are deemed to be stressful and those that are engaging and enjoyable, both of which may be found to elicit greater physiological arousal. Findings from Study 1 did not imbue patterns of physiological functioning with any particular social meaning, as there was a lack of such information gathered. However, Studies 2 and 3 found no differences between subjective ratings of stress tasks between those higher and lower in dominance. Thereby, allowing for a relatively consistent subjective view of the tasks across participants.

An alternative interpretation for the observed blunted effects in the current thesis relate to concordance between social stress and dimensions of cardio-active traits. For example, higher dominant people may be pre-disposed to effectively cope with social stress, such that it doesn't elicit the same degree of stress for them as it does for those lower in dominance. However, previously, research hasn't generally supported such a position, given that greater CVR has been reported for social stress, relative to asocial forms of stress in women (Hughes & Callinan, 2007). Indeed the findings from Study 3 show that rather than blunted cardiovascular functioning indicating a lack of physiological arousal for high trait-dominant individuals, potentially reduced cardiovascular arousal could be explained by different hemodynamic determinants, as seen in Study 3 with increased vascular stress responses on repeated exposure to social stress.

Methodological Limitations

The present thesis clarifies the relationship between some socially oriented personality dimensions and cardiovascular functioning. However, it is hampered by some limitations. The focus on women was primarily due to the small available sample of men, for whom inferential analyses were not viable in the current thesis due to reasons of statistical power. Future research would benefit from the consideration of gender differences in the examination of the association between socially relevant personality traits and cardiovascular functioning. The research could further be enhanced with larger sample sizes for both women and men, particularly for Study 1, where the extended self-monitoring nature of the protocol could benefit from additional statistical power.

The replication of the present findings using a wider, more representative sample would enhance generalisability of the research, as the current thesis used an exclusively

undergraduate sample. While the use of healthy, young participants has benefits for cardiovascular research, such that participants are free from CVD and provide an effective “baseline” to examine the impact of stress on physiological responding, a less homogeneous sample would be interesting to examine. It would be of interest to investigate if associations between socially relevant personality traits and physiological functioning might differ between the current young, healthy sample, and older adults, or those who are experiencing chronic stress (Chida & Hamer, 2008). It would for example be interesting to explore if working adults exhibit differing sensitivity to particular types of (social) stress, due to the nature of their occupational requirements (Evans & Steptoe, 2001). The assessment of hemodynamic patterns of cardiovascular functioning in ambulatory or self-monitoring designs with other adult samples could inform further about the generalisability of some of the observed (laboratory; Study 2 and Study 3) findings to more real life field settings. It has been shown that physiological responses to laboratory stress induction are generally lower than real life stress outside of the laboratory (Zanstra & Johnston, 2011), thus investigations of real life acute (and chronic) stressors could be beneficial to further understanding the association between personality and health.

Documentation of social and contextual factors with further field-based research studies would enhance the ability of future research findings to posit explanations for physiological trends, such as the blunted variability and depressed cardiovascular functioning observed in Study 1. Cardiovascular functioning during ecologically appropriate stress could also be documented, such as during a job interview, or during oral presentations. Corporate environments where sales pitches or presentations are part of everyday occupational duties could be an environment that researchers may wish to target, given the social and interpersonal nature of these types of tasks. Examination of the effects

of physiological functioning and reactivity to acute stress in environments outside of the laboratory could provide further validation of the current research findings.

The possibility that some other psychometric factors could account for the observed relationships between socially relevant personality traits and cardiovascular functioning should be considered. Marshall et al. (1994) described how research in health psychology has at times suffered from the proliferation of some narrowly defined health-relevant measures, with little relationship to each other. However, a strength of Study 1 was the examination of Five-Factor personality dimensions. Marshall et al. (1994) found that the investigation of broad personality domains had benefits for research interested in examining associations between personality and health, as they provide an appropriate framework for many other personality constructs. The use of such a structure in Study one provided a foundation for the investigation of other socially relevant constructs (i.e., trait dominance) subsequently in Study 2 and Study 3.

Problems traditionally associated with self-report measures should too be acknowledged. The current thesis relied on self-report measures to provide estimates of individual personality dispositions. Although only measures previously used in empirical literature and shown to have good reliability were chosen, there is always the possibility that participants may choose to represent themselves in a certain manner. This may be particularly true of trait dominance, a measure with overt behavioural implications. Some participants may have chosen to respond in a manner that either enhanced or suppressed their innate tendencies towards dominance. As such, future research could benefit from a dual approach to the measurement of such socially relevant constructs, with assessment of some behavioural characteristics, as well as psychometric factors.

Overall Conclusions

Socially relevant personality traits were associated with blunted cardiovascular functioning during real life cardiovascular measurement, during social and asocial stress, and during repeated social stress manipulation. Further, associations between trait dominance and CVR during social stress were found to be characterised by vascular stress responses and attenuated habituation to repeated stress exposures. Overall, this thesis aimed to investigate the association between cardio-active personality traits and cardiovascular functioning during stress. Firstly, everyday stress in field settings was considered, with a monitoring of patterns of normative cardiovascular functioning. Subsequent experimental protocols extended the findings to more specific contextual factors (social and asocial stress), as well as investigating cardiovascular adaptation to stress (social). Social stress may be a particularly important avenue for exploration of the longitudinal consequences of stress. In particular, when vascular stress responses are prompted by stress, this may lead to poor long-term cardiovascular health.

Overall, this thesis has established that cardio-active personality traits moderated; (i) blunted physiological functioning in field settings, (ii) cardiovascular functioning during social and asocial stress, and (iii) vascular stress responses and attenuated habituation to repeated social stress. This research adds substantially to investigations of cardio-active personality traits on health, particularly with consideration of blunted cardiovascular functioning, suggesting important implications for long-term cardiovascular well-being.

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Appendix A

Sample Behavioural Diary Entry

Bedtime reading			
Time:			
<i>Location</i>			
<i>Home</i>	<i>Uni</i>	<i>Leisure</i>	
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	
<i>First reading</i>			
SBP:			
DBP:			
HR:			
<i>Second reading</i>			
SBP:			
DBP:			
HR:			
<i>In the last 15 minutes...</i>			
Social contact:			
<i>Low</i>	<i>Med</i>	<i>High</i>	
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	
Stressed?			
<i>Not at all</i>		<i>Extremely</i>	
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Angry?			
<i>Not at all</i>		<i>Extremely</i>	
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
In a positive mood?			
<i>Not at all</i>		<i>Extremely</i>	
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

Appendix B**Words used in Speech Task (Study 2)**

Academic	Private
Beginning	Story
Building	Student
Century	Symptom
Degree	Water
Doctor	Material
Early	Important
Evening	Medicine
Expensive	Music
Funny	Picture
Interview	Public
Lecturer	August
Order	Bottle
Poetry	
Present	

Appendix C

Participant Information Sheet (Study 1)



SCHOOL OF PSYCHOLOGY

INFORMATION SHEET

PERSONALITY, SOCIAL SUPPORT AND BLOOD PRESSURE

Invitation to participate

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

The research

This research study wants to look at how people behave and react in different places and at different times. We want to look at the way that people express their anger at home, at college and during weekdays and weekends. Anger is an emotion that everybody expresses but to different extents depending on the situation that they find themselves in. We want to see if there is a difference in people's blood pressure, depending on how they express their anger and if the people that they have around them, friends or family, affects this.

This study looks at undergraduate students, and this is why you have been asked to participate. Up 99 other students similar to yourself will also be asked to participate in this study.

This study was published on the SONA section of the School of Psychology website. People were invited to contact the researcher if they were interested in taking part in the research.

Taking Part – What it involves

Do I have to take part?

It is up to you to decide whether or not to take part. If you do decide to take part you will be given this *Information Sheet* to keep and be asked to sign a *Consent Form*. If you decide to take

part you are still free to withdraw at any time and without giving a reason. A decision to withdraw at any time, or a decision not to take part, will not affect your rights in any way.

What will happen to me if I take part?

If you decide that you *do* want to participate, you will be asked to come to the laboratory building (Cois Abhann) to meet the researcher. There will be an introductory meeting with other students who are interested in taking part in the study. You will be taught how to take measurements of your blood pressure using a portable blood pressure device. You will also be shown how to record your blood pressure in behaviour diaries that will be given out. This is in order that you have some practice using the blood pressure monitor before you are asked to use it on your own over the course of the study. You will be given the small blood pressure monitor and a booklet of questions to take with you for five days.

You are asked to complete 4 short questions in the accompanying booklet each time that you take a blood pressure reading, as well as writing your blood pressure and heart rate into the booklet. You are asked to record two measures of blood pressure five times daily, at any point during the times outlined in the booklet. For example, at anytime between the time period 16.00 and 17.50. Taking the blood pressure measurements is very quick and easy and should only take approximately 30 seconds per measurement.

You will also be asked to fill out another questionnaire in your own time over the five days that you will be involved with the study.

You are asked to follow this procedure for five consecutive days, from Wednesday until Sunday. After this time is over, you are asked to come back to the laboratory building (Cois Abhann) on Monday to return the blood pressure monitors, the questionnaire and the booklet.

How long will my part in the study last?

You will be asked to be in the study for five days in a row (Wednesday to Sunday). Within this time period, your participation will not require too much disruption to your everyday life. You do not need to do anything other than what you would normally do.

What are the possible benefits in taking part?

Taking part in this study may offer you more of an insight into the ways in which you express your anger. You may also become aware of the natural patterns of your blood pressure.

What are the possible disadvantages and risks of taking part?

There are no risks to participation in this study. Should you become concerned at any stage about your blood pressure being especially high or low, you are advised to contact the Student Health Unit at NUI Galway or to consult with your local GP. Contact information for the Student Health Unit is provided below.

The Student Health Unit is currently located in the National Diagnostic Centre which is the building adjacent to Aras Ui Cathail; the new entrance will be on the left hand side of the building toward the pass machine and not the front door. You can contact the Health Unit on 091 – 492604.

What happens at the end of the study?

After you have returned the blood pressure monitor, questionnaire and booklet, you will be finished with your participation in this study. The researcher will analyse the results and send a general report about the study to you when the results are completed.

What happens if I change my mind during the study?

You are entitled to change your mind about participating in this study at any time without disadvantage or penalty.

Who do I contact for more information or if I have further concerns?

Should you require any further information about this study, please do not hesitate to contact the researcher Eimear Lee at e.lee3@nuigalway.ie

If you have any concerns about this study and wish to contact someone in confidence, you may contact: **The Head, School of Psychology, National University of Ireland, Galway.**

Appendix D

Participant Information Sheet (Study 2)



SCHOOL OF PSYCHOLOGY

INFORMATION SHEET

Title of Project: The effects of personality and different tasks on blood pressure

Invitation to participate

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

The research

This research study wants to look at how people with different types of personalities react to different types of tasks. Anger is an emotion that everybody expresses but to different extents depending on the situation that they find themselves in. We want to see if people's blood pressure is influenced by how they express their anger in different situations. In addition, we want to examine if this anger is influenced by whether that person is surrounded by friends or family.

This study primarily looks at students, and this is why you have been asked to participate. Up to 120 other students, similar to yourself, will also be asked to participate in this study.

Taking Part – What it involves

Do I have to take part?

It is up to you to decide whether or not to take part. If you do decide to take part you will be given this *Information Sheet* to keep and be asked to sign a *Consent Form*. If you decide to take part you are still free to withdraw at any time and without giving a reason. A decision to withdraw at any time, or a decision not to take part, will not affect your rights in any way.

What will happen to me if I take part?

Initially, you will be asked to complete a brief questionnaire about yourself and the way you feel for 10 minutes. Following this, you will be asked to relax for another 10 minutes. You will be given some magazines to read if you would like. During the relaxation time, your blood pressure

will be taken by the researcher, using an automated blood pressure monitor. Your blood pressure will then be taken every two minutes for the remainder of the study.

After the resting period, you will be asked to complete the first of two tasks, each of which lasts 6 minutes (*participants received instructions for either a maths or speech task*).

The researcher will give you a set of cards, each of which has a different word printed on it. You will be asked to talk for as long as you can about the word on the top card. If you get really stuck, and cannot think of anything else to say about the word, you can move on to the next card and repeat the procedure for the remainder of the task duration. While you are talking about the word(s) on the top card, your speech will be recorded using a video camera. We ask that you speak as clearly as possible as would like to evaluate your speech performance later for the way in which you talked about the words, for content and overall clarity.

You will be asked to complete some mental arithmetic problems presented to you on a computer screen. Each question is a subtraction problem, in response to which you are asked to input the correct solution within 10 seconds into the computer.

After the task, there will be a break of 4 minutes, during which time the research will continue measuring your blood pressure. After these 4 minutes, the blood pressure monitor will be removed from your arm.

Finally you will be asked to complete some questionnaires which will ask about different aspects of your personality and the ways in which you react and cope with different situations in your everyday life. The questionnaire also asks about your relationship with family and friends. All responses remain strictly confidential. Once this is completed, this will be the end of your research participation.

How long will my part in the study last?

Your participation in the study is expected to last approximately an hour.

What are the possible benefits in taking part?

Taking part in this study may offer you an insight into the ways in which your personality may influence the way that you respond to different situations. You may learn something about your general level of cardiovascular fitness and the different factors that influence the level of your blood pressure.

What are the possible disadvantages and risks of taking part?

There are no risks to participation in this study. Should you become concerned about your blood pressure being especially high or low, you are advised to contact the Student Health Unit at NUI Galway or to consult with your local GP.

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

For NUI Galway students, the Student Health Unit is located in Aras na MacLéinn, upstairs beside the Students Union. You can call to arrange an appointment with the Student Health Unit on 091 - 492604.

What happens at the end of the study?

The researcher will analyse the results and send a general report about the study to you when the results are completed.

What happens if I change my mind during the study?

A decision to withdraw at any time, or a decision not to take part, will not affect your rights in any way.

Who do I contact for more information or if I have further concerns?

Should you require any further information about this study, please do not hesitate to contact the researcher Eimear Lee at e.lee3@nuigalway.ie

If you have any concerns about this study and wish to contact someone in confidence, you may contact:

The chairperson of the Research Ethics Committee

Research Office,

Science & Engineering Technology Building,

National University of Ireland Galway,

University Road, Galway.

Phone: +353 (0)91 495312

Email: vpresearch@nuigalway.ie

Appendix E

Participant Information Sheet (Study 3)



Title of Project: Effects of personality and different tasks on blood pressure

Invitation to participate

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

The research

This research study wants to look at how people with different types of personalities react to different types of tasks. We want to see if people's blood pressure response to situations differs depending on their personality styles. In addition, we want to examine if this anger is influenced by whether that person is surrounded by friends or family. This study looks at students, and this is why you have been asked to participate. Up to 60 other students similar to yourself will also be asked to participate in this study.

Taking Part – What it involves

Do I have to take part?

It is up to you to decide whether or not to take part. If you do decide to take part you will be given this *Information Sheet* to keep and be asked to sign a *Consent Form*. A decision to withdraw at any time, or a decision not to take part without giving a reason, will not affect your rights in any way.

What will happen to me if I take part?

- The researcher will record some demographic information and your answers to questions about your general health
- Following this, you will be introduced to the Finometer blood pressure apparatus and it will be attached to you.
- You will be asked to relax and given some questionnaires to complete for 10 minutes
- At the end of the 10 minutes, the continuous blood pressure monitoring will start. At this time, you will be asked to leave the questionnaires aside and to relax. A magazine will be provided for you.
- After 10 minutes spent relaxing and reading the magazine, you will be asked to complete the first speech task.

- For the speech tasks during this experiment, the researcher will present you with a hypothetical scenario, and you will be asked to give a speech about this scenario.
- The scenarios presented are randomly generated scenarios, and any resemblances to any previous experiences of yours are completely coincidental.
- The researcher will briefly describe this scenario to you, and you will be asked to then spend 2 minutes preparing a speech in response to the scenario.
- When you are thinking about the scenario, you should try to imagine how you might feel, and how you might react if you were faced with this same situation.
- After the 2 minutes of preparation time, the researcher will ask you to begin to deliver your 2 minute speech.
- Your speech will be video-recorded and evaluated later by the research team who will rate your speech based on content and delivery.
- It will be up to you to decide what you want to say during your speech and how you want to interpret the situation presented to you. However, we would ask that you try to continue speaking for the full two minutes. The researcher will tell you when the two minutes are up.
- After the 4 minutes of the task, you will be asked to rest for 3 minutes, by going back to reading the magazine provided.
- After this 3 minute rest, you will be asked to do the speech task again with a different scenario. Again you will be given 2 minutes to prepare for a 2 minute speech.
- Finally, you will finally be asked to complete some more questionnaires which you will be asked to return to the researcher. These questions will ask about different aspects of your personality, and the ways in which you react and cope with different situations in your everyday life. The questionnaire also asks about your relationship with family and friends. As with all aspects of the study, all responses remain strictly confidential.
- Once this questionnaire is completed, this will be the end of your research participation.

How long will my part in the study last?

Your participation in the study is expected to last approximately an hour.

What are the possible benefits in taking part?

Taking part in this study may offer you an insight into the ways in which your personality may influence the way that you respond to different situations. You may learn something about your general level of cardiovascular fitness and the different factors that influence the level of your blood pressure.

What are the possible disadvantages and risks of taking part?

There are no risks to participation in this study. Should you become concerned about your blood pressure being especially high or low, you are advised to contact the Student Health Unit at NUI Galway or to consult with your local GP. Contact information for the Student Health Unit is provided below. This study includes some questions that measure your feelings and well-being now and in the recent past. You might find while you are answering them that you would like to talk to someone about some of the issues raised. We will be happy to recommend someone to you. The Student Health Unit is located in Aras na MacLeinn, upstairs beside Students Union. You can call to arrange an appointment with the Student Health Unit on 091 - 492604.

What happens at the end of the study?

The researcher will analyse the results and send a general report about the study to you when the results are completed.

What happens if I change my mind during the study?

A decision to withdraw at any time, or a decision not to take part, will not affect your rights in any way.

Who do I contact for more information or if I have further concerns?

Should you require any further information about this study, please do not hesitate to contact the researcher Eimear Lee at e.lee3@nuigalway.ie

If you have any concerns about this study and wish to contact someone in confidence, you may contact:

**The chairperson of the Research Ethics Committee,
Research Office,
Science & Engineering Technology Building,
National University of Ireland Galway,
University Road, Galway.
Phone: +353 (0)91 495312
Email: vpresearch@nuigalway.ie**

Appendix F

Participant Consent Form



School of Psychology

Consent Form

Participant Identification No.: _____

Title of Project: Personality and cardiovascular functioning during stress

Name of Researcher: Eimear Lee

Please initial box

1. I confirm that I have read the information sheet for the above study and have had the opportunity to ask questions.
2. I am satisfied that I understand the information provided and have had enough time to consider the information.
3. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my legal rights being affected.
4. I agree to take part in the above study.

Name of Participant	Date	Signature
<u>Eimear Lee</u> Researcher	Date	Signature