

**CONSTITUTIONAL AND BEHAVIOURAL CORRELATES OF INDIVIDUAL
DIFFERENCES IN BIOLOGICAL STRESS REACTIVITY**

by

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ABSTRACT

This thesis examined potential corollaries of individual differences in cardiovascular and/or cortisol reactions to acute psychological stress, with specific focus upon personality and behavioural characteristics. The varying magnitudes of cardiovascular stress reactivity have been implicated in a variety of adverse health and behavioural outcomes including cardiovascular disease development. Chapter 2 reported that a negative constellation of the Big 5 personality traits, higher neuroticism and lower openness and agreeableness, was associated with blunted physiological reactivity. Chapter 3 demonstrated that, in comparison to individuals without Type D personality, Type Ds had greater physiological reactivity during social stress, but lower reactivity when exposed to largely asocial stress. Both these studies also reported dissociation between subjective and physiological stress responses. Chapter 4 reported that individuals with problematic Internet use and/or excessive alcohol consumption did not differ in physiological stress reactivity in comparison to non-dependent controls. Finally, Chapter 5 demonstrated that, compared to exaggerated cardiovascular stress responders, blunted reactors had greater levels of behavioural impulsivity. Overall, the research reported provides evidence that there is dissociation between affective and physiological stress responses, the context in which the stressful situation is experienced is important, and finally, blunted reactivity appears to be related to adverse outcomes which are stable rather than transient, suggesting that it may be a peripheral marker of dysfunction in the brain systems that support motivated behaviour.

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This thesis is comprised of four original empirical papers which form the following chapters:

- Chapter 2
Bibbey, A., Carroll, D., Roseboom, T. J., Phillips, A. C., & de Rooij, S. R. (2013). Personality and physiological reactions to acute psychological stress. *International Journal of Psychophysiology*, 90, 28-36.
- Chapter 3
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- Chapter 4
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- Chapter 5
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Oral Presentations

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- **Bibbey, A.,** Phillips, A. C., Ginty, A. T., & Carroll, D. (2014). We are watching you: Type D personality is associated with exaggerated cardiovascular and cortisol stress reactivity but only under high social evaluative threat. *11th Annual Psychology, Health & Medicine Conference*, University of Limerick, Ireland, May 2014.
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* Awarded conference press release as the society's programme committee recognised it as being especially newsworthy and significant in the scientific and general community.

Poster Presentations

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- **Bibbey, A.**, Carroll, D., Roseboom, T. J., Phillips, A. C., & de Rooij, S. R. (2013) How stressed do you get? A negative personality constellation is associated with higher feelings of stress but lower blood pressure, heart rate, and hormone responses to stressful experiences. *Annual University of Birmingham Graduate School Research Poster Conference*. Birmingham, June 2013**
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During the period of postgraduate study at the University of Birmingham, the following papers were also published:

- Carroll, D., **Bibbey, A.**, Roseboom, T. J., Phillips, A. C. Ginty, A. T., & de Rooij, S. R. (2012). Forced expiratory volume is associated with cardiovascular and cortisol reactions to acute psychological stress. *Psychophysiology*, 49, 866-72.
- Carroll, D., Phillips, A. C., Der, G., Hunt, K., **Bibbey, A.**, Benzeval, M., & Ginty, A. T. (2013). Low forced expiratory volume is associated with blunted cardiac reactions to acute psychological stress in a community sample of middle-aged men and women. *International Journal of Psychophysiology*, 90, 17-20.

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LIST OF ABBREVIATIONS

AUDIT	Alcohol Use Disorders Identification Test
BART	Balloon Analogue Risk Task
BFI	Big Five Inventory
BMI	body mass index
BP	blood pressure
DBP	diastolic blood pressure
DS14	Type D Scale-14
HADS	Hospital Anxiety and Depression Scale
HPA	hypothalamic-pituitary-adrenal
HR	heart rate
IAT	Internet Addiction Test
ISEI-92	International Socio-Economic Index-92
NA	negative affectivity
PASAT	Paced Auditory Serial Arithmetic Test
PIUS	Pathological Internet Use Scale
SAM	sympathetic-adrenal-medullary
SBP	systolic blood pressure
SES	socio-economic status
SI	social inhibition
SPQ	Shorter PROMIS Questionnaire
SSRT	stop signal reaction time

CHAPTER ONE

GENERAL INTRODUCTION

It is now common knowledge that individuals vary markedly in the magnitude of their physiological reactions to acute psychological stress (Carroll, 1992). Psychological stress can be defined as a mental experience which exceeds an individual's coping resources, therefore leading to physiological and emotional reactions which disturb the body's stable state (Selye, 1955). Acute stress, in this context is generally operationalized as short-term (minutes) exposure within the laboratory to stressful tasks or situations such as mental arithmetic e.g., serial subtraction, giving a speech in front of an audience, or a time-pressured computer task. Consistent individual differences in physiological stress reactivity have commonly been observed in the sympathetic-adrenal-medullary (SAM) system, as indexed by cardiovascular parameters such as increased blood pressure and heart rate (HR) (Lovallo, 1997), and in the hypothalamic-pituitary-adrenal (HPA) axis, as indexed by the stress steroid hormone cortisol (Kudielka & Wust, 2010). Indeed, this individual variation has been observed in response to a wide variety of acute psychological stress tasks within a laboratory setting, e.g. mental arithmetic, speech, Stroop, and mirror tracing tasks (Chida & Hamer, 2008). Further, these individual differences in stress reactivity appear to be relatively stable over time (Cohen et al., 2000; Ginty et al., 2013; Hassellund et al., 2010) and to have a discernible genetic basis (Wu et al., 2010).

Health Implications

It is also clear that individual differences in stress reactivity have implications for health (Carroll et al., 2009a; Chida & Steptoe, 2010). With regards to SAM system activation, the reactivity hypothesis postulated that large magnitude cardiovascular reactions in response to acute psychological stress would be associated with and contribute to cardiovascular disease (Chida & Steptoe, 2010; Obrist, 1981; Taylor et al., 2003). In line with this, large scale

cross-sectional and prospective studies have reported that individuals who show exaggerated cardiovascular stress responses are at increased risk of developing cardiovascular disease including hypertension (Carroll et al., 2012b; Carroll et al., 2003; Everson et al., 1996; Newman et al., 1999; Treiber et al., 1997), systemic atherosclerosis and increased carotid intima wall thickness (Everson et al., 1997; Lynch et al., 1998; Matthews et al., 1998), left ventricular hypertrophy (Georgiades et al., 1997; Kapuku et al., 1999; Murdison et al., 1998), and coronary artery calcification (Matthews et al., 2006). Further, it has recently been found that large cardiovascular reactions to stress are associated with an increased likelihood of dying from cardiovascular disease (Carroll et al., 2012a). Given that there is minimal increase in metabolic demand during acute psychological stress exposure, large cardiovascular responses can therefore be considered to be metabolically excessive (Balanos et al., 2010; Carroll et al., 2009b); it is this uncoupling of cardiovascular activity from contemporary metabolic activity and its consequences that has been proposed to underlie these adverse cardiovascular health outcomes (Obrist, 1981; Turner & Carroll, 1985). Exaggerated cardiovascular reactivity has also been associated with adverse health outcomes through its effects upon increased lipoprotein concentration (Howes et al., 1997), insulin resistance (Waldstein & Burns, 2003) and inflammation (Danesh et al., 2004). Additionally, exaggerated cortisol reactivity to stress has been associated with coronary artery calcification (Hamer et al., 2010) and increased hypertension (Hamer & Steptoe, 2012), leading to increased cardiovascular disease risk (Girod & Brotman, 2004).

Blunted reactions to stress

An implicit assumption of the reactivity hypothesis is that low or blunted cardiovascular stress reactions might be viewed as benign or even protective (Carroll et al., 2009a). Recent

evidence, however, suggests that both blunted cardiovascular and cortisol reactivity may be associated with a range of adverse health and behavioural outcomes (Carroll et al., 2009a; Lovallo, 2011; Phillips et al., 2013). Largely the same epidemiological studies that implicate exaggerated cardiovascular reactions in cardiovascular pathology have also shown that blunted cardiovascular and/or cortisol reactivity are associated with depression (Carroll et al., 2007; de Rooij et al., 2010; Phillips et al., 2011a), obesity (Carroll et al., 2008; Phillips, 2011; Phillips et al., 2012), and poor self-reported health (de Rooij & Roseboom, 2010; Phillips et al., 2009a). Further, prospective analyses revealed that blunted reactivity was also associated with staying or becoming depressed, obese, and in poor health over a period of five years (Carroll et al., 2008; Phillips et al., 2009a; Phillips et al., 2011b). The findings cited emerged from two large independent epidemiological studies, the West-of-Scotland Twenty-07 Study and the Dutch Famine Birth Cohort study, and, accordingly, indicate the consistency of the effects across national borders, different samples, varying protocols, and different stress task exposure. The West-of-Scotland study utilised three separate age cohorts of adults from Glasgow and the surrounding area, and subjected them to a three minute Paced Auditory Serial Arithmetic Test (PASAT) (Gronwall, 1977), which consists of hearing single digit numbers played from a cassette tape and adding them together, while retaining the last number heard in each instance to add it to the next number played by the tape. In contrast, the Dutch Famine Birth Cohort study sampled Dutch adults born between 1943 to 1947, and utilised three stress tasks, which were a Stroop, mirror drawing, and a speech test. They also measured salivary cortisol, as well as systolic blood pressure (SBP), diastolic blood pressure (DBP), and HR. Finally, researchers from other independent laboratories, again using a range of stress tasks, have reported associations between low reactivity and both depression (Brindle et al., 2013; Salomon et al., 2009; Schwerdtfeger &

Rosenkaimer, 2011; York et al., 2007) and obesity (Miller et al., 2013; Singh & Shen, 2013).

Addictions and blunted responses to stress

In addition to the negative health outcomes, blunted stress reactions have also been observed to be a characteristic of individuals with addictive behaviours. For instance, in comparison to non-smokers, habitual smokers have shown diminished cardiovascular (Evans et al., 2012; Ginty et al., 2014; Girdler et al., 1997; Roy et al., 1994) and cortisol (al'Absi et al., 2003; Ginty et al., 2014; Kirschbaum et al., 1993; Rohleder & Kirschbaum, 2006) stress reactions to range of acute psychological stress tasks. Further, smokers wearing nicotine patches still display diminished reactivity, indicating the blunted responses cannot be attributed to temporary abstinence (Girdler et al., 1997). Blunted reactivity may also have prognostic value as diminished responses have been found to predict relapse in smokers who had quit (al'Absi et al., 2005). Similarly, those dependent on alcohol (Bernardy et al., 1996; Dai et al., 2007; Errico et al., 1993; Lovallo et al., 2000; Panknin et al., 2002; Sinha et al., 2011) and/or other non-prescription drugs (Lovallo et al., 2000; Panknin et al., 2002; van Leeuwen et al., 2011) have also been found to show blunted biological responses to a range of stress tasks. In addition, the offspring of such dependent individuals who have not yet become dependent also appear to display low physiological stress reactivity (Sorocco et al., 2006), suggesting that diminished reactivity may actually precede addictive behaviour rather than being a consequence of substance abuse. In further support, there is evidence that blunted reactivity would appear to be a feature of individuals with behavioural as well as substance dependencies: i.e. exercise dependence (Heaney et al., 2011), disordered eating (Ginty et al., 2012a; Koo-Loeb et al., 1998), and gambling addictions (Paris et al., 2010). Thus blunted

stress reactivity may be a general feature of dependency, irrespective of the precise nature of the dependency.

Motivational dysregulation and stress reactivity

It has been argued that the link between these diverse correlates of blunted stress reactivity is a dysfunction in motivation. Indeed, low cardiovascular and cortisol reactions to acute stress have been considered a peripheral marker for central motivational dysregulation (Carroll et al., 2009a; Carroll et al., 2011; Lovallo, 2011), i.e. dysregulation of the neural systems that support motivation and goal-directed behaviour. Evidence in support comes from functional Magnetic Resonance Imaging studies. For example, individuals characterised by blunted biological stress responses have been found to show diminished activation in both the posterior and anterior cingulate cortex, and in the amygdala during stress exposure (Gianaros et al., 2005; Ginty et al., 2013). These areas are implicated in motivational processes and goal-directed behaviour as well as autonomic nervous system regulation (Bush et al., 2000; Hagemann et al., 2003; Lovallo, 2005). Indeed, individuals with some of the characteristics shown to be related to blunted cardiovascular reactivity have been shown to have dysregulation or blunted activation of these same brain areas. For example, obese individuals have been shown to have low sympathetic nervous system activity *per se*, as indexed by muscle sympathetic nerve activity (Spraul et al., 1993; Tentolouris et al., 2006) and to have hypoactivation of the prefrontal and limbic regions during emotional tasks (Stice et al., 2008). Further, those with depression show blunted responses to reward in the frontal and subcortical limbic regions (Holsen et al., 2011). Performing well on a range of tests of cognitive ability also relies of motivated behaviour, and these same brain areas discussed above, and it has recently been shown that individuals with poorer cognitive ability on

reaction time and numerical and verbal reasoning also display blunted reactivity to acute stress (Ginty et al., 2011a, 2011b; Ginty et al., 2012b). Overall it would appear that both exaggerated and blunted physiological stress reactions can have adverse consequences; whereas exaggerated reactivity may contribute to cardiovascular disease development, blunted reactivity may be a marker of motivational dysregulation and, thus, signal increased risk of developing a whole host of maladaptive health and behavioural outcomes.

Overview of the present thesis

The research reported in this thesis is concerned, then, with cardiovascular and cortisol reactions to stress. More specifically, it seeks to elaborate further the behavioural corollaries of individual differences in the magnitude of physiological stress reactivity: first, by considering the association between reactivity and personality in Chapters 2 and 3; second, by further examining addictions/dependent behaviour in Chapter 4; and, finally, by looking at the proximal behavioural characteristics of extreme high and low reactors in Chapter 5. The sections below provide summary justification for the four empirical studies that comprise the main body of the thesis. These are necessarily brief, as each of the subsequent chapters include a more protracted and detailed introduction to each topic.

Personality and stress reactivity (Study 1, Chapter 2)

Personality refers to individual differences in the characteristics of a person which subsequently influence their cognitions, motivations, emotions, and behaviours (McCrae & Costa, 1987). Indeed individuals' personality traits have been shown to remain consistent for periods of over 45 years (Soldz & Vaillant, 1999; Terracciano et al., 2006). It is expected that variations in stress reactivity would map onto individual differences in

personality traits, as personality has been shown to affect stress perception (Connor-Smith & Flachsbart, 2007) and, according to cognitive theories of stress, should therefore affect physiological stress reactions (Carver & Connor-Smith, 2010; Dickerson & Kemeny, 2004; Lazarus, 1996). However, previous research examining the relationship between personality and physiological stress reactivity has yielded mixed dividends and no clear consensus. Accordingly, the first aim of the present research was to provide a broader examination of whether physiological stress reactivity is associated with key personality dimensions.

The Big Five Trait taxonomy identifies five broad personality dimensions; neuroticism, agreeableness, openness, extraversion, and conscientiousness (McCrae & Costa, 1987). Neuroticism has been associated with blunted HR (Hughes et al., 2011), DBP (Jonassaint et al., 2009) and cortisol stress reactivity (Kirschbaum et al., 1992; Oswald et al., 2006; Phillips et al., 2005), with a meta-analysis also concluding that neuroticism, anxiety, and negative affect tended to be linked to attenuated cardiovascular stress reactions (Chida & Hamer, 2008). However, some studies have reported no association between neuroticism and cardiovascular (Hutchinson & Ruiz, 2011; Kirkcaldy, 1984; Schneider, 2004; Stemmler & Meinhardt, 1990; Williams et al., 2009b), and cortisol (Kirschbaum et al., 1992; Kirschbaum et al., 1995; Schommer et al., 1999; Verschoor & Markus, 2011; Wirtz et al., 2007) reactions to stress. Less attention has been paid to the other Big Five dimensions in this context. For agreeableness, null findings have been reported for cardiovascular (Williams et al., 2009b) and cortisol (Oswald et al., 2006; Wirtz et al., 2007) stress reactivity. Openness has been reported to show a positive (Oswald et al., 2006), negative (Wirtz et al., 2007), and no (Schoofs et al., 2008) association with cortisol stress reactions. In the one study examining the relationship between cardiovascular stress reactivity and openness, a negative association emerged for blood pressure reactivity (Williams et al., 2009b). Null outcomes have also

been observed between extraversion and both cardiovascular (Kirkcaldy, 1984; Vassend & Knardahl, 2005; Williams et al., 2009b) and cortisol stress reactivity (Kirschbaum et al., 1992; Schommer et al., 1999; Wirtz et al., 2007). Finally, null findings also characterise the few studies that have examined conscientiousness and cardiovascular (Williams et al., 2009b) and cortisol (Oswald et al., 2006; Wirtz et al., 2007) reactions to stress.

It is important to note that previous research on personality and biological stress reactivity suffers from a number of limitations. Among them are small sample sizes (Kirschbaum et al., 1995; Oswald et al., 2006; Wirtz et al., 2007) leading to low power to detect associations, the predominance of young student samples (Kirschbaum et al., 1992; Verschoor & Markus, 2011; Williams et al., 2009b), a restricted range of trait scores (Schommer et al., 1999; Wirtz et al., 2007), dichotomised trait variables (Kirkcaldy, 1984) which leads to potential misclassification of personality trait, and the failure to adjust statistically for a range of possible confounding variables such as sex, age, medication use and health behaviours (Williams et al., 2009b; Wirtz et al., 2007). It is likely that such limitations have contributed to the inconsistent findings that characterise previous research in the field. The first study reported in this thesis re-examines, in a large middle aged cohort, the relationship between the Big Five personality traits and both cardiovascular and cortisol reactions to a comprehensive stress task protocol. The analyses also incorporated statistical adjustment for a number of potential confounders. Self-reported stress task impact was also measured, to possibly shed light on the psychological mechanisms underlying any link between personality and stress reactivity.

Type D personality and stress reactivity (Study 2, Chapter 3)

More recently, research has focussed on a particular personality trait known as Type D personality. Type D or ‘distressed’ personality is characterised by the propensity to experience high levels of negative emotion (negative affectivity: NA) and the tendency to inhibit emotions in social situations (social inhibition: SI) (Denollet, 2005). Type D personality has been associated with a range of negative cardiovascular disease outcomes (Mols & Denollet, 2010), although the underlying mechanisms behind such associations remain unclear. Given that exaggerated cardiovascular and cortisol stress reactions have been associated with similar adverse cardiovascular outcomes (Carroll et al., 2012a; Carroll et al., 2012b; Carroll et al., 2003; Everson et al., 1997; Girod & Brotman, 2004; Hamer et al., 2010; Hamer & Steptoe, 2012; Kapuku et al., 1999; Matthews et al., 2006; Murdison et al., 1998), excessive stress reactivity may be an underlying pathway. Previous studies examining Type D personality and physiological stress reactivity in healthy populations have reported inconsistent findings. A study conducted by Habra and colleagues reported the SI component to be related to exaggerated blood pressure reactivity, with both the NA and SI components of Type D related to a greater cortisol stress response (Habra et al., 2003). In comparison to non-Type D students, Type D individuals have shown exaggerated cardiac output responses to a stress task, with no differences in blood pressure or HR (Williams et al., 2009a). Conversely, lower HR and cardiac output reactivity has been reported in Type D females, again with no differences in SBP or DBP responses (Howard et al., 2011).

A potential reason for the mixed findings for Type D personality may be the social evaluation components of the stress tasks employed. This is pertinent because the social inhibition component of Type D refers to the inhibition of emotions in social situations. Emotional inhibition has been shown to relate to exaggerated cardiovascular (Gross & Levenson, 1997) and cortisol (Lam et al., 2009) stress reactions. Indeed, there is evidence

that social evaluation *per se* increases cardiovascular (Allen et al., 1991; Smith et al., 1997; Wright et al., 1995) and cortisol (Dickerson et al., 2004) reactivity. It is possible that stress tasks with high levels of social evaluation may prove particularly provocative for those with a Type D personality.

Although the effects of social evaluation *per se* on stress reactivity have been studied, to date nobody has examined the potential differential effects according to Type D status. Given that it is possible that variations in the social nature of the stress tasks used in previous studies helps explain the mixed results, i.e. it would appear that when a social task has been used, Type D individuals displayed exaggerated physiological stress reactions (Habra et al., 2003; Williams et al., 2009a), whereas exposure to an asocial task appears to result in attenuated reactivity (Howard et al., 2011), the second aim of the present thesis was to directly compare cardiovascular and cortisol stress reactions of Type D and non-Type D individuals in two settings varying in social evaluation characteristics.

Stress reactivity and substance and non-substance abuse (Study 3, Chapter 4)

As previously mentioned, blunted stress reactions have been observed to be characteristic of individuals with substance addictions such as tobacco (al'Absi et al., 2003; Evans et al., 2012; Ginty et al., 2014; Girdler et al., 1997; Kirschbaum et al., 1993; Rohleder & Kirschbaum, 2006; Roy et al., 1994), alcohol (Bernardy et al., 1996; Dai et al., 2007; Errico et al., 1993; Lovallo et al., 2000; Panknin et al., 2002; Sinha et al., 2011), and/or other non-prescription drugs (Lovallo et al., 2000; Panknin et al., 2002; van Leeuwen et al., 2011). The fact that the offspring of these dependent individuals also display relatively attenuated physiological stress reactions (Moss et al., 1999; Sorocco et al., 2006), suggests that diminished reactivity precedes the development of addictive behaviour and is not a

consequence of protracted substance abuse. In further support, there is evidence that blunted reactivity would appear to be a feature of individuals with behavioural as well as substance dependencies. For example, those with problematic exercise behaviour and who meet the criteria for exercise dependence have been shown to display diminished cardiovascular and cortisol reactions to a mental arithmetic stress task, the PASAT, in comparison to healthy controls (Heaney et al., 2011). Additionally, in response to a similar stress protocol, individuals with disordered eating behaviour also displayed blunted cardiac output, HR, stroke volume, and cortisol stress reactions (Ginty et al., 2012a). This largely replicates and extends a previous finding that diagnosed bulimics were characterised by attenuated rather than excessive cardiovascular stress reactivity (Koo-Loeb et al., 1998). Finally, individuals with gambling addictions have been shown to display attenuated biological reactions (Paris et al., 2010). Thus blunted stress reactivity may be a general feature of dependency, irrespective of the precise nature of the dependency. It would certainly seem sensible to explore the limits of this proposition, especially given that behavioural dependencies have received little attention within this area.

With the increasing use of the Internet, there is a growing concern that individuals are showing signs of dependent behaviour (Widyanto & Griffiths, 2006). Indeed, problematic and excessive Internet use has many characteristics in common with substance addiction: disturbed psychological functioning, inability or unwillingness to reduce use with potential relapse, and increased tolerance (Griffiths, 1996; Young, 1996). Further, there is at least preliminary evidence linking Internet and alcohol misuse (Ko et al., 2008). High alcohol intake *per se* during adolescence has been linked to a range of long-term adverse outcomes such as increased anti-social behaviour (Swahn et al., 2004), mental health disorders (Marmorstein, 2009), relationship problems (Odgers et al., 2008), and life-long impaired

cognitive function (Brown et al., 2008; Ehlers & Criado, 2010; Hanson et al., 2011). Thus, both excessive Internet and alcohol use appear to be associated with a host of maladaptive outcomes. In addition, they would appear to be a common co-morbidity and previous research indicates that comorbid conditions are related to poorer health outcomes than single disorders (Vogeli et al., 2007). Similarly, there is at least preliminary evidence that those with multiple substance dependencies, i.e. alcohol and non-prescription drugs, are more likely to show blunted cardiovascular and cortisol responses to an acute stress task than those with a single dependency, i.e. alcohol or non-prescription drugs (Bernardy et al., 1996; Errico et al., 1993; Panknin et al., 2002).

However, to date, problematic Internet behaviour has received no attention in the context of stress reactivity, nor has the comorbidity of behavioural and substance dependence, e.g. problematic Internet behaviour and alcohol use. Further, few studies have examined alcohol use and stress reactions in student samples. Accordingly, the third aim of this thesis was to compare cardiovascular and cortisol stress responses of individuals with comorbid Internet and alcohol dependence, Internet dependence *per se*, alcohol dependence *per se*, and individuals showing no signs of dependence.

The proximal behavioural characteristics of those showing blunted stress reactivity (Study 4, Chapter 5)

Given that blunted stress reactivity may signify central motivational dysregulation (Carroll et al., 2009a; Carroll et al., 2011b; Lovallo, 2011), leading to the range of adverse outcomes, such as addiction, it is likely that blunted stress reactors will be characterised by behaviours considered to be prevalent in those susceptible to such adverse outcomes. Candidate behavioural characteristics include high impulsivity and risk taking, and lack of persistence,

as all three have been implicated in the substance and behavioural addictions associated with blunted reactivity (Lovallo, 2013). In support, individuals with greater impulsivity (Horn et al., 2003; Inuggi et al., 2014) and risk taking (Hare et al., 2008) have been shown to display deficiencies in their frontolimbic areas of the brain: the same areas proposed to lead to the reduced biological stress reactivity (Lovallo, 2005, 2013). However, there has been little research into the proximal behavioural characteristics of blunted and exaggerated stress reactors. If blunted stress responders displayed proximal behavioural characteristics which may signify increased risk for addiction and negative health outcomes, this would provide further support for the central motivational dysregulation model of blunted stress reactivity, and suggest attenuated reactivity may have some prognostic value. Consequently, the fourth aim of the current thesis was to examine impulsivity, risk taking, and persistence in individuals previously identified as extreme exaggerated or extreme blunted cardiovascular stress reactors.

Impulsivity is a multi-dimensional construct which includes the rapid unplanned responses to stimuli and difficulty in refraining from actions (de Wit, 2009), and thus conceptually implicated in addictive behaviour (Brewer & Potenza, 2008). There is strong empirical support; those with addictions to alcohol (Aragues et al., 2011), tobacco (Balevich et al., 2013; Mitchell, 1999), other non-prescription substances (Ersche et al., 2010; Perry & Carroll, 2008), gambling (Leeman & Potenza, 2012), and exercise (Freimuth et al., 2011), as well as those suffering from eating disorders/obesity (Schag et al., 2013; Zalar et al., 2011) and all appear to exhibit elevated levels of impulsivity. Regarding the association between impulsivity and cardiovascular/cortisol stress reactivity, few studies have examined this issue in non-clinical populations and the results are mixed. Whereas two studies reported high impulsivity was related to low cardiac reactivity (Allen et al., 2009; Munoz &

Anastassiou-Hadjicharalambous, 2011), one study reported an association in the opposite direction (Diller et al., 2011), with no clear association also observed (Mathias & Stanford, 2003). A study of pre-adolescent children reported that those with high impulsivity had diminished cardiac responses to mental arithmetic stress (Bennett et al., 2014). This was also one of the few previous studies to include behavioural as well as self-report measures of impulsivity.

Risk taking would also appear to be elevated in individuals with addictions to alcohol (Lawrence et al., 2009), tobacco (Cavalca et al., 2013), other non-prescription substances (Schutter et al., 2011), gambling (Lawrence et al., 2009) and exercise (Freimuth et al., 2011), as well as in those with eating disorders (Fischer & le Grange, 2007). In the context of physiological stress reactivity, risk taking has attracted limited attention. The one published study to date of cardiovascular stress reactions reported no association between blood pressure reactivity and risk taking in adolescent boys (Liang et al., 1995). Again, this study relied on self-reports of risk taking and did not include behavioural measures. Persistence and perseverance are key components in achieving daily life goals, and are related to greater academic and career progress (Andersson & Bergman, 2011) and the ability to refrain from unhealthy behaviours (Steinberg et al., 2012). Given that blunted stress reactivity may signal central motivational dysregulation, it is possible that attenuated reactivity may be associated with lower levels of persistence. Indeed, individuals with attenuated cardiovascular and cortisol stress responses have been shown to be more likely to relapse during smoking cessation, possibly reflecting reduced persistence (al'Absi et al., 2005). To the best of my knowledge, however, no studies have examined the association between biological stress reactivity and persistence.

Given that impulsivity, risk taking and persistence are associated with the unhealthy outcomes linked to attenuated biological stress reactivity, the present thesis examined their relationship with cardiovascular stress reactivity. However, instead of the correlational approach favoured in the few previous studies, we adopted a case-control design, stress testing a substantial sample and then selecting sub-samples of unambiguously exaggerated and blunted cardiac reactors. The selected individuals then completed a series of behavioural tasks designed to measure impulsivity, risk taking, and persistence. It was considered that such an approach would afford a more powerful test of the hypothesis that blunted stress reactors would be characterised by greater impulsivity and risk taking, and reduced persistence.

Hypotheses informing the four empirical studies

With regard to Study 1, it was hypothesized that neuroticism would be negatively associated with both cortisol and cardiovascular stress reactivity. Given the paucity and inconsistency of previous research, there were no clear expectations regarding the size and the direction of any association between stress reactivity and the other personality traits that make up the Big Five. The hypothesis that guided Study 2 was that increased social evaluation would enhance cardiovascular and cortisol stress reactivity in general, although this would be particularly evident among individuals with Type D personality. It was expected that within the asocial condition, Type D individuals would show inclinations towards attenuated reactivity. In Study 3 it was hypothesised that those with symptoms of dependence, whether to alcohol or the Internet, would show blunted stress reactivity, and this blunted reactivity would be a particular feature of those with comorbid Internet and alcohol problems. Finally,

Study 4 proceeded from the hypothesis that those with blunted stress reactions would be characterised by increased impulsivity and risk taking, and decreased persistence.

My contribution to the studies reported in the thesis

The present thesis comprises of three experimental studies (Chapters 3 to 5, Studies 2 to 4) and one secondary analysis of epidemiological data (Chapter 2) to yield four empirical chapters, each written as an article for scientific publication. The idea to examine the Big Five personality traits and stress reactivity, Chapter 2 (Study 1), was mine. DC and AP secured the data from Amsterdam and I completed the statistical analyses and initial drafts of the paper with input from DC and AP. S deR (Amsterdam) and TR (Amsterdam) provided feedback upon the drafts, with all authors contributing to the final manuscript. The idea to assess Type D personality and stress reactivity (Chapter 3) was mine, and I collected all of the data. I undertook the statistical analyses and produced the draft manuscript with input from DC and AP. AG then provided final comments on the manuscript. The idea for the study reported in Chapter 4 was created by all authors, especially AG, and I collected all of the data. AP provided input during the statistical analyses, with DC and AG providing input and feedback on the manuscripts which I wrote. Finally, the idea for the behavioural study reported in Chapter 5 was created by DC, with all authors providing feedback. I chose the behavioural tests and established the study protocol. RB and I collected the initial data regarding the screening of the participants; I then completed the data collection of the performance on the behavioural tasks and conducted the statistical analyses. I drafted the manuscripts with input from DC and AP, whilst AG and RB provided feedback on the final manuscript.

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CHAPTER TWO

**PERSONALITY AND PHYSIOLOGICAL REACTIONS TO ACUTE
PSYCHOLOGICAL STRESS**

ABSTRACT

Stable personality traits have long been presumed to have biological substrates, although the evidence relating personality to biological stress reactivity is inconclusive. The present study examined, in a large middle aged cohort (N = 352), the relationship between key personality traits and both cortisol and cardiovascular reactions to acute psychological stress. Salivary cortisol and cardiovascular activity were measured at rest and in response to a psychological stress protocol comprising 5-minutes each of a Stroop task, mirror tracing, and a speech task. Participants subsequently completed the Big Five Inventory to assess neuroticism, agreeableness, openness to experience, extraversion, and conscientiousness. Those with higher neuroticism scores exhibited smaller cortisol and cardiovascular stress reactions, whereas participants who were less agreeable and less open had smaller cortisol and cardiac reactions to stress. These associations remained statistically significant following adjustment for a range of potential confounding variables. Thus, a negative personality disposition would appear to be linked to diminished stress reactivity. These findings further support a growing body of evidence which suggests that blunted stress reactivity may be maladaptive.

Keywords: Acute stress; Agreeableness; Cardiovascular activity; Cortisol; Neuroticism; Openness.

INTRODUCTION

It is now commonly known that individuals vary markedly in the way their body reacts to stressful and challenging environmental exposures (Carroll, 1992). Consistent individual differences in stress reactivity have been observed in the hypothalamic-pituitary-adrenal (HPA) axis, as indexed by cortisol, and in the sympathetic-adrenal-medullary (SAM) system, as indexed by cardiovascular activity (Lovallo, 1997). It is also clear that these individual differences have implications for health and behaviour (Carroll et al., 2009b; Chida & Steptoe, 2010). For example, greater cortisol and cardiovascular reactivity to acute stress has been associated with increased risk of cardiovascular disease (Carroll et al., 2011a; Chida & Steptoe, 2010; Hamer et al., 2010; Treiber et al., 2003). In contrast, however, recent evidence also implicates diminished cortisol and cardiovascular reactions in a range of adverse health and behavioural outcomes, such as smoking, alcohol dependence, obesity, and depression (Carroll et al., 2009a; Carroll et al., 2011b). What is less certain is whether individual differences in biological stress reactivity reflect consistent variations in basic human personality traits. Early research on Type A behaviour and stress reactivity proved inconclusive (Carroll, 1992), although there is evidence that one component of the Type A behaviour, hostility, is associated with greater cortisol and cardiovascular reactions to stress, e.g., Smith et al. (2004). However, this is not a completely consistent finding (Carroll et al., 1997). There is, nevertheless, compelling theoretical reasons for expecting the variations in stress reactivity to map on to individual differences in personality traits; if personality, as has been proposed, affects stress perception (Connor-Smith & Flachsbart, 2007), cognitive stress theories and previous research would suggest it should also affect biological stress reactions (Carver & Connor-Smith, 2010; Dickerson & Kemeny, 2004; Lazarus, 1996).

Recent research on personality has frequently turned to the Big Five trait taxonomy which identifies five broad personality dimensions; neuroticism, agreeableness, openness, extraversion, and conscientiousness (McCrae & Costa, 1987). Each trait has demonstrated high stability for up to 45 year intervals (Soldz & Vaillant, 1999; Terracciano et al., 2006). Neuroticism refers to a tendency toward negative affectivity and an inclination toward impulsive behaviour. Agreeableness connotes a willingness to be helpful and trusting, and to possess a pro-social orientation towards others. Individuals high in openness to experience tend to be imaginative, creative, attentive to inner feelings, prefer variety, and are flexible in their thinking. Extraversion refers to the inclination to be energetic, sociable, and assertive, and conscientiousness encompasses organization, self-discipline, and determination (McCrae & John, 1992).

Higher neuroticism has been associated with lower cortisol stress reactivity (Kirschbaum et al., 1993; Oswald et al., 2006; Phillips et al., 2005) although it should be conceded that numerous studies reported no association between neuroticism and cortisol reactions to a range of stress exposures (Kirschbaum et al., 1992; Kirschbaum et al., 1995; Schommer et al., 1999; Verschoor & Markus, 2011; Wirtz et al., 2007). Nevertheless, in support of the evidence suggesting higher neuroticism is linked to blunted physiological stress responses, a meta-analysis of 71 laboratory studies concluded that neuroticism, anxiety, and negative affect tended to be linked to attenuated cardiovascular stress reactivity (Chida & Hamer, 2008), with more recent studies reporting blunted heart rate (HR) (Hughes et al., 2011) and diastolic blood pressure (DBP) stress responses (Jonassaint et al., 2009) in highly neurotic individuals. Again, however, a number of studies have also reported no association between neuroticism and cardiovascular stress reactions (Hutchinson & Ruiz, 2011; Kirkcaldy, 1984; Schneider, 2004; Stemmler & Meinhardt, 1990; Williams et al., 2009). It is important to

note that such null findings between neuroticism and physiological stress reactivity may well have been due to low power (Kirschbaum et al., 1995), restricted range (Schommer et al., 1999; Wirtz et al., 2007) or arbitrary categorization of neuroticism scores (Hutchinson & Ruiz, 2011), examination of anticipatory rather than stress reactions (Verschoor & Markus, 2011), insufficiently provocative stress exposures (Kirkcaldy, 1984; Williams et al., 2009), or a host of other methodological issues (Stemmler & Meinhardt, 1990). Therefore, due to these methodological flaws, evidence may well suggest that high levels of neuroticism are related to blunted biological stress reactivity.

The other personality traits of the Big Five have received far less attention in this context. For agreeableness, null findings have been reported for cortisol (Oswald et al., 2006; Wirtz et al., 2007) and cardiovascular (Williams et al., 2009) stress reactivity. Openness has been reported to show a positive (Oswald et al., 2006), negative (Wirtz et al., 2007), and no (Schoofs et al., 2008) association with cortisol stress reactivity. In the one study we know of examining the relationship between cardiovascular stress reactivity and openness, a negative association emerged for blood pressure reactivity (Williams et al., 2009). Research on extraversion has generally yielded null outcomes for both cortisol (Kirschbaum et al., 1992; Schommer et al., 1999; Wirtz et al., 2007) and cardiovascular (Kirkcaldy, 1984; Vassend & Knardahl, 2005; Williams et al., 2009) stress reactivity. Finally, null findings also characterise the few studies that have examined conscientiousness and cortisol (Oswald et al., 2006; Wirtz et al., 2007) and cardiovascular (Williams et al., 2009) reactions to stress.

Previous research on personality and biological stress reactivity suffers from a number of limitations. Among them are small sample sizes (Kirschbaum et al., 1995; Oswald et al., 2006; Wirtz et al., 2007), the predominance of young student samples (Kirschbaum et al.,

1992; Verschoor & Markus, 2011; Williams et al., 2009), restricted range of trait scores (Schommer et al., 1999; Wirtz et al., 2007), dichotomised trait variables (Kirkcaldy, 1984), and the failure to adjust statistically for a range of possible confounding variables (Williams et al., 2009; Wirtz et al., 2007). The aim of the present study was to re-examine, in a large middle aged cohort, the relationship between the Big Five personality traits and both cortisol and cardiovascular reactions to a comprehensive stress protocol comprising three acute psychological stress tasks. The nature of the study allowed us to adjust for a number of potential confounders. In addition, examination of the self-reported stress task impact will also extend the previous literature, and possibly shed light on the psychological mechanisms linking the personality traits to physiological stress reactions. It was hypothesized that neuroticism would be negatively associated with both cortisol and cardiovascular stress reactivity. Given the paucity and inconsistency of previous research, we had no clear expectations regarding the size and the direction of any association between stress reactivity and the other personality traits that make up the Big Five.

METHODS

Participants

Participants were selected from the Dutch Famine Birth Cohort, which comprises 2414 men and women who were born in Amsterdam, the Netherlands, between November 1943 and February 1947. The selection procedures and subsequent loss to follow up have been described in detail elsewhere (Painter et al., 2005). The Dutch Famine Birth Cohort Study was designed to investigate the potential consequences of prenatal exposure to famine on health in later life. It might, therefore, be suggested that population characteristics may

hamper generalization of the present analyses. However, this is very unlikely as health effects pertain in the group of people exposed to famine in early gestation (Roseboom et al., 2006). Only 8% of the total study sample and 9.5% (N=37) of the present sample were exposed to famine in early gestation. Nevertheless, we chose to exclude them to prevent any possible contamination. Seven hundred and twenty five of the sample attended a clinic assessment between 2002 and 2004, during which time cortisol and cardiovascular reactions to acute psychological stress were measured. In 2008-2009, participants were asked to complete a questionnaire package which included the Big Five Inventory (BFI) (Denissen et al., 2008). Six hundred and one participants returned the questionnaires. The effective sample size for the present analyses, i.e., cohort members who undertook stress testing and completed the Big Five, was 352 (190 women). The mean (SD) temporal lag between the questionnaire assessment and the stress session was 5.5 (0.6) years. Both arms of study were approved by the local Medical Ethics Committee and carried out in accordance with the Declaration of Helsinki. All participants gave written informed consent. The sociodemographic, anthropometric, health behaviour and medication status characteristics of the effective sample are shown in Table 2.1.

Table 2.1. Characteristics of final sample at clinic assessment (N= 352)

Variable	M/N	SD/%
Age (years)	58.23	0.95
Sex (female)	190	52.5
Socio-economic status (ISEI-92)	51.29	13.64
Body mass index (kg/m ²)	28.76	4.90
Alcohol (units of per week)	9.83	15.01
Current smoker	74	20.5
Anti-hypertensive medication	96	26.5
Anti-depressant or anxiolytic	45	12.4

Psychological stress testing

The stress protocol has been described in detail elsewhere (de Rooij et al., 2006) and is illustrated in Figure 2.1. In short, the stress testing was performed in the afternoon, about an hour after participants had eaten a light lunch. The protocol started with a 20-minute baseline period, followed by three 5-minute psychological stress tests (Stroop, mirror tracing, and speech); the inter-task interval was 6 minutes. The final task, the speech, was followed by a 30-min recovery period. The Stroop test was a single trial computerized colour-word conflict challenge. After a short introduction, participants were allowed to practice until they fully understood the requirements of the task. Errors and exceeding the response time limit of 5 seconds triggered a short auditory beep. In mirror tracing, a star had to be traced that could only be seen in mirror image (Lafayette Instruments Corp, Lafayette, IN, USA). Every divergence from the line of the star induced a short beep. In the speech

test, participants were told to imagine being accused of pick-pocketing and instructed to give a 3-minute defence of the accusation, which was videotaped. They were given 2 minutes to prepare their defence. Participants were told that the number of repetitions, eloquence, and persuasiveness of their performance would be marked by a team of communication-experts and psychologists.

Saliva samples were collected using Salivettes (Sarstedt, Rommelsdorf, Germany) at seven time points during the protocol: at 5 and 20 minutes in the baseline period; at 6 min after completion of the Stroop; at 6 min after completion of the mirror-drawing test; and at 10, 20 and 30 min after completion of the speech test. Salivary cortisol concentrations were measured using a time-resolved immunofluorescent assay (DELFI) (Wood et al., 1997). The assay had a lower detection limit of 0.4 nmol/l and an inter-assay variance of 9-11% and an intra-assay variance of less than 10%. Continuous blood pressure (BP) and HR recordings were made using a Finometer or a Portapres Model-2 (Finapres Medical Systems, Amsterdam, Netherlands). There were no differences in measurements between the two instruments. We designated four 5-minute periods as the key measurement periods: baseline (15 min into the baseline period), and the 5 minutes each of Stroop, mirror-tracing, and speech (including preparation time) exposure. We calculated mean systolic blood pressure (SBP), DBP, and HR for each measurement period. A questionnaire was completed after each of the stress tasks which included questions on stress task commitment, perceived stressfulness, stress task difficulty, and perceptions of control. Answers for each item were given on a 7-point scale with scores ranging from 1 (not at all) to 7 (very much); thus, overall item scores could range from 3 to 21.

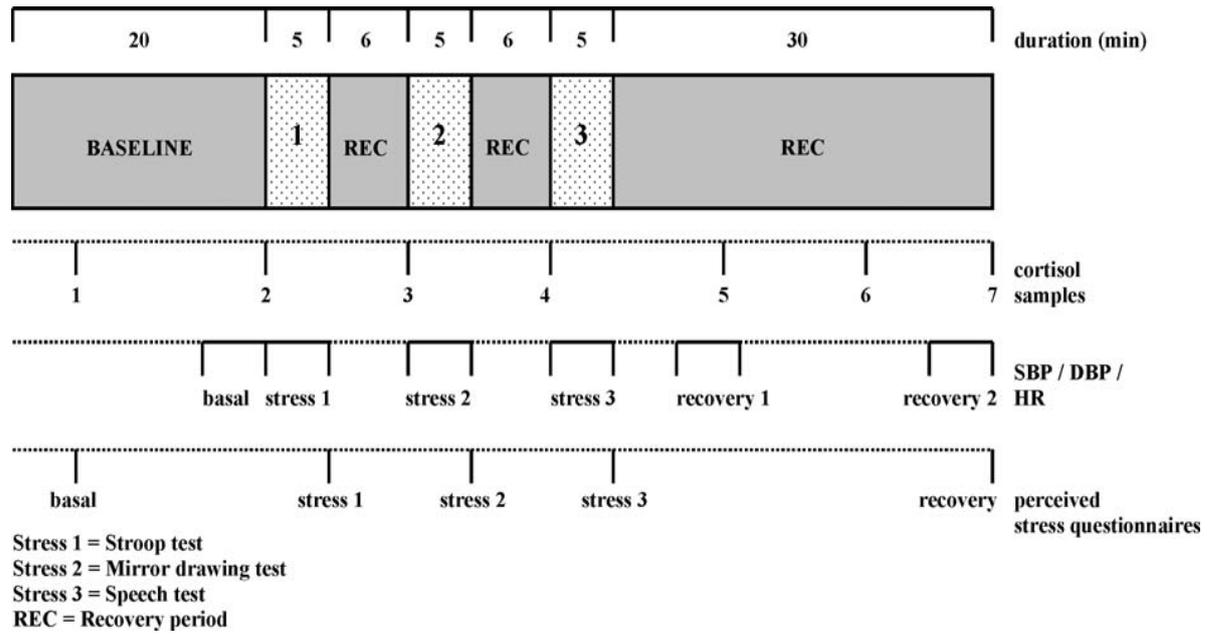


Figure 2.1. Schematic representation of the psychological stress protocol

Personality

We used a Dutch validated translation of the BFI (Denissen et al., 2008). The BFI is based on an established and well-validated model of personality. The inventory comprised five scales with a variable number of items to be self-rated on a 5-point Likert scale ranging from strongly disagree to strongly agree (John & Srivastava, 1999). The five scales include: neuroticism (8 items, scores range from 8 to 40), extraversion (8 items, scores range from 8 to 40), agreeableness (9 items, scores range from 9 to 45), conscientiousness (9 items, scores range from 9 to 45) and openness to experience (10 items, scores range from 10 to 50). In the present study, Cronbach's α was 0.86 for neuroticism, 0.80 for extraversion, 0.75 for agreeableness, 0.77 for conscientiousness and 0.81 for openness, indicating good internal consistency for all scales.

Other study parameters

In the 2002-2004 study, height was measured twice using a fixed or portable stadiometer and weight twice using Seca and portable Tefal scales. Body Mass Index (BMI) in kg/m^2 was computed from the averages of the two height and weight measurements. A standardized interview was performed in which information was obtained about socio-economic status (SES), lifestyle, and use of medication. Alcohol consumption was represented as the number of units of alcohol consumed per week and smoking behaviour was characterised as current, ex-, and never smoker. Medication status (antihypertensive, antidepressant, and anxiolytic use) was determined by questioning followed by medication check. Two binary variables were derived: taking versus not taking anti-hypertensive medication, taking versus not taking either anti-depressant or anti-anxiolytic medication. We defined current SES according to International Socio-Economic Index (ISEI)-92, which is based on the participant's or their partner's occupation, whichever status is higher (Bakker & Sieben, 1997). Measured values on the ISEI-92 scale ranged from 16 (low status, e.g. a cleaning person) to 87 (high status, e.g. a lawyer).

Statistical analyses

Baseline cortisol was calculated as the mean of the two cortisol concentration measurements during the baseline period. The cortisol concentrations of the fifth and sixth samples, i.e., those taken 10 minutes and 20 minutes following stress exposure, were used to determine cortisol stress reactivity. These were the peak cortisol values in the present study and these time lags also characterise peak response in other stress research (for review see: Dickerson & Kemeny, 2004). These values were averaged and baseline cortisol subtracted to yield the stress reactivity values. Baseline cardiovascular activity was the average of values recorded

in the 5-minute period 15 minutes into the baseline. SBP, DBP, and HR measures were averaged across each of the three stress tasks, and the mean of these three averages then determined. Cardiovascular stress reactivity was defined in each case as the difference between the overall stress mean value and baseline for each of the three cardiovascular variables. We calculated a total perceived task commitment score by adding the scores on the questionnaires performed after each stress task.

We applied linear regression analyses to analyse the associations between personality traits and stress reactivity. For all five personality traits, we first tested an unadjusted model, followed by a model adjusting for sex, age and SES and, finally, a model which additionally adjusted for alcohol consumption, smoking, BMI, use of anti-hypertensive medication, use of anti-depressant or anxiolytic medication, perceived commitment to the stress task, and baseline cortisol/cardiovascular activity (as appropriate). In the adjusted models, a hierarchical approach was followed in which the covariates were entered at Step 1 and the personality variables individually at Step 2. Correlation analyses were run to determine the associations between each of the personality traits as well as their association with self-reported stress task impact variables.

RESULTS

Physiological stress reactions

Summary baseline and stress task physiological data are presented in Table 2.2. Cortisol concentrations increased following stress task exposure, $F(1,266) = 41.64, p < .001, \eta^2 = .135$. The stress tasks also reliably perturbed SBP, $F(1,351) = 302.48, p < .001, \eta^2 = .788$, DBP, $F(1,351) = 314.62, p < .001, \eta^2 = .473$, and HR, $F(1,350) = 110.56, p < .001, \eta^2 = .240$.

Table 2.2. Mean (SD) cortisol and cardiovascular activity at baseline and following, and in the case of cardiovascular during, stress task exposure

	Cortisol nmol/L	HR bpm	SBP mmHg	DBP mmHg
Baseline	4.70 (3.22)	73.63 (10.11)	128.36 (19.80)	67.09 (11.78)
Stress	6.32 (4.48)	81.87 (13.65)	160.31 (26.20)	80.19 (11.98)

*All stress measures significantly different from baseline for each variable, $p < .001$

Big five personality traits

The summary personality trait statistics are presented in Table 2.3. Neuroticism was negatively associated with the other four traits which were all positively related to each other. Although highly significant, the correlation coefficients were generally small to moderate in size.

Table 2.3. Association among the Big Five personality traits

Characteristic		Extraversion	Agreeableness	Conscientiousness	Openness
	Mean (SD)				
Neuroticism	20.33 (5.75)	-.45	-.33	-.25	-.36
Extraversion	28.11 (5.11)		.29	.39	.44
Agreeableness	33.54 (4.79)			.26	.19
Conscientiousness	33.99 (4.76)				.26
Openness	34.62 (5.88)				

*All correlations between personality traits were statistically significant, $p < .001$

Personality and self-reported stress task impact

The summary statistics for stress task impact are reported in Table 2.4. The stress tasks were regarded as moderately stressful and difficult, and participants did not perceive themselves as being particularly in control. However, they regarded themselves as being strongly committed to the tasks. Those scoring high in neuroticism perceived the stress tasks as more stressful, $r = .26, p < .001$, and more difficult, $r = .32, p < .001$. They also regarded themselves as less in control, $r = -.30, p < .001$. There was no association between neuroticism and stress task commitment. Those scoring high on openness and extraversion, in contrast, found the tasks less stressful, $r = -.14, p = .01$ and $r = -.16, p = .002$ respectively, and less difficult, $r = -.23, p < .001$ and $r = -.20, p < .001$ respectively. They also felt in greater control, $r = .24, p < .001$ and $r = .16, p = .002$ respectively. Neither openness nor extraversion was associated with commitment to the stress task. No other associations of note emerged from these analyses.

Table 2.4. Self-reported impact of the stress task

Variable	Mean (SD)
Commitment	14.80 (4.10)
Stressfulness	11.09 (4.05)
Difficulty	14.38 (3.54)
Control	10.20 (3.61)

Answers for each item given on a 7-point scale with scores ranging from 1 (not at all) to 7 (very much); thus, overall item scores could range from 3 to 21

Personality and cortisol stress reactivity

In the unadjusted regression, neuroticism and cortisol reactivity were negatively associated whereas agreeableness and openness were positively associated with cortisol reactivity. No associations with cortisol reactivity emerged for extraversion and conscientiousness. The regression models for neuroticism, agreeableness, and openness are summarized in Table 2.5. In regression models adjusting for sex, age, and SES, high neuroticism, low agreeableness, and low openness continued to be related to low cortisol reactivity. In models that additionally adjusted for alcohol consumption, smoking, BMI, use of anti-hypertensive medication, use of anti-depressant or anxiolytic medication, perceived commitment to the stress task, and baseline cortisol activity, similar outcomes emerged. In addition the associations emerging from these fully adjusted models are illustrated in Figure 2.2 where tertiles of personality trait are plotted against cortisol stress reactions. Figure 2.2 indicates that whereas high neuroticism was associated with low reactivity, low agreeableness and openness were associated with low reactivity. In the fully adjusted models, sex and baseline

cortisol were also associated with cortisol reactivity; women and those with higher cortisol baseline concentrations exhibited lower cortisol stress reactions.

Table 2.5. Regression models for neuroticism, agreeableness, conscientiousness and cortisol reactivity

	β	t	p	R ²
Neuroticism and cortisol reactivity				
Unadjusted Model	-.19	3.20	.002	.035
Adjusted 1	-.14	2.30	.02	.018
Adjusted 2	-.14	2.27	.02	.016
Agreeableness and cortisol reactivity				
Unadjusted Model	.15	2.49	.01	.021
Adjusted 1	.16	2.75	.006	.025
Adjusted 2	.16	2.73	.007	.023
Openness and cortisol reactivity				
Unadjusted Model	.19	3.31	.001	.037
Adjusted 1	.15	2.51	.01	.021
Adjusted 2	.13	2.21	.03	.015

Adjusted model 1= adjustment for sex, age and SES; Adjusted model 2= additional adjustment for alcohol consumption, smoking, BMI, use of anti-hypertensive medication, use of anti-depressant or anxiolytic medication, perceived commitment to the stress task, and baseline cortisol.

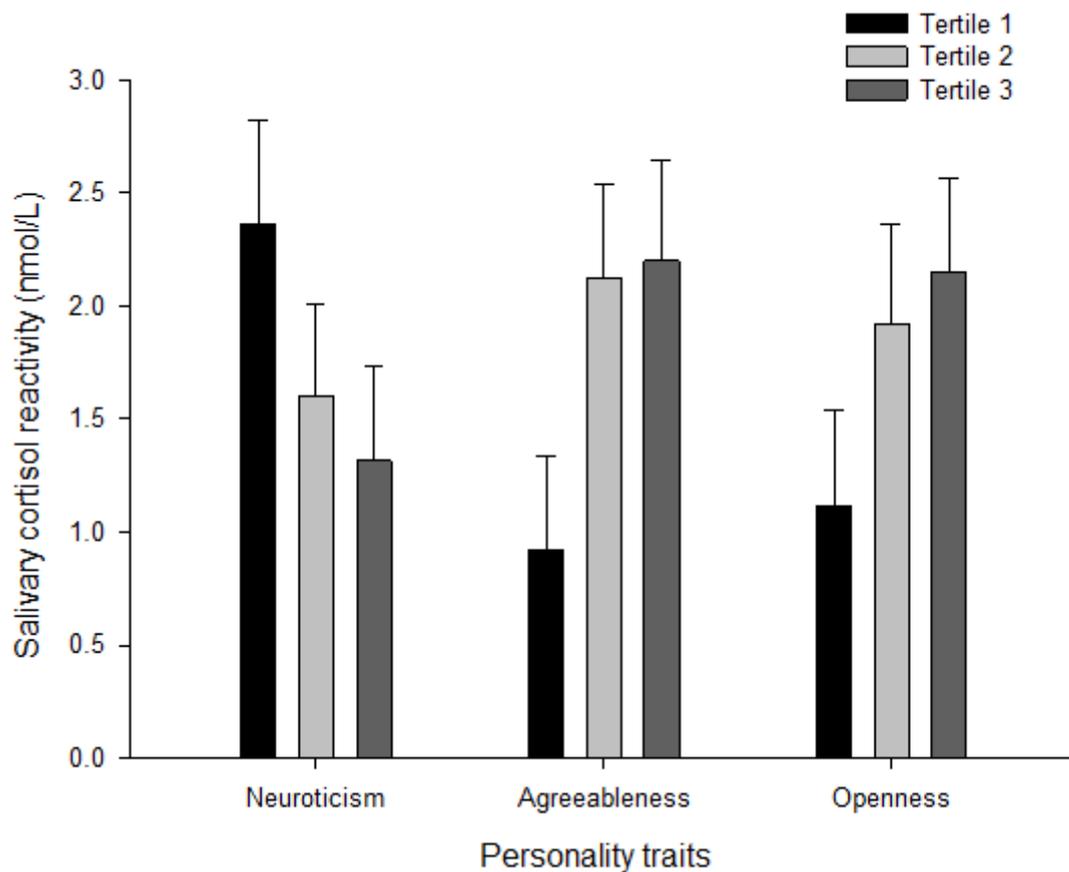


Figure 2.2. Mean (SE) salivary cortisol reactivity by tertiles of neuroticism, agreeableness and openness

Personality and cardiovascular stress reactivity

As was the case with cortisol reactivity, HR reactivity was consistently associated with neuroticism, agreeableness and openness. The unadjusted regression model revealed that high neuroticism, low agreeableness, and low openness were related to low HR reactivity. The associations for neuroticism, agreeableness, and openness withstood adjustment for sex, age and SES, and for the additional variables included in the fully adjusted model. These outcomes are summarized in Table 2.6. Again, conscientiousness and extraversion were not related to HR reactivity. Figure 2.3 illustrates these associations from the fully adjusted

regression models, by plotting tertiles of neuroticism, agreeableness and openness against HR reactivity.

Table 2.6. Regression models for neuroticism, agreeableness, conscientiousness and HR reactivity

	β	t	p	R ²
Neuroticism and HR reactivity				
Unadjusted Model	-.20	3.89	<.001	.042
Adjusted 1	-.19	3.55	<.001	.034
Adjusted 2	-.15	2.65	.008	.019
Agreeableness and HR reactivity				
Unadjusted Model	.12	2.21	.03	.014
Adjusted 1	.12	2.15	.03	.013
Adjusted 2	.11	2.05	.04	.012
Openness and HR reactivity				
Unadjusted Model	.17	3.15	.002	.028
Adjusted 1	.14	2.63	.01	.020
Adjusted 2	.11	2.00	.05	.011

HR= heart rate; Adjusted model 1= adjustment for sex, age and SES; Adjusted model 2 = additional adjustment for alcohol consumption, smoking, BMI, use of anti-hypertensive medication, use of anti-depressant or anxiolytic medication, perceived commitment to the stress task, and baseline HR.

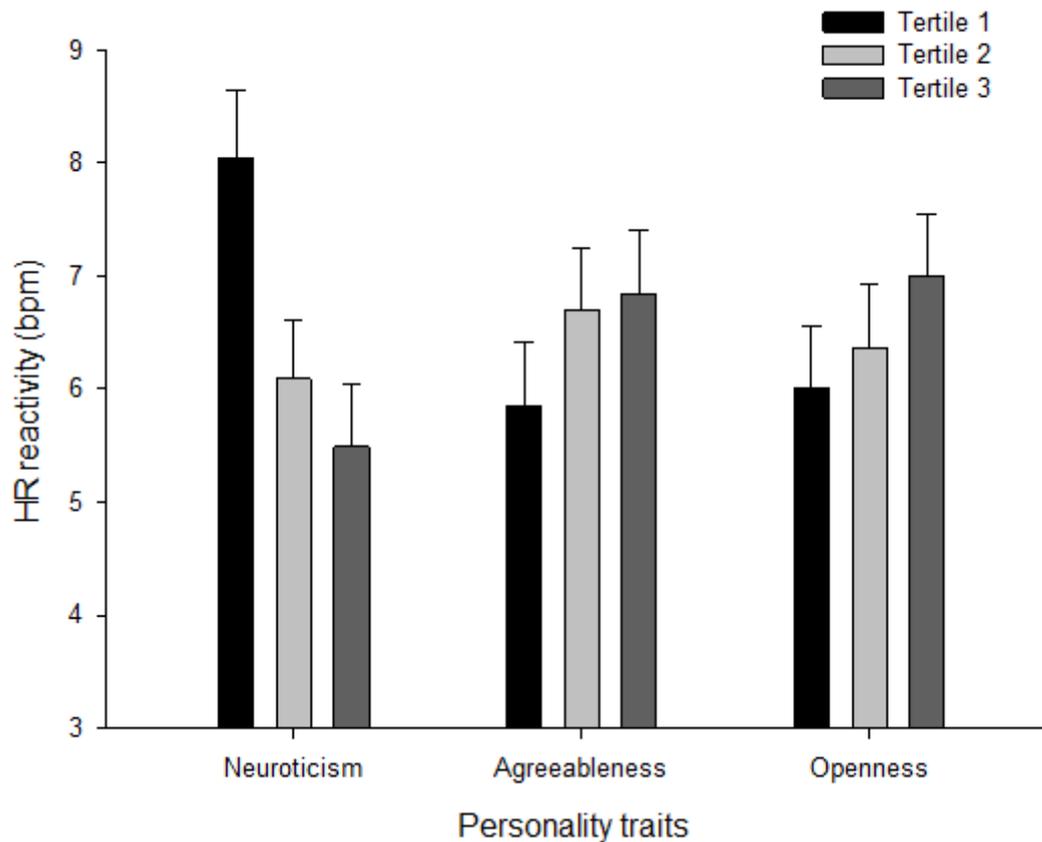


Figure 2.3. Mean (SE) heart rate reactivity by tertiles of neuroticism, agreeableness and openness

Neuroticism was the only personality trait that was consistently associated with SBP and DBP reactivity. A summary of the outcomes for regression analysis involving neuroticism, agreeableness, and openness is provided in Table 2.7. In the unadjusted models, neuroticism was negatively associated, whereas openness was positively associated with SBP reactivity. Agreeableness, extraversion, and conscientiousness were not related to SBP reactivity. In the models adjusting for sex, age and SES, neuroticism was still negatively associated with SBP reactivity but the association between openness and SBP reactivity was attenuated to non-significance. The negative association between neuroticism and SBP

reactivity was still evident in the fully adjusted regression model. As illustration, Figure 2.4 plots tertiles of neuroticism against SBP reactivity for the fully adjusted model. In the unadjusted regression model, the model that adjusted for sex, age and SES, and the fully adjusted model, high neuroticism scores were related to low DBP stress reactivity. The fully adjusted association is illustrated in Figure 2.4. None of the other personality characteristics were related to DBP stress reactivity.

In these fully adjusted models, smoking was also associated with SBP, DBP, and HR reactivity; current smokers exhibited smaller reactions. BMI was also associated with HR reactivity; those with higher BMI values were characterised by smaller HR reactions.

Table 2.7. Regression models for neuroticism, agreeableness, conscientiousness and SBP and DBP reactivity

	β	t	p	R ²
Neuroticism and SBP reactivity				
Unadjusted Model	-.16	2.99	.003	.025
Adjusted 1	-.13	2.40	.02	.016
Adjusted 2	-.12	2.18	.03	.014
Agreeableness and SBP reactivity				
Unadjusted Model	.08	1.58	.12	.007
Adjusted 1	.08	1.49	.14	.006
Adjusted 2	.09	1.53	.13	.007
Openness and SBP reactivity				
Unadjusted Model	.11	1.98	.05	.011
Adjusted 1	.07	1.32	.19	.005
Adjusted 2	.08	1.32	.19	.005
Neuroticism and DBP reactivity				
Unadjusted Model	-.15	2.88	.004	.023
Adjusted 1	-.15	2.73	.007	.021
Adjusted 2	-.14	2.43	.02	.017
Agreeableness and DBP reactivity				
Unadjusted Model	.09	1.63	.11	.008
Adjusted 1	.08	1.38	.17	.006
Adjusted 2	.08	1.34	.18	.005
Openness and DBP reactivity				
Unadjusted Model	.10	1.94	.06	.008
Adjusted 1	.09	1.54	.12	.007
Adjusted 2	.08	1.42	.16	.014

SBP = systolic blood pressure; DBP= diastolic blood pressure; Adjusted model 1; adjustment for sex, age and SES; Adjusted model 2= additional adjustment for alcohol consumption, smoking, BMI, use of anti-hypertensive medication, use of anti-depressant or anxiolytic medication, perceived commitment to the stress task, and baseline SBP/DBP activity (as appropriate).

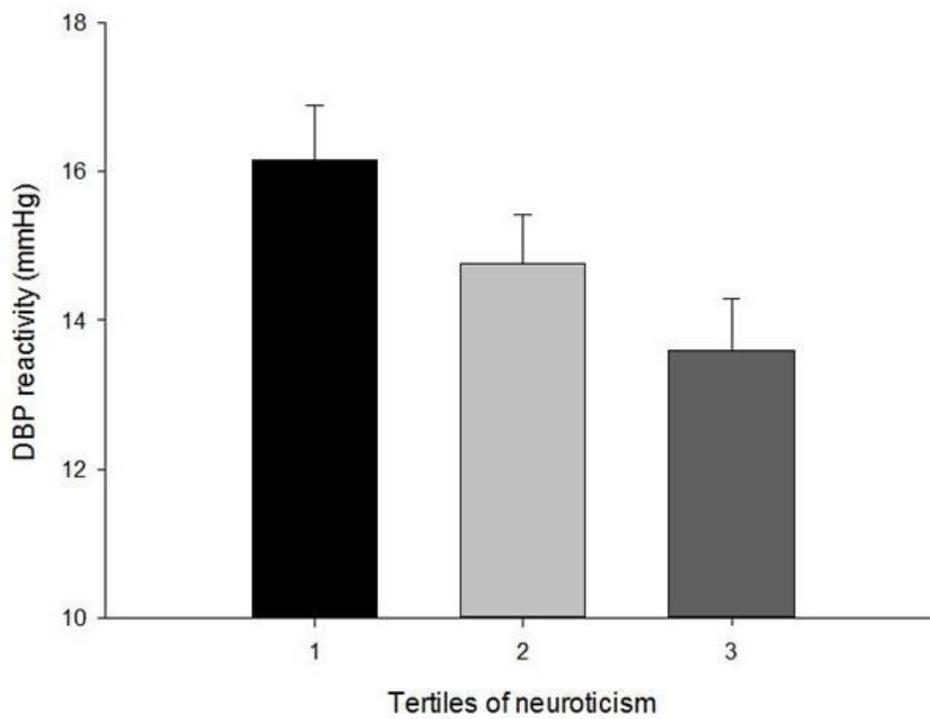
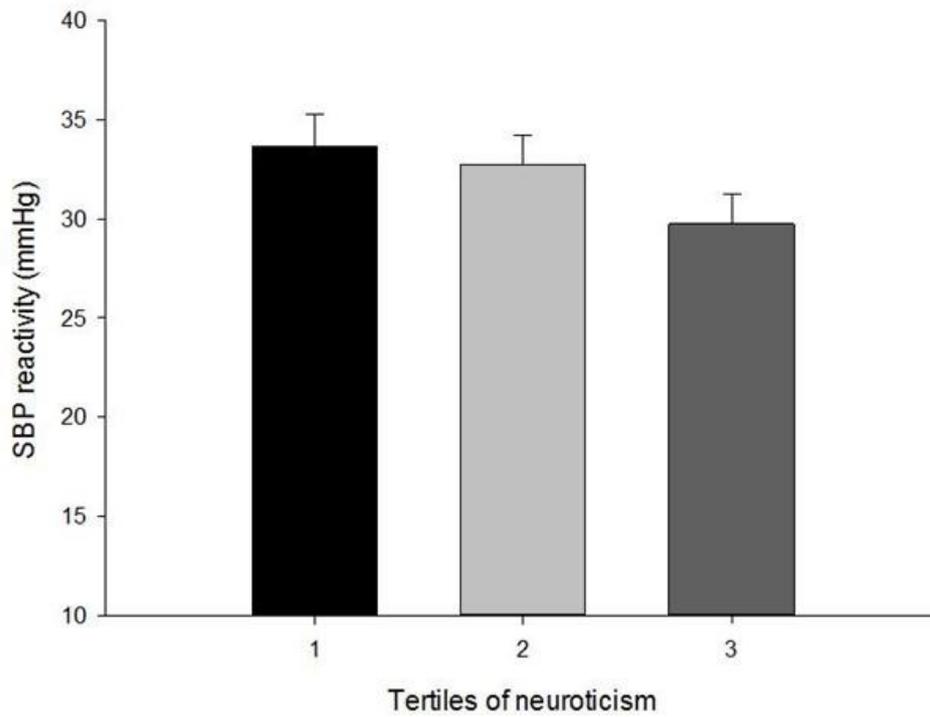


Figure 2.4. Mean (SE) systolic and diastolic blood pressure reactivity by tertiles of neuroticism

DISCUSSION

The present study examined, in a large middle aged cohort, the relationship between the Big Five personality traits and both cortisol and cardiovascular reactions to acute psychological stress. Individuals scoring higher on neuroticism and lower on agreeableness and openness had smaller cortisol stress reactions. These associations remained statistically significant following adjustment for a range of potential confounders: sex, age, SES, alcohol consumption, smoking, BMI, use of anti-hypertensive medication, use of anti-depressant or anxiolytic medication, perceived commitment to the stress task, and baseline cortisol activity. Neuroticism was also negatively associated with SBP, DBP, and HR stress reactivity, and these associations also survived full statistical adjustment. Agreeableness and openness were positively associated with HR reactivity in all the regression models tested. Extraversion and conscientiousness were not related to either cortisol or cardiovascular stress reactivity. Thus, it would appear that a negative constellation of personality traits, i.e., higher neuroticism, but lower agreeableness and openness, was associated with diminished stress reactions both of the cardiovascular system and the HPA axis.

The low cortisol reactivity observed in individuals with high neuroticism scores is in accordance with findings from previous research (Kirschbaum et al., 1993b; Oswald et al., 2006; Phillips et al., 2005). Similarly, the present observation that low cardiovascular stress reactivity characterises those with high neuroticism scores is also not without precedent (Hughes et al., 2011; Jonassaint et al., 2009). Indeed, from a recent meta-analysis of 71 laboratory studies, it was concluded that neuroticism, anxiety, and negative affect were associated with blunted rather than exaggerated cardiovascular reactions to acute stress (Chida & Hamer, 2008). The current study, then, adds further weight to the proposition that neuroticism is characterised by blunted reactions to acute stress exposure. As previously

suggested, null findings for cortisol (Kirschbaum et al., 1992; Kirschbaum et al., 1995; Schommer et al., 1999; Verschoor & Markus, 2011; Wirtz et al., 2007) and cardiovascular (Hutchinson & Ruiz, 2011; Kirkcaldy, 1984; Schommer et al., 1999; Verschoor & Markus, 2011; Wirtz et al., 2007) stress reactivity may well have been due to a variety of limitations including low power (Kirschbaum et al., 1995), insufficiently provocative stress exposures (Kirkcaldy, 1984; Williams et al., 2009), arbitrary categorization (Hutchinson & Ruiz, 2011) or restricted range (Schommer et al., 1999; Wirtz et al., 2007) of neuroticism scores (Hutchinson & Ruiz, 2011), examination of anticipatory rather than stress reactivity (Verschoor & Markus, 2011), or a range of other methodological issues (Stemmler & Meinhardt, 1990).

The diminished cortisol stress reactivity shown by individuals low on openness is in line with the results of some (Oswald et al., 2006) but not all (Schoofs et al., 2008; Wirtz et al., 2007) previous research. To the best of our knowledge the present study is the first to demonstrate that openness is positively associated with HR reactivity, with one previous study reporting no association (Williams et al., 2009). Further, in contrast to previous research which failed to find an association with cortisol (Oswald et al., 2006; Wirtz et al., 2007) or cardiovascular reactivity (Williams et al., 2009), low agreeableness in the present study was associated with attenuated cortisol, as well as HR, stress reactions. Again, previous null findings in the field (Oswald et al., 2006; Schoofs et al., 2008; Wirtz et al., 2007) may be attributed to low power and other methodological issues. The absence of an association between extraversion or conscientiousness and cortisol (Kirschbaum et al., 1992; Oswald et al., 2006; Schommer et al., 1999; Wirtz et al., 2007) or cardiovascular (Kirkcaldy, 1984; Vassend & Knardahl, 2005; Williams et al., 2009) stress reactivity is a common outcome. It is proposed that emotionally relevant stressors may be required to reveal any

effects of extraversion (Jonassaint et al., 2009), with effects for conscientiousness dependent upon the degree of control afforded during stress exposure (Hogan & Ones, 1997).

The reason that HR reactivity is more broadly associated with personality than blood pressure reactivity may reflect the fact that the former more closely reflects β -adrenergic activation. Sympathetic nervous system blockade studies indicate that cardiac reactivity closely reflects β -adrenergic activation, with indices of cardiac reactivity more sensitive to β -adrenergic blockade than blood pressure reactivity (Sherwood et al., 1986; Winzer et al., 1999). Although it has been proposed that β -adrenergic and HPA axis activation can be dissociated under some circumstances (Dickerson & Kemeny, 2004; Frankenhaeuser, 1982), substantial evidence demonstrates that they frequently co-vary, such that variations in the magnitude of β -adrenergic system reactions to acute stress, as indexed by cardiac reactivity, predict subsequent variation in HPA axis reactions, as indexed by cortisol reactivity (al'Absi et al., 1997; Bosch et al., 2009; Cacioppo, 1994).

Examination of the associations between personality and the self-reported stress task impact may also shed light on potential psychological mechanisms linking personality traits to physiological stress reactivity. Regarding neuroticism there would appear to be a paradox. Despite higher neuroticism being associated with greater perceptions of task stressfulness and difficulty, and lower feelings of control, those high in neuroticism exhibited blunted biological stress reactions. Previous research reporting attenuated cortisol and cardiovascular stress reactions in individuals exposed to high levels of chronic stress (Kudielka et al., 2009; Melamed et al., 2006) may help explain the blunted acute stress reactions in the context of greater threat perception in highly neurotic individuals (Schneider et al., 2012). Personality is proposed to be an enduring trait (Soldz & Vaillant, 1999; Terracciano et al., 2006). Thus, neurotic individuals will experience maladaptive

psychological states, high subjective stress and low feelings of control, each time they encounter acute stress. Over time, this would amount to something similar to the experience of chronic stress and contribute to blunted physiological stress reactivity as a result of “allostasis”: a down-regulation of the HPA axis and autonomic nervous system that impairs the physiological stress reactions to the acute challenges of daily life (McEwen, 1998, 1999). In support, the current findings emerged in a middle aged sample, one where there was presumably scope for prolonged personality effects on stress experience and, thus, on subsequent “allostasis”. It is important to note here that studies examining young student samples are prominent among those reporting no association between neuroticism and reactivity (Kirschbaum et al., 1992; Verschoor & Markus, 2011; Williams et al., 2009).

A dissociation between subjective and physiological stress reactions was also apparent for openness. Those scoring higher in openness reported the stress tasks as less stressful and difficult, and reported greater feelings of control, despite displaying higher cortisol and HR reactions. This dissociation may have been due to greater emotional suppression in highly open individuals as attention to inner feelings is a proposed component of openness (McCrae & John, 1992). Accordingly, individuals high in openness may have been attempting to avoid a vulnerable emotional state marked by feelings of stress, difficulty, and lack of control, as indicated in their subjective task ratings. Indeed, emotional suppression has been linked to greater physiological stress reactivity (Gross, 2002). Conversely, as greater openness to experience would suggest a propensity to relish and enjoy a variety of tasks (McCrae & John, 1992), in this case the stress tasks, highly open individuals may have experienced the more adaptive affective responses. A recent review suggests that there is often a dissociation between affective and physiological stress reactions (Campbell & Ehlert,

2012). The results of the present study add further support and suggest that the drivers of subjective stress reactions are different from those that drive physiological stress reactivity.

Given their implications for health and behaviour, it is important to identify the factors which contribute to the individual variation in the magnitude of HPA axis and cardiovascular stress reactivity (Bale, 2006; Uchino et al., 2007). Greater cortisol reactivity to acute stress has been associated with coronary artery calcification (Hamer et al., 2010), potentially increasing cardiovascular disease risk (Girod & Brotman, 2004); with hypercortisolism possibly also involved in the pathogenesis of mood and anxiety disorders (Holsboer, 2000; Susman et al., 2010; Young et al., 2000) and increased inflammatory disease susceptibility (Mason, 1991). Similarly, large magnitude cardiovascular stress reactions have been associated with cardiovascular disease pathology, such as hypertension, markers of systemic atherosclerosis, and left ventricular hypertrophy, e.g., Carroll et al., (2011a); Chida & Steptoe, (2010); Treiber et al., (2003).

However, recent evidence indicates that attenuated cortisol and cardiovascular stress reactions are also associated with a range of adverse health and behavioural outcomes (Carroll et al., 2009a; Carroll et al., 2011b). Blunted cortisol and cardiovascular stress reactions have shown cross-sectional and prospective associations with lower self-reported health (de Rooij & Roseboom, 2010; Phillips et al., 2009a), obesity (Carroll et al., 2008; Phillips et al., 2012), tobacco, alcohol, and substance dependence along with addiction risk (for a review see: Lovallo, 2007), and more recently exercise dependence (Heaney et al., 2011), risk of re-offending in delinquents (De Vries-Bouw et al., 2011), and disordered eating behaviour (Ginty et al., 2012). Further, depressed individuals have shown diminished cortisol (de Rooij et al., 2010; Taylor et al., 2006; Young et al., 2000) and cardiovascular (de

Rooij et al., 2010; Phillips et al., 2011; Salomon et al., 2009; Schwerdtfeger & Rosenkaimer, 2011) reactions to stress. On the basis of such results, it has been hypothesized that blunted physiological reactivity to stress may be a peripheral marker of dysregulation in the brain systems that support motivation (Carroll et al., 2009a; Carroll et al., 2011b; Lovallo, 2011). The present finding that negative personality characteristics are associated with blunted stress reactivity is certainly in keeping with this hypothesis. After all, such characteristics have been linked to many of the adverse health and behavioural outcomes associated with blunted stress reactivity e.g., depression (Bienvenu et al., 2004), poor self-reported health (Vassend & Skrondal, 1999), obesity (Sutin et al., 2011), disordered eating and exercise dependence (Bamber et al., 2000; Bulik et al., 2006; Cassin & von Ranson, 2005), and tobacco, alcohol, and substance dependence or addiction risk (Martin & Sher, 1994; Munafò et al., 2007; Terracciano & Costa, 2004; Terracciano et al., 2008). Dysregulation of the neural systems that support motivation have been proposed to play a role in all of these outcomes (Carroll et al., 2009a; Carroll et al., 2011b; Lovallo, 2011), as well as in determining personality (Cremers et al., 2010; DeYoung & Gray, 2009).

The present study is not without its limitations. First, personality traits and physiological stress reactivity were measured on average 5.5 years apart. However, these traits have been found to demonstrate high temporal stability across 45 years (Soldz & Vaillant, 1999; Terracciano et al., 2006). Second, personality assessments relied on self-report and, accordingly, the social desirability of reporting a positive constellation of personality traits may have influenced our findings. However, this seems unlikely. Self-reported traits have proved to be strong predictors of actual behaviour and have shown high correlations with personality ratings provided by spouses, peers and experts (Fleeson & Gallagher, 2009; McCrae, 1991). Third, it is not possible to determine causality or direction of causality from

observational analyses, and confounding by some unmeasured variable can never be wholly discounted (Christenfeld et al., 2004). Nevertheless a strength of the current study was that we were able to adjust statistically for an extensive range of potential confounders, many more than previous studies. Fourth, the observed effect sizes were small. The effect sizes were nonetheless comparable though to those observed for other variables known to affect cortisol and cardiovascular stress reactivity (Carroll et al., 2012a; de Rooij & Roseboom, 2010; Phillips et al., 2012). Finally, it is unclear whether or not the observed associations between personality factors and reactivity are independent of one another. The five personality traits measured were correlated, although imperfectly so, and it is probably best to view personality as an intersecting constellation of traits rather than as a set of independent factors (Digman, 1997). It may be beneficial for future research to use multivariate analyses to examine the predictive role of profiles/clusters of traits as opposed to individual traits in isolation (Marshall et al., 1994).

In conclusion, the present analyses indicated that cortisol and HR stress reactivity were negatively associated with neuroticism, but positively associated with agreeableness and openness. High neuroticism scores were also negatively associated with diminished blood pressure reactivity. As such, the results provide further support to the notion that blunted cortisol and cardiovascular reactivity may be maladaptive and may reflect dysregulation of the neural systems supporting motivated behaviour.

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CHAPTER THREE

**CARDIOVASCULAR AND CORTISOL REACTIONS TO ACUTE
PSYCHOLOGICAL STRESS UNDER CONDITIONS OF HIGH
VERSUS LOW SOCIAL EVALUATIVE THREAT: ASSOCIATIONS
WITH THE TYPE D PERSONALITY CONSTRUCT**

ABSTRACT

Type D personality has been associated with cardiovascular disease risk, with exaggerated physiological stress reactions a potential mechanism. This study compared physiological stress reactions of Type D and non-Type D individuals in settings varying in social evaluation characteristics. Two thousand and three hundred students were screened for Type D personality, with 130 selected individuals assigned to an asocial stress exposure condition (31 Type D, 30 non-Type D: 52% female) or a condition high in social evaluative threat (35 Type D, 34 non-Type D: 55% female). Blood pressure, heart rate and salivary cortisol were measured at rest and in response to the stress protocols. The greatest cardiovascular stress reactions were exhibited by Type D participants in the high social evaluation condition; this was reflected in significant group x condition interactions for systolic, $F(1,126) = 7.29, p = .008, \eta^2 = .055$, and diastolic, $F(1,126) = 5.23, p = .024, \eta^2 = .040$, blood pressure and heart rate, $F(1,126) = 5.04, p = .027, \eta^2 = .038$, reactivity. Moreover, only Type Ds in the social condition mounted a positive cortisol response, $F(1,33) = 5.07, p = .031, \eta^2 = .133$. In the asocial condition, Type Ds tended to show blunted systolic ($p = .091$) and diastolic ($p = .054$) blood pressure reactivity relative to their non-Type D counterparts. It would appear Type D individuals show exaggerated or blunted stress reactions depending upon the social evaluative nature of the stress exposure, with this dysregulation of the stress response potentially increasing cardiovascular disease risk.

Keywords: Type D personality; social evaluation; cardiovascular reactivity; cortisol reactivity; psychological stress.

INTRODUCTION

Type D, 'distressed', personality is characterised by the propensity to experience high levels of negative emotion (negative affectivity: NA) and the tendency to inhibit emotions in social situations (social inhibition: SI) (Denollet, 2005). It has been associated with a range of negative cardiovascular outcomes (Mols & Denollet, 2010), although a recent meta-analysis suggests the early Type D studies may have overestimated the prognostic relevance (Grande et al., 2012). The negative cardiovascular outcomes include increased risk of mortality in existing coronary artery disease patients (Denollet et al., 2010; Grande et al., 2012), and increased risk of developing coronary heart disease in healthy populations (Svansdottir et al., 2013). However, the underlying mechanisms of these associations remain unclear, although dysregulation of the sympathetic-adrenal-medullary (SAM) system and hypothalamic-pituitary-adrenocortical (HPA) axis, as evidenced by exaggerated physiological reactions to acute stress, have been proposed. There is substantial evidence that those who show exaggerated cardiovascular stress responses are at increased risk of developing cardiovascular disease due to various manifestations such as hypertension (Carroll et al., 2012b; Carroll et al., 2003), systemic atherosclerosis (Everson et al., 1997), left ventricular hypertrophy (Kapuku et al., 1999; Murdison et al., 1998), coronary artery calcification (Matthews et al., 2006), as well as increased cardiovascular disease mortality (Carroll et al., 2012a). Additionally, exaggerated cortisol reactivity has been associated with coronary artery calcification (Hamer et al., 2010) and increased hypertension (Hamer & Steptoe, 2012) and cardiovascular disease risk (Girod & Brotman, 2004).

Previous studies investigating the cardiovascular and cortisol response to acute psychological stress in Type D individuals have yielded inconsistent findings. In response to a mental arithmetic challenge, exaggerated systolic (SBP) reactivity was associated with

increased SI, whereas blunted heart rate (HR) responses were related to, albeit non-significantly, high NA in undergraduate males (Habra et al., 2003). Additionally, both high NA and SI were associated with exaggerated cortisol reactivity (Habra et al., 2003). Further, in comparison to non-Type D college students, males with Type D personality exhibited exaggerated cardiac output responses during a mental arithmetic task, with no differences in blood pressure or HR reactivity (Williams et al., 2009). Conversely, lower HR and cardiac output reactivity has been reported in Type D females, again with no differences in SBP or diastolic blood pressure (DBP) responses (Howard et al., 2011).

A potential reason for the mixed findings for Type D may be the social nature of the stress tasks. This is pertinent because the SI component of Type D refers to the inhibition of emotions in social situations. Emotional inhibition has been shown to relate to exaggerated cardiovascular (Gross & Levenson, 1997) and cortisol (Lam et al., 2009) stress reactions. For example, using a serial mathematical subtraction task designed to be socially evaluative with elements of harassment, reward, and overt performance monitoring, individuals with high SI showed exaggerated blood pressure and cortisol reactivity (Habra et al., 2003). On the other hand, no differences between Type D and non-Type D participants in blood pressure or HR stress responses have been observed when a mental arithmetic stressor but with minimal social evaluation was employed (Williams et al., 2009). Further, diminished HR and cardiac output reactivity were observed in Type D individuals during a serial subtraction task without social elements that used a non-verbal keypad response and the experimenter scored performance behind an opaque screen (Howard et al., 2011). Taken together, these studies support the contention that Type D individuals may exhibit exaggerated cardiovascular and cortisol reactions to socially evaluative stressors, but not in asocial conditions.

There is evidence that social evaluation *per se* increases cardiovascular responses to stress (Allen et al., 1991; Smith et al., 1997; Wright et al., 1995). A similar pattern emerges for cortisol reactivity (Dickerson et al., 2004). Indeed it has been demonstrated that social-evaluative threat is required to elicit activation of the HPA-axis (Dickerson et al., 2008; Gruenewald et al., 2004; Kirschbaum et al., 1993a). Indeed, a dose-dependent increase in cardiovascular and cortisol reactivity to a speech stress task was observed under conditions of increasing social-evaluative threat, as a consequence of increasing audience size during a speech stressor (Bosch et al., 2009). Thus, the overall consensus suggests that increasing social-evaluation perturbs increased cardiovascular and cortisol stress reactivity.

Given the potential role of the social nature of the stressor used in explaining the previous mixed associations between Type D personality and physiological stress reactivity, the current study, was designed to compare cardiovascular and cortisol stress reactions of Type D individuals with individuals who were non-Type D in two settings varying in social evaluation characteristics. It was expected that increased social evaluation would enhance cardiovascular and cortisol reactivity regardless of Type D classification, and that the enhanced stress reactivity to socially evaluative stress would be particularly evident among individuals with Type D personality.

METHODS

Participants

A questionnaire screening for Type D personality was administered to 2300 University of Birmingham students (1350 women) via e-mail and online recruitment. Based on screening scores, 130 (66 Type D) healthy participants were invited to attend a laboratory session

which consisted of either a social or asocial stress testing condition. The mean (SD) age of the selected sample was 20.5 (1.87) years and their mean (SD) body mass index (BMI) was 22.8 (2.94) kg/m². Table 3.1 presents the socio-demographics and health behaviours of the four sub-groups (Type D social, Type D asocial, non-Type D social, non-Type D asocial). The majority of the participants indicated they were “white” (89%). None had a history of cardiovascular disease, a current illness or infection, or were taking medication, with the exception of three individuals using anti-depressive medication: 2 Type D asocial and 1 Type D social. All participants provided written informed consent and the study was approved by the University of Birmingham ethics committee. Data collection took place between November 2012 and March 2013.

Questionnaires

Type D personality

Type D personality was assessed using the Type D Scale-14 (DS14) (Denollet, 2005) which comprises two 7-item subscales, measuring NA, e.g., “I am often in a bad mood; “I often make a fuss about unimportant things”, and SI, e.g., “I find it hard to start a conversation”; “I am a closed kind of person”. Respondents indicate their answers on a 5-point Likert scale ranging from 0, false, to 4, true. These subscales are summed to yield an overall measure of Type D. A score of ≥ 10 on both NA and SI subscales has been used to indicate Type D personality classification (Denollet, 2005; Emons et al., 2007). Both the NA and SI subscales have shown good test-retest stability, $r = .72$ and $.82$ respectively, and high internal validity, Cronbach’s $\alpha = 0.88$ and 0.86 , respectively (Denollet, 2005). The present study found a Cronbach’s α of $.95$ and $.94$ for the NA and SI scales respectively.

Health behaviours and depression

A questionnaire adapted from the Whitehall II study (Marmot et al., 1991) was administered to measure average daily smoking (0, 1-5, 6-10, 11-20, 21-40, 41+ cigarettes per day) and weekly alcohol intake (0, 1-5, 6-10, 11-20, 21-40, 41+ units per week) which were subsequently dichotomised to current/no smoker and $<11/\geq 11$ weekly units. In order to calculate cardio-respiratory fitness, participants indicated how much time they spent in activities of different intensities which were allocated category scores from 1 to 5, where 1 indicates inactivity and 5 indicates participation in brisk exercise for over 3 h per week, with the physical activity levels of 1, 2, 3, 4, and 5 assigned scores of .00, .32, 1.06, 1.76, and 3.03, respectively (Jurca et al., 2005). The following formula was used to calculate cardio-respiratory fitness in METS: $(0 \text{ if female or } 2.77 \text{ if male}) - ((\text{age} \times 0.10) - ((\text{BMI}) \times 0.17) - ((\text{resting heart rate}) \times 0.03) + (\text{physical activity score}) + 18.07$ (Jurca et al., 2005).

The seven item depression subscale of the Hospital Anxiety and Depression Scale (HADS) (Zigmond & Snaith, 1983) was used to measure depressive symptoms. Responses are indicated on a 4-point scale, ranging 0-3, with higher scores indicating greater depression. Psychometric analysis indicates good test-retest reliability with a coefficient of .85 (Herrmann, 1997), and Cronbach's alpha's of .90 (Moorey et al., 1991).

Psychological stress task questionnaire

Participants indicated perceived stressfulness and task engagement immediately following each stress task by using a 7 point Likert-type scale ranging 0, not at all to 6, extremely. These scales have been used successfully in previous studies investigating physiological reactivity to acute psychological stress (Ginty et al., 2012; Heaney et al., 2011). To check the success of the social evaluation manipulation, following each stress task, participants

indicated on a 7 point Likert-type scale, ranging 0, not at all to 6, extremely, the extent to which they felt they were being socially evaluated by others.

Acute psychological stress task

The 5-minute modified Stroop colour-word interference task was presented on a computer screen and required participants to identify the incongruent colour in which a target word was presented by selecting, one of four identifier words naming the colour (Gianaros et al., 2008). Performance was titrated to ~50% to control for individual differences in task performance and percentage of correct responses was recorded as a check. Responses were made on a keypad. A mistake, resulted in an 'X', and a response exceeding the 5-second time limit resulted in a 'Too late' message on screen, both accompanied by a short auditory beep. The Stroop was followed by a 5-minute inter-task rest period and then the 10-minute Paced Auditory Serial Addition Test (PASAT) (Gronwall, 1977), which has been shown to perturb both cardiovascular and salivary cortisol activity (Ginty et al., 2012; Ring et al., 2002), and demonstrates good test-retest reliability (Willemsen et al., 1998). Participants are presented, via a CD player, with a series of single digit numbers and are required to add the present number to the previously presented number, and report their answer aloud. They then have to remember the last number they heard in order to add it to the next number read out from the CD. During the last five numbers of each block of 10 numbers, a brief burst of loud aversive noise was presented; all participants received 21 noise bursts. Instructions for both tasks were presented via a video on a computer screen followed by a short practice. To maintain engagement for both tasks participants were informed they would start with 1000 points with 5 points deducted for each wrong answer.

Social manipulation conditions

Asocial condition: To maintain consistency in the auditory beeps received but to minimise social evaluation, during the Stroop and PASAT tasks respectively, the beeps were relayed via the computer programme and CD player. To maintain engagement for both tasks, participants were informed to perform to the best of their ability but that they were not being evaluated. The only individual present was the experimenter measuring physiological activity. Performance score on the PASAT was calculated from a Dictaphone recording which the participant was unaware of.

Social condition: To introduce social evaluation and comparison for both tasks, participants were informed that their performance was being assessed and they were in direct competition with fellow participants. They were also informed that their scores would be displayed on a prominent leader board, which they could see, and that they should attempt to beat scores currently displayed. Participants were also filmed and this was displayed live on a television screen, which they were requested to remain focused on during the PASAT. They were also told that the recording would be assessed by “body language experts”, although no such assessment was undertaken. Following task instruction and practice, an additional experimenter wearing a laboratory coat entered the room and stood in close proximity to participant to obtrusively observe them. The additional experimenter left the room following each task. During the Stroop test, the experimenter sounded a buzzer each time they gave an incorrect answer or exceeded 5 seconds before responding. During the PASAT, the experimenter conspicuously scored the participants and sounded a buzzer once during the last five numbers of each block of 10 numbers, mostly corresponding with an error or hesitation. The amount of auditory beeps presented in the social and asocial conditions were exactly the same; only the method of delivery varied.

Cardiovascular and salivary cortisol measures

The laboratory session consisted of six periods; 10-minute adaptation, 10-minute baseline, 5-minute Stroop Task, 5-minute inter-task period, 10-minute PASAT stress task, and 10-minute recovery. SBP, DBP and HR were measured discontinuously using a semi-automatic sphygmomanometer (Omron, IL) at minute 1, 3, 5, 7 and 9 during baseline and PASAT, and at minute 1 and 3 during the Stroop task. A single measure was also taken during the adaptation period for familiarity, although this measure was discarded. Two stimulated 2-minute saliva samples were obtained using salivettes at minute 8 of baseline and 8-minutes into the recovery period. Salivettes were centrifuged for five minutes at 4000rpm before being stored at -20°C until assay. ELISA kits (IBL International, Germany) were used to analyse all cortisol samples in duplicate. The mean intra-assay coefficient of variation was 9.9% and the inter-assay coefficient was 4.5%. Due to collection difficulties with one Type D participant in the social condition, cortisol assays were analysed for 129 participants.

Procedure

To ensure accurate Type D classification, the current study used more stringent cut-off criteria than those suggested by Denollet (2005): a DS14 score of ≥ 14 and ≤ 8 on both the NA and SI subscales classified Type D and non-Type D, respectively. These cut-offs were based upon the upper and lower quartiles of our sample, as although median splits (Habra et al., 2003; Howard & Hughes, 2013; Howard et al., 2011) have also been employed to create Type D dichotomies, this can create a risk of misclassification and is generally advised against (Veiel, 1988). Type D and non-Type D individuals were randomly allocated to either the social or asocial condition. The study employed a double-blind testing procedure

with the laboratory sessions commencing at 13:30, 15:30 or 17:30. Prior to testing, participants were requested to refrain from eating for 1hr, drinking caffeine or smoking for 2hr, and from physical exercise and drinking alcohol for 12hr. Participants' height and weight were measured, and BMI subsequently calculated, and they completed the questionnaire pack, including a further DS14 to ensure correct Type D classification and test-retest reliability. During the adaptation period, participants lay in a semi-recumbent position, and remained in that position throughout the session. The blood pressure cuff was attached and then participants lay quietly for 10- minutes. This was followed by a formal 10-minute resting baseline before the stress tasks. Following each task, participants provided ratings of subjective impact and social evaluation. During the 5-minute inter-task period participants lay quietly. To exclude any social evaluation from the experimenter initiating physiological measures, participants were informed this experimenter was only present to conduct such measurements and was not concerned with task performance.

Data Analysis

For the cardiovascular measures, averages of the baseline and stress (combined average of Stroop and PASAT) periods were calculated. Reactivity scores were calculated by subtracting baseline from stress averages. Repeated measures ANOVAs were performed to confirm the stress tasks perturbed cardiovascular and cortisol activity. Group differences in socio-demographics, health behaviours, depression, stress task perceptions and social manipulation ratings (both averaged across tasks), performance, and baseline cardiovascular and cortisol variables were tested using 2 (Type D versus non-Type D group) x 2 (social versus asocial condition) ANOVAs for continuous variables, and chi-square for categorical variables. Similar 2 x 2 ANOVAs were conducted to analyse group differences in

physiological reactivity. 2 x 2 ANCOVAs were utilised to determine whether group differences withstood adjustment for potential confounding variables. Pairwise comparisons were undertaken to elucidate significant differences. Partial η^2 is reported as an index of effect size throughout. p -values of $\leq .05$ were considered statistically significant.

RESULTS

Validating group allocation

All participants allocated to the Type D and non-Type D groups conformed to the respective cut off criteria of ≥ 14 and ≤ 8 on the NA and SI subscales. For both the NA and SI subscales, and the Total DS14 scores, there were significant main effects of group ($p < .001$); as would be expected, Type D individuals registered higher scores. There were no main effects for condition nor group x condition interactions ($p > .050$). This pattern of results was identical for the scores on the DS14 completed in the laboratory to confirm the robustness of group allocation. For the total scores, the individual group means and SDs were as follows: Type D asocial (38.4 ± 4.88), Type D social (39.9 ± 5.50), non-Type D asocial (7.1 ± 2.83) and non-Type D social (6.4 ± 2.79). The test-retest scores over an average 4-month period were $r = .93$ and $.92$, for the NA and SI scales, respectively.

Socio-demographics, health behaviours and depression

The summary data for socio-demographics, health behaviours and depression are presented in Table 3.1. Group differences emerged only for age, estimated cardio-respiratory fitness and depression, $F(1,126) = 8.10, p = .005, \eta^2 = .060$; $F(1,126) = 11.56, p = .001, \eta^2 = .084$; $F(1,124) = 70.26, p < .001, \eta^2 = .362$, respectively; Type D individuals were slightly older, had lower cardio-respiratory fitness, and scored higher on the depression subscale. The main

effects for condition and group x condition interactions for these variables were not statistically significant ($p > .060$).

Social manipulation

As expected, social evaluation ratings differed between conditions, $F(1,125) = 46.30, p < .001, \eta^2 = .270$. Participants in the asocial condition ($M \pm S.D$: Type D = 3.3 ± 1.50 . Non-Type D = 2.3 ± 1.57) rated the tasks as low-to-moderate in terms of social evaluation, whereas participants in the social condition ($M \pm S.D$: Type D = 4.7 ± 1.16 . Non-Type D = 4.4 ± 1.47) rated the tasks as moderate-to-highly socially evaluative. There was also a significant main effect of Type D group, $F(1,125) = 7.69, p = .006, \eta^2 = .058$, with Type D individuals overall reporting the tasks, irrespective of condition, as more socially evaluative. There was no group x condition interaction effect ($p = .15$).

Stress task ratings and performance

Summary stress task ratings and performance data are also presented in Table 3.1. There were no significant main effects or interactions for self-reported engagement or PASAT total score indicating no overall differences in task engagement ($p > .050$). Additionally, the mean correct Stroop response rate was 57.5% (S.D= 6.35) with no main effects or interactions indicating successful titration and therefore similar task engagement. Although there was no condition effect or group x condition interaction ($p > .19$ in both cases) for rated stressfulness, Type D participants perceived the tasks as more stressful than their non-Type D counterparts, $F(1,125) = 8.18, p = .005, \eta^2 = .061$.

Table 3.1. Characteristics and stress task ratings and performance of Type D and Non-Type D participants stratified by asocial and social conditions

	Asocial		Social	
	Type D	Non-Type D	Type D	Non-Type D
	Mean (SD)/N (%)			
N	31 (24)	30 (23)	35 (27)	34 (26)
Gender (females)	18 (58)	14 (47)	18 (51)	20 (59)
Age (years)	21.5 (2.41) ^a	19.9 (1.53)	20.4 (1.91) ^a	20.1 (1.17)
BMI (kg/m ²)	22.9 (2.45)	22.8 (3.10)	22.7 (3.57)	22.8 (2.62)
HADS (depression subscale)	5.6 (3.11) ^a	1.6 (1.63)	6.2 (3.96) ^a	1.7 (1.96)
Current smoker	6 (19)	2 (7)	5 (14)	7 (21)
Units of alcohol per week (≥ 11)	2 (7)	9 (30)	10 (29)	9 (27)
Cardio-respiratory fitness (METS)	12.7 (2.06) ^a	14.2 (1.95)	12.9 (1.96) ^a	13.8 (2.18)
Stressfulness	4.2 (1.32)	3.5 (1.23)	4.4 (1.07)	3.9 (1.31)
Engagement	4.2 (1.53)	4.5 (1.31)	4.4 (1.21)	4.3 (1.42)
Correct responses to Stroop (%)	58.0 (7.31)	58.3 (5.99)	57.2 (6.12)	56.6 (6.09)
PASAT total score	672.6 (147.17)	685.7 (148.00)	728.1 (139.55)	677.5 (117.69)

^a = $p < .05$ compared to Non- Type D

Cardiovascular stress reactions

There were no group differences in any of the baseline cardiovascular values ($p > .16$).

Repeated measures ANOVAs confirmed that the stress tasks increased SBP, DBP and HR.

Table 3.2 presents the summary data and pertinent statistics. There were no group main effects for any of the cardiovascular reactivity variables ($p > .30$ in all cases). However, the social condition proved more provocative than the asocial condition: for SBP, $F(1,126) = 12.70, p = .001, \eta^2 = .092$; for DBP, $F(1,126) = 7.72, p = .006, \eta^2 = .058$; for HR, $F(1,126) = 7.88, p = .006, \eta^2 = .059$. Importantly, the group x condition interaction was significant in each case: for SBP, $F(1,126) = 7.29, p = .008, \eta^2 = .055$; for DBP, $F(1,126) = 5.23, p = .024, \eta^2 = .040$; for HR, $F(1,126) = 5.04, p = .027, \eta^2 = .038$. These interaction effects are illustrated in Figure 3.1. For both HR and blood pressure, the Type D participants in the social condition had significantly higher reactivity than the asocial condition Type D participants ($p \leq .050$ in all cases). In addition SBP and HR reactivity was greater for the Type D participants in the social condition than the non-Type D participants in both the social and asocial conditions ($p < .035$ in all cases). Within the asocial condition, Type D individuals displayed a significantly lower DBP reactivity ($p = .054$) than non-Type D participants; for the same comparison there were similar indications of blunted SBP reactivity for Type Ds in the asocial condition ($p = .091$). Similarly within the asocial condition, compared to their non-Type D counterparts, Type D participants had lower HR reactivity, although this was not significant ($p = .41$). Both SBP and DBP reactivity ($p < .020$ in both cases) for Type D individuals in the asocial condition was significantly attenuated relative to that shown by non-Type D participants in the social condition. Finally, there were no differences in reactivity between the Non-Type D individuals across the conditions ($p > .05$).

Table 3.2. Baseline and stress levels for cardiovascular and cortisol parameters

	Baseline	Stress	F (1, 129)	<i>p</i>	η^2
	Mean (SD)				
SBP (mmHg)	103.9 (9.49)	118.8 (15.13)	293.95	<.001	.695
DBP (mmHg)	53.9 (5.47)	65.0 (7.50)	489.23	<.001	.791
HR (bpm)	63.1 (10.31)	78.4 (14.66)	262.04	<.001	.671
Cortisol (nmol/L)	8.4 (5.14)	8.4 (5.78)	<.01	.99	<.001

Statistical analyses: Repeated measures ANOVAs comparing baseline to stress levels.

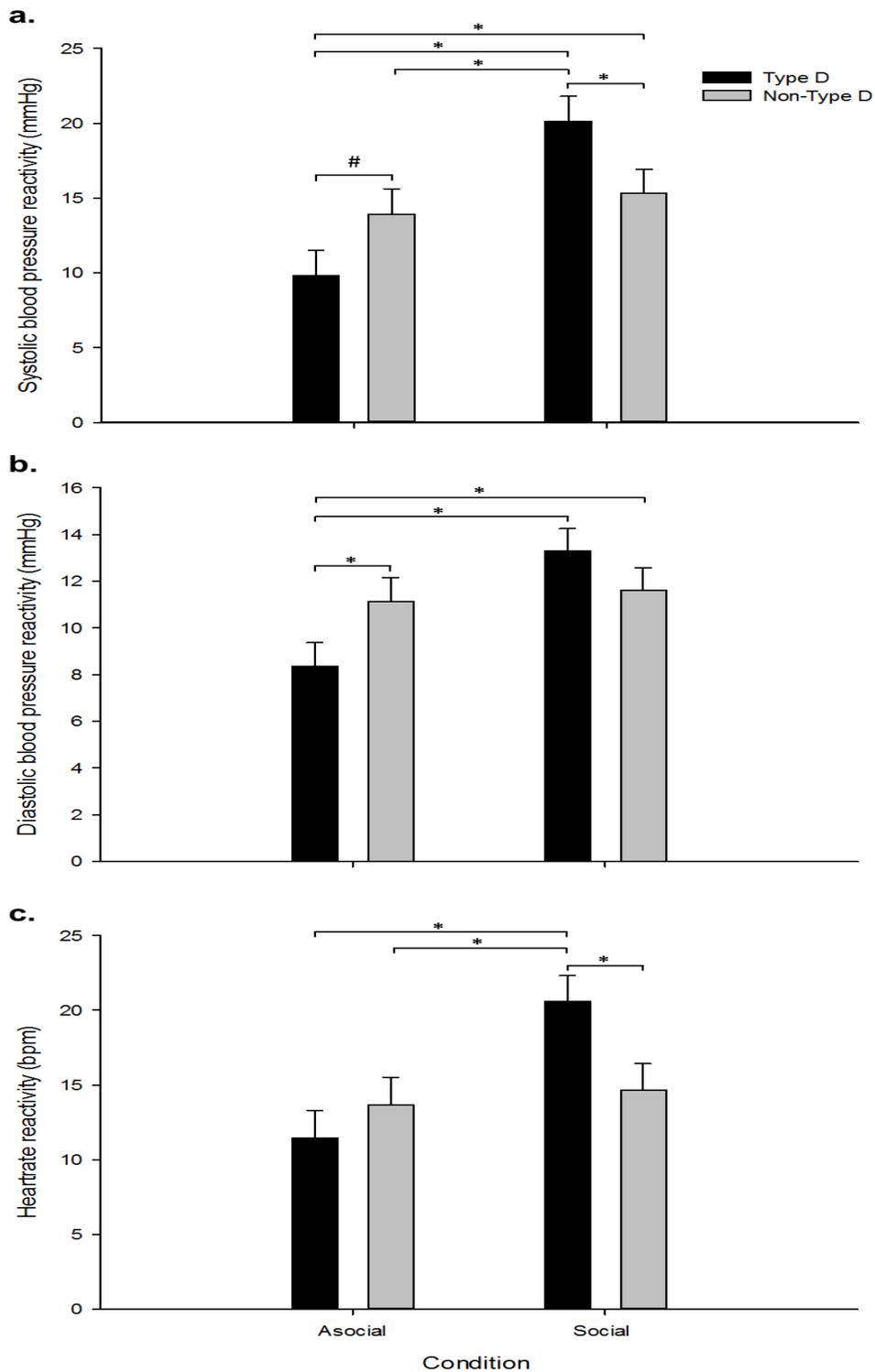


Figure 3.1. Mean (SE) (a) systolic blood pressure, (b) diastolic blood pressure, and (c) heart rate reactivity levels across social manipulation and Type D classification. * $p \leq .05$, # $p \leq .10$

Cortisol stress reactions

Baseline cortisol levels did not differ between groups ($p = .14$). Overall, the stress tasks failed to increase cortisol concentration. Table 3.2 presents the summary data and statistics. There were, however, significant main effects for both group, $F(1,125) = 7.87, p = .006, \eta^2 = .059$, and condition, $F(1,125) = 5.31, p = .023, \eta^2 = .041$, for cortisol change; Type D participants were more reactive and the social condition was more provocative. The group x condition interaction effect did not meet the criterion for statistical significance ($p = .16$). The summary cortisol data are presented in Figure 3.2. As can be seen, there was a positive cortisol reaction only for Type D participants in the social condition; pairwise comparisons indicated that the cortisol change score for Type D individuals in the social condition differed from that observed for the other three groups ($p < .050$ in each case). As a sensitivity analysis, repeated measures ANOVAs comparing baseline and stress cortisol concentrations were conducted separately for each of the four groups. The only group to demonstrate a significant increase in cortisol was the Type D participants in the social condition, $F(1,33) = 5.07, p = .031, \eta^2 = .133$; the non-Type D asocial participants actually showed a decrease, $F(1,29) = 8.42, p = .007, \eta^2 = .225$. For Type D asocial ($p = .50$) and non-Type D social participants ($p = .25$) cortisol did not change significantly from baseline to stress exposure.

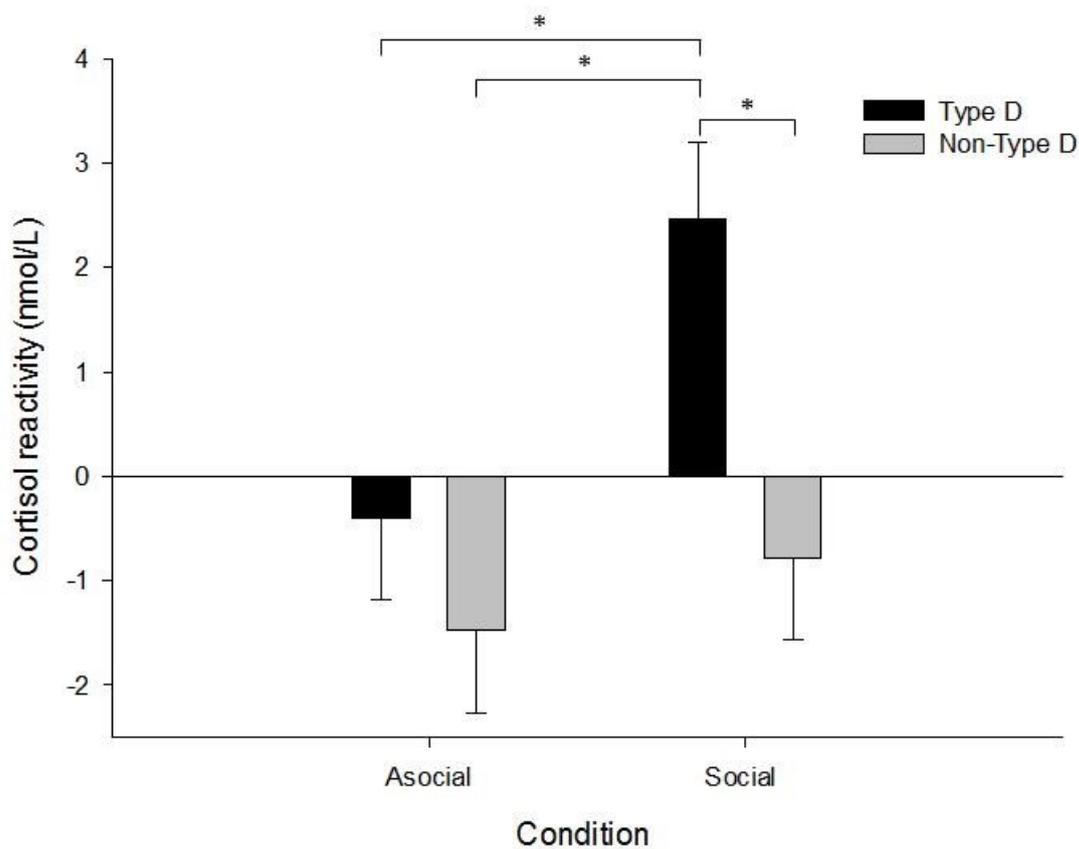


Figure 3.2. Mean (SE) salivary cortisol reactivity levels across social manipulation and Type D classification. * $p \leq .05$

Covariate analyses

As there were sub-group differences in age, cardio-respiratory fitness, depression and perceived stressfulness, the main analyses were repeated, adjusting for these variables in addition to the appropriate baseline cardiovascular and cortisol levels. The SBP, $F(1,118) = 5.45, p = .021, \eta^2 = .044$, DBP, $F(1,118) = 4.01, p = .048, \eta^2 = .033$, and HR, $F(1,118) = 4.27, p = .041, \eta^2 = .035$, reactivity group x condition interactions withstood covariate adjustment. Similarly, for cortisol reactivity the main effect of condition, $F(1,117) = 6.58, p$

= .012, $\eta^2 = .053$ was preserved, although the main effect of group was reduced to a trend, $F(1,117) = 3.04, p = .084, \eta^2 = .025$. Additionally controlling for anti-depressant medication use did not alter these outcomes. Similarly, although there were no group differences in BMI ($p = .78$), sex ($p = .74$), or smoking status ($p = .41$), controlling for these potential confounders did not change the outcomes. There were no differences in any of the physiological reactivity variables across the three testing times (all p 's > .055) and controlling for testing time did not change any of the outcomes.

DISCUSSION

The present study was the first we are aware of to compare cardiovascular and cortisol stress reactions of extreme Type D and non-Type D individuals in two settings varying in social evaluation characteristics. As expected, the condition high in social evaluative threat elicited greater cardiovascular and cortisol reactions, irrespective of the Type D status of the participants; this very much resonates with the findings of previous research into the physiological impact of social evaluation (Allen et al., 1991; Bosch et al., 2009; Dickerson & Kemeny, 2004; Smith et al., 1997; Wright et al., 1995). Importantly, given the primary aim of the current study, this enhanced reactivity was a particular feature of Type D participants. Whereas non-Type D individuals in the social and asocial conditions exhibited cardiovascular reactions of a comparable magnitude, Type D individuals in the social condition were characterised by greater SBP, DBP and HR reactions than their asocial Type D counterparts. Regarding cortisol stress reactions, only Type D individuals in the social condition mounted a positive cortisol response. There is also evidence in the present study that Type D individuals in the asocial condition exhibit blunted blood pressure reactions relative to their non-Type D counterparts. The overall pattern of results was still evident

following statistical adjustment for potential confounders; age, cardio-respiratory fitness, depression, perceived stressfulness and baseline physiological levels.

As indicated, previous studies of stress reactivity and Type D personality have produced mixed results. The findings of the present study would appear to go some way toward accounting for these discrepancies. Type D individuals seem to be characterised by higher cardiovascular and cortisol reactivity mainly when exposed to stress tasks high in social evaluative threat (Habra et al., 2003). Where the stress exposure is largely asocial, Type D individuals either do not differ from their non-Type D counterparts in terms of cardiovascular stress reactivity (Williams et al., 2009) or actually show diminished cardiovascular reactions (Howard et al., 2011). Our finding of blunted reactivity in Type D individuals within the asocial condition is in line with this.

It should also be noted that Type D individuals, regardless of the social evaluative condition, reported the tasks to be more stressful. Indeed, research has suggested that Type D individuals may have a cognitive bias towards interpreting threat and this may increase their vulnerability to perceived social stress (Grynberg et al., 2012). This is supported by neuroimaging data which has shown the two components of Type D personality, SI and NA, are associated with unique brain activity patterns in response to perceived socially threatening stimuli (Kret et al., 2011). Interestingly, despite the Type D individuals in the asocial condition reporting the tasks as significantly more stressful, they actually had lower SBP, DBP and HR reactions. This is in line with previous personality research which has demonstrated that there is a paradox between subjective self-report ratings and physiological responses (Bibbey et al., 2013). It would appear that there may be further thematic links

between Type D emotional patterns and the particular physiological response profiles observed here, thus it would be recommended for future research to investigate this further.

Interest in Type D personality arises from its association with increased risk for cardiovascular disease morbidity (Svansdottir et al., 2013) and mortality (Denollet et al., 2010; Grande et al., 2012). Dysregulation of the SAM system and HPA-axis has been implicated (Sher, 2005) and there is now substantial evidence that those who show exaggerated cardiovascular stress responses are at increased risk of developing cardiovascular disease morbidity (Carroll et al., 2012b; Carroll et al., 2003; Everson et al., 1997; Kapuku et al., 1999; Matthews et al., 2006; Murdison et al., 1998) and mortality (Carroll et al., 2012a). In addition, exaggerated cortisol reactivity has been associated with coronary artery calcification (Hamer et al., 2010), hypertension (Hamer & Steptoe, 2012) and cardiovascular disease risk (Girod & Brotman, 2004). Accordingly, the increased stress reactions observed in Type D individuals when exposed to situations high in social evaluation may increase their risk of cardiovascular disease via two mechanisms. Similarities between the increased stress reactivity in Type D individuals under social evaluative stress can also be made with the concept of interpersonal sensitivity; a stable trait characterized by ongoing concerns about negative social evaluation (Marin & Miller, 2013). Individuals high in interpersonal sensitivity are vigilant and sensitive to others' evaluation, and consequently adopt defensive behaviours such as SI to avoid negative social evaluation. Indeed, like Type D personality, a recent meta-analysis has demonstrated individuals with high interpersonal sensitivity may be at increased risk of cardiovascular disease (Marin & Miller, 2013).

On the other hand, the tendency for Type D individuals to show a pattern of diminished blood pressure reactions to asocial stress exposure could be seen as counterbalancing and

protective. However, there are two arguments against such a proposition. First, most of the stressors that people face in life are social rather than asocial (Dickerson et al., 2004; Kamarck et al., 2005). Second, it has recently become clear that diminished or blunted stress reactivity is far from benign (Phillips et al., 2013); blunted cardiovascular and cortisol reactivity has been associated with abdominal adiposity and obesity (Carroll et al., 2008; Phillips et al., 2012), depression and symptoms of depression (Brindle et al., 2013; de Rooij et al., 2010; Phillips et al., 2011; Salomon et al., 2013; Schwerdtfeger & Rosenkaimer, 2011) and smoking (Kirschbaum et al., 1993b; Phillips et al., 2009), all also regarded as risk factors for cardiovascular disease. Accordingly, what may be important in the case of Type D individuals is the departure of their reactivity profile from the norm, i.e., their facility to exhibit extreme responses, inclinations towards blunted when faced with asocial stress exposures and exaggerated when faced with stressors high in social evaluation. It should be noted, however, that only DBP was significantly lower in the asocial condition for Type D individuals therefore caution should be taken when making comparisons to the associations between the potential blunting and adverse outcomes. Nevertheless, this explanation very much fits with recent attempts to expand our understanding of the role of individual differences in stress reactivity that suggest that both extremes in the distribution may reflect dysregulation of the SAM and HPA systems and have adverse consequences for health (Carroll et al., 2009; Lovallo, 2011). In short, the question now is not whether Type D individuals do or do not show exaggerated biological reactions to stress; rather, it is more about delineating the conditions in which they show either exaggerated or blunted stress reactions, both of which may contribute to an adverse disease risk profile. From another perspective, if Type D personality is considered to be a continuous variable, then it may

actually be most adaptive around a mid-point, with high or low extremes representing the maladaptive tails of a normal distribution.

The present study is not without limitations. First, although the Type D personality construct has classically been treated as a dichotomised variable, based on a median split on the DS14 subscales (Denollet, 2005), more recent research has suggested that Type D personality may be best viewed as continuous rather than categorical (Ferguson et al., 2009; Grande et al., 2012; Smith, 2011). For example, a meta-analysis reported that the use of a categorical approach may have over-estimated the prognostic value of Type D (Grande et al., 2012). However, it should be noted that recent research has reported that both the interaction of continuous NA and SI scores (continuous measure of Type D) and the Type D versus non-Type D classification defined by cut-off scores were both associated with an increased risk of cardiovascular events (Denollet et al., 2013). Similarly, a study examining stress reactivity found that using either a continuous or categorical measure of Type D personality produced analogous associations with cardiovascular reactions (Howard et al., 2011). A categorical approach can also reduce power and increases the risk of false positives (Cohen & Cohen, 1983), and so caution may be necessary in interpreting the results. However, a strength of the current study was the use of more stringent classification criteria than previous studies (Howard & Hughes, 2013; Williams et al., 2009), with ≥ 14 and ≤ 8 classifying Type D and Non-Type D groups, respectively; this would serve to limit the possibility of misclassification. Additionally, test-retest scores over an average 4-month period were $r = .93$ and $.92$, for the NA and SI scales, respectively, indicating strong stability. Regarding reduced power it should be noted that the current study included a larger sample size than previous studies of reactivity and Type D (Howard & Hughes, 2013; Howard et al., 2011; Williams et al., 2009). Future research may consider the use of a

continuous Type D component. Second, it may have been informative to analyse the separate effects of NA and SI, or the NA x SI interaction term on reactivity (Ferguson et al., 2009; Habra et al., 2003; Kupper & Denollet, 2007) but clearly the current design precludes this. In the present study, the NA and SI subscales for the selected participants were highly correlated both at screening ($r = .92, p < .001$) and in the laboratory ($r = .85, p < .001$). Accordingly, determining the separate and interactive contributions of NA and SI to reactivity differences was not possible. Nevertheless, it would be informative for future research to assess the NA x SI interaction and the first order effects of NA and SI on stress reactivity. Third, the associations between Type D personality and adverse disease outcomes has received some criticism, citing failures to replicate earlier results and the likelihood of confounding (de Voogd et al., 2012; Grande et al., 2012; Smith, 2011). It should be noted, however, that the current study controlled for a number of potential confounders including depression. Fourth, it could be argued that the manipulations in the social stress condition, i.e. presence of evaluators, scoring, and live recording, may have also increased factors such as objective self-awareness and competition which may have contributed to the differential reactivity profiles. However, the participants in the social condition rated it as significantly more socially-evaluative ($p < .001$) and research has shown that social evaluative threat *per se* often includes elements of self-awareness and competition (Gruenewald et al., 2004). Fifth, we employed a between-subject design whereby Type D and non-Type D individuals were assigned to either a social or asocial condition. This was purposive as we wanted to avoid the risk of habituation of reactivity that may have arisen from subjecting the same participants to two stress exposures (Howard & Hughes, 2013; Pruessner et al., 1997). Further, participants' DS14 scores and demographics were matched according to condition

assignment and, importantly and consequentially, there were no group x condition interactions for any of these variables.

In summary, the present analyses indicated that Type D individuals exhibit exaggerated cardiovascular and cortisol reactivity under conditions of high socially evaluative threat, but appear to have somewhat blunted cardiovascular responses in asocial stress conditions.

These results help resolve some of the previous inconsistencies in the literature and suggest Type D individuals may exhibit exacerbated or attenuated stress reactions depending on the social evaluative nature of the stressor. Thus, dysregulation of the SAM system and HPA-axis in either direction during stress exposure may contribute to the association between Type D personality and increased cardiovascular disease risk.

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CHAPTER FOUR

**PROBLEMATIC INTERNET USE, EXCESSIVE ALCOHOL CONSUMPTION,
THEIR COMORBIDITY AND CARDIOVASCULAR AND CORTISOL
REACTIONS TO ACUTE PSYCHOLOGICAL STRESS IN A STUDENT
POPULATION**

ABSTRACT

Problematic Internet use and excessive alcohol consumption have been associated with a host of maladaptive outcomes. Further, low (blunted) cardiovascular and stress hormone (e.g. cortisol) reactions to acute psychological stress are a feature of individuals with a range of adverse health and behavioural characteristics, including dependencies such as tobacco and alcohol addiction. The present study extended this research by examining whether behavioural dependencies, namely problematic Internet use, excessive alcohol consumption, and their comorbidity would also be associated with blunted stress reactivity. A large sample of university students (N= 2313) were screened using Internet and alcohol dependency questionnaires to select four groups for laboratory testing: comorbid Internet and alcohol dependence (N=17), Internet dependence (N=17), alcohol dependence (N=28), and non-dependent controls (N=26). Cardiovascular activity and salivary cortisol were measured at rest and in response to a psychological stress protocol comprising of mental arithmetic and public speaking tasks. Neither, problematic Internet behaviour nor excessive alcohol consumption, either individually or in combination, were associated with blunted cardiovascular or cortisol stress reactions. It is possible that problematic Internet behaviour and excessive alcohol consumption in a student population were not related to physiological reactivity as they may not reflect ingrained addictions but rather an impulse control disorder and bingeing tendency. The present results serve to indicate some of the limits of the developing hypothesis that blunted stress reactivity is a peripheral marker of the central motivational dysregulation in the brain underpinning a wide range of health and behavioural problems.

Keywords: Internet dependence; Alcohol; Comorbid dependence; Cardiovascular reactivity; Cortisol reactivity; Acute stress

INTRODUCTION

The Internet has undoubtedly provided numerous benefits, but there is a growing concern that some individuals are displaying dependent behaviour, with the prevalence of dependency increasing within many countries including the United States of America and the United Kingdom (Durkee et al., 2012). Indeed, problematic and excessive Internet use has many characteristics in common with substance addiction: disturbed psychological functioning, inability or unwillingness to reduce use with potential relapse, and increased tolerance (Griffiths, 1996; Young, 1996). There is also evidence that excessive Internet use disrupts social, occupational, and personal relationships (Greydanus & Greydanus, 2012; Morrison & Gore, 2010), with support to suggest associations with alcohol misuse (Ko et al., 2008). High alcohol intake during adolescence has been linked to a range of long-term adverse outcomes such as increased anti-social behaviour (Swahn et al., 2004), mental health disorders (Marmorstein, 2009), relationship problems (Odgers et al., 2008), and life-long impaired cognitive function (Ehlers & Criado, 2010; Hanson et al., 2011). Thus, both excessive Internet and alcohol use appear to be associated with a host of maladaptive outcomes, with previous health research often showing comorbid conditions, as opposed to single disorders, are related to poorer health outcomes (Vogeli et al., 2007).

Psychological stress is experienced by most individuals on a daily basis, and it is now known that individuals differ markedly in their biological reactions to a standard psychological stress exposure (Carroll, 1992). Substantial evidence demonstrates that those who exhibit exaggerated cardiovascular and stress hormone (e.g. cortisol) reactions to acute psychological stress are at increased risk of developing various manifestations of cardiovascular disease (Carroll et al., 2012; Hamer et al., 2010). More recently, however, a range of evidence is accumulating that individuals who show blunted or low cardiovascular

and cortisol reactions, are at increased risk from a number of negative health and behavioural outcomes, such as depression (Brindle et al., 2013; de Rooij et al., 2010; Phillips et al., 2011b) and obesity and adiposity (Carroll et al., 2008; Phillips et al., 2012; Singh & Shen, 2013).

Even more striking is the association between blunted stress reactivity and tobacco, alcohol, and other substance dependences. For example, there is now a reasonable consensus that smokers are characterised by both blunted cardiovascular (Evans et al., 2012; Girdler et al., 1997; Roy et al., 1994; Sheffield et al., 1997; Straneva et al., 2000) and cortisol (al'Absi et al., 2003; Kirschbaum et al., 1994; Kirschbaum et al., 1993b; Rohleder & Kirschbaum, 2006; Stemmler & Meinhardt, 1990) stress reactivity and that this cannot be accounted for by the temporary abstinence usually required in stress testing protocols (al'Absi et al., 2003; Roy et al., 1994). Similarly, those dependent on alcohol (Bernardy et al., 1996; Dai et al., 2007; Errico et al., 1993; Lovallo et al., 2000; Panknin et al., 2002; van Leeuwen et al., 2011), other non-prescription drugs or both (Lovallo et al., 2000; Panknin et al., 2002; van Leeuwen et al., 2011) have also been found to show blunted biological responses to a range of stress tasks. Furthermore, diminished cardiovascular and cortisol reactions to stress would appear to be a feature of individuals with behavioural addictions and disorders such as bulimia (Ginty et al., 2012; Koo-Loeb et al., 1998; Monteleone et al., 2011; Pirke et al., 1992), exercise dependence (Heaney et al., 2011), and gambling addictions (Paris et al., 2010). This suggests that blunted reactivity is a general feature of dependencies, including behavioural addiction, and not specific to those that involve the abuse of a substance. Finally, there is at least preliminary evidence that those with co-existing substance dependencies are more likely to show blunted cardiovascular and cortisol responses to an

acute stress task than those with single dependencies (Bernardy et al., 1996; Errico et al., 1993; Panknin et al., 2002).

It has been argued that the link between these diverse correlates, including dependency and addiction, of blunted stress reactivity is deficiencies in motivation. Indeed, low cardiovascular and cortisol reactions to acute stress have been considered a peripheral marker for central motivational dysregulation (Carroll et al., 2009; Carroll et al., 2011; Lovallo, 2011), i.e., dysregulation of the neural systems in the brain that support motivation and goal-directed behaviour. Evidence in support comes from functional Magnetic Resonance Imaging studies. For example, individuals characterised by blunted cardiovascular stress responses have been found to be characterised by diminished activation in both the posterior and anterior cingulate cortex, and in the amygdala during stress exposure (Gianaros et al., 2005; Ginty, 2013). These brain areas are indeed implicated in motivational processes, goal-directed behaviour, and autonomic nervous control (Bush et al., 2000; Hagemann et al., 2003; Lovallo, 2005).

However, to date, problematic Internet behaviour has received no attention in the context of stress reactivity, nor has the comorbidity of behavioural and substance dependence, e.g., problematic Internet behaviour and alcohol use, with few studies investigating alcohol use and stress reactions within student samples (Evans et al., 2012; Wemm et al., 2013). Thus, the present study was designed to assess whether individuals with problematic Internet use and alcohol consumption, and therefore possible deficiencies in central motivation, would be characterised by diminished stress reactivity. Accordingly, a substantial student population was screened to identify individuals who appear to have comorbid Internet and alcohol dependence, Internet dependence *per se*, alcohol dependence *per se*, and individuals showing

no signs of dependence. Group differences in cardiovascular and cortisol reactions to acute psychological stress were then compared. We hypothesized that those with signs of dependence, whether to alcohol or the Internet, would show blunted stress reactivity, with this blunted reactivity being a particular feature of those with comorbid Internet and alcohol problems.

METHODS

Participants

Questionnaires measuring Internet and alcohol dependence were administered to 2313 University of Birmingham students (1556 women). On the basis of questionnaire responses, a sub-sample of 88 participants were selected to form four groups who attended laboratory testing. Table 4.2 shows the N in each group and scores on the Internet and alcohol dependence scales used in screening and those used subsequently to confirm status. The mean (SD) age of the selected sample was 19.8 (1.84) years and their mean (SD) body mass index was 22.3 (3.04) kg/m². Fifty-eight (66%) participants were female, the majority of the participants indicated they were “white” (86%) and five individuals smoked. Demographics and health behaviours are presented in Table 4.2. The groups did not vary in any of these characteristics with the exception of body mass index, which was significantly lower in the control group, and, as would be expected, alcohol consumption which was greatest in the comorbid and alcohol dependent groups. All participants provided written informed consent and the study was approved by the University of Birmingham ethics committee.

Table 4.1. Responses to the Internet Addiction Test (IAT), Alcohol Use Disorders Identification Test (AUDIT), Pathological Internet Use Scale (PIUS) and the Shorter PROMIS Questionnaire (SPQ) alcohol sub-scale

	Comorbid	Internet dependent	Alcohol dependent	Control	
	Mean (SD)				<i>p</i>
IAT	62.9 (8.47)	57.2 (4.25)	31.9 (4.86)	28.6 (4.00)	<.001
AUDIT	18.9 (4.32)	6.5 (3.36)	20.0 (4.14)	2.9 (1.79)	<.001
PIUS	6.4 (2.27)	5.4 (2.45)	2.4 (1.79)	1.7 (1.25)	<.001
SPQ alcohol sub-scale	27.8 (7.54)	16.4 (8.33)	28.4 (8.17)	10.8 (8.22)	<.001

Table 4.2. Characteristics of the comorbid, Internet dependent, alcohol dependent, and control groups

	Comorbid	Internet dependent	Alcohol dependent	Control	
	Mean (SD)/ N (%)				<i>p</i>
N	17 (19)	17 (19)	28 (32)	26 (30)	
Gender (females)	13 (77)	12 (71)	15 (54)	18 (69)	.38
Age (years)	19.6 (1.80)	19.8 (1.29)	19.2 (.67)	20.5 (2.94)	.11
Body Mass Index (kg/m ²)	22.3 (2.32)	23.7 (4.40)	23.6 (2.67)	20.4 (2.36)	.001
Ethnicity (white)	14 (82)	14 (82)	27 (96)	21 (81)	.73
Current smoker	2 (12)	0 (0)	1 (4)	2 (8)	.46
Units of alcohol per week (≥ 11)	5 (29)	2 (12)	18 (64)	1 (4)	<.001
Sleep (≥ 8 hrs)	8 (47)	6 (35)	14 (50)	14 (54)	.68
Calculated cardio-respiratory fitness (metabolic equivalents)	13.8 (1.49)	13.4 (1.80)	14.3 (1.58)	13.9 (1.57)	.31

Measures

Internet dependence

The Internet Addiction Test (Young, 1998) was used to screen for pathological Internet use. Scores from 20 to 49 indicate average online use, 50 to 79 indicate frequent problems due to Internet usage, and 80 to 100 represent Internet use causing significant problems (Young, 1998). High internal reliability was found in the present study, Cronbach's $\alpha = .94$. The Pathological Internet Use Scale (Morahan-Martin & Schumacher, 2000) was used to confirm group allocation. A total of ≥ 4 , 1-3, and 0 indicate, respectively, pathological Internet use, limited symptoms, and no symptoms. Current internal reliability was good, $\alpha = .73$.

Alcohol dependence

The Alcohol Use Disorders Identification Test (Saunders et al., 1993) was used to screen for alcohol dependence. A score of ≥ 15 or ≥ 13 for men and women, respectively, indicates alcohol dependence, and a score of ≥ 8 indicates harmful or hazardous drinking (Saunders et al., 1993). The current Cronbach's $\alpha = .88$. The alcohol sub-scale of the Shorter PROMIS Questionnaire (Christo et al., 2003) was used to check group allocation. Total scores range from 0 to 50, with higher scores indicating greater dependence. The present study found a Cronbach's $\alpha = .87$.

Health behaviours

A questionnaire adapted from the Whitehall II study (Marmot et al., 1991) was administered to measure average daily smoking, weekly alcohol intake, and sleep duration. Estimated cardio-respiratory fitness was calculated by a previously validated formula (Jurca et al., 2005).

Psychological stress task questionnaire.

Following stress task completion, participants rated how difficult, stressful, exciting, confusing, and engaging they found the tasks, and how well they thought they performed. Responses were recorded on Likert-type scale ranging 0, not at all to 6, extremely.

Acute psychological stress tasks

The 10-min Paced Auditory Serial Addition Test (Gronwall, 1977) has demonstrated reliability in perturbing both cardiovascular and salivary cortisol reactions (Ginty et al., 2012a; Ring et al., 2002). Participants are presented with a series of single digits and are required to add the present number to the previously presented number, and report their answer aloud. The protocol has been described in detail elsewhere (Heaney et al., 2011).

The speech task required participants to deliver two consecutive speeches, each lasting 6 min comprising 2 min preparation and 4 min delivery (Bosch et al., 2003). The task has been shown to reliably elicit both cardiovascular and cortisol responses (Bosch et al., 2009). To increase social stress, each speech was performed in the presence of two experimenters, one of whom prominently observed and prompted the participant to continue if they paused. The first speech required the participants to defend themselves following a false shop-lifting accusation, whereas the second speech was a presentation of their best and worst personal characteristics.

Cardiovascular and salivary cortisol measures

The laboratory session consisted of five periods; 10 min adaptation, 10 min baseline, 10 min Paced Auditory Serial Addition Test, 15 min speech stress task, and 20 min recovery. Systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate (HR) were measured discontinuously using a semi-automatic sphygmomanometer (Critikon Inc, Tampa, FL/Omron, IL) at min 2, 4, 6, and 8 during baseline, Paced Auditory Serial Addition

Test and recovery, and at 30 sec and 2 min 30 sec into delivery phase of each speech. Two stimulated 2 min saliva samples were obtained using salivettes at min eight of baseline and eight min into the recovery period. Salivettes were centrifuged for 5 min at 4000rpm before being stored at -20°C until assay. ELISA kits (IBL International, Germany) were used to analyse all cortisol samples in duplicate. The mean intra-assay coefficient of variation was 9.8% and the inter-assay coefficient was 4.4%. Due to collection difficulties with two participants (one Internet dependent and one control), cortisol assays were analysed for 86 participants.

Procedure

Alcohol dependence was defined as an Alcohol Use Disorders Identification Test score ≥ 15 for men and ≥ 13 for women. Internet dependence was signified by an Internet Addiction Test score ≥ 53 . The comorbid group met both of these criteria. The control group scored < 8 on the Alcohol Use Disorders Identification Test, and between 20-35 on the Internet Addiction Test. An Internet Addiction Test score of ≥ 53 was adopted as this was greater than the 50-79 criteria proposed to reflect Internet use causing frequent problems (Young, 1998), and was more stringent than cut-offs which have demonstrated sound psychometric properties (Widyanto & McMurrin, 2004) and criteria used by previous studies examining problematic Internet use (Hardie & Tee, 2007; Lam & Peng, 2010). Thus, the current study adopted a cut-off which incorporated individuals with problematic Internet use from the substantial screening of over 2300 students. Further, 20-35 was lower than the suggested criteria of < 49 indicating average online use, therefore providing further support for stringent group allocation.

Prior to laboratory testing, participants abstained from eating for 1 h, drinking caffeine or smoking for 2 h, and from physical exercise and drinking alcohol for 12 h. During the adaptation period, participants completed the Pathological Internet Use Scale and Shorter PROMIS Questionnaire whilst sat quietly. This was followed by the formal resting baseline before the psychological stress tasks were presented in a counterbalanced order, with the subsequent recovery period.

Statistical Analysis

For the cardiovascular measures, averages of each period were calculated; baseline, stress (combined average of Paced Auditory Serial Addition Test and Speech), and recovery. Salivary cortisol measures were log₁₀ transformed. Group differences in demographic, health behaviour, dependency, stress task perceptions and performance, and baseline cardiovascular and cortisol variables were tested using one-way ANOVA for continuous and chi-square for categorical variables. Repeated measures ANOVAs were performed to confirm the stress tasks perturbed cardiovascular activity and cortisol, and to examine group differences over time. Greenhouse-Geisser correction was applied where appropriate and pairwise comparisons undertaken to elucidate significant differences. Partial η^2 was reported as an index of effect size throughout.

RESULTS

Questionnaires and task performance scores

As indicated in Table 4.1, average scores on the Internet and alcohol dependence scales significantly differentiated the groups. As expected, the comorbid and Internet dependent groups reported significantly higher scores on the Internet Addiction Test and Pathological Internet Use Scale than the alcohol dependent and control groups ($p < .001$). The comorbid and alcohol dependent groups had significantly higher scores on the Alcohol Use Disorders Identification Test and Shorter PROMIS Questionnaire alcohol sub-scale than the Internet dependent and control groups ($p < .05$). There were no significant group differences in Paced Auditory Serial Addition Test performance or self-reported task ratings: see Table 4.3.

Table 4.3. Subjective stress task ratings and Paced Auditory Serial Addition Test performance

	Overall	Comorbid	Internet dependent	Alcohol dependent	Control	
	Mean (SD)					<i>p</i>
Difficulty	4.4 (.97)	4.3 (1.05)	4.2 (.66)	4.4 (1.07)	4.4 (1.02)	.92
Stressful	4.4 (1.04)	4.3 (1.00)	4.6 (.87)	4.2 (1.23)	4.5 (.99)	.61
Exciting	2.6 (1.54)	2.4 (1.77)	2.8 (1.55)	2.7 (1.31)	2.6 (1.68)	.89
Perceived performance	2.4 (1.19)	2.4 (1.33)	2.4 (1.23)	2.5 (1.23)	2.2 (1.07)	.87
Confusing	2.7 (1.54)	3.1 (1.30)	2.3 (1.40)	3.1 (1.65)	2.3 (1.57)	.16
Engaging	3.8 (1.36)	3.8 (1.38)	3.8 (1.44)	3.8 (1.38)	3.7 (1.35)	.99
Paced Auditory Serial Addition Test total score	715.7 (138.83)	656.3 (119.59)	750.3 (76.64)	722.0 (148.57)	722.9 (164.15)	.25

Cardiovascular and cortisol reactions to acute psychological stress

The stress tasks significantly perturbed cardiovascular activity. ANOVA revealed a significant time effect for SBP, $F(2, 168) = 253.30, p < .001, \eta^2 = .751$, DBP, $F(2, 168) = 223.08, p < .001, \eta^2 = .726$, and HR, $F(2, 168) = 154.91, p < .001, \eta^2 = .648$, whereby all variables increased significantly from baseline to stress then decreased during recovery but remained significantly higher than baseline: indicated in Figure 4.1. There were no main effects of group overall for SBP, DBP, or HR; $F(3, 84) = 2.28, p = .08, \eta^2 = .075$; $F(3, 84) = .76, p = .52, \eta^2 = .026$; $F(3, 84) = 1.58, p = .20, \eta^2 = .053$, respectively, nor were there any significant group differences in baseline cardiovascular levels. There were no significant group x time interactions for SBP, $F(6, 168) = .44, p = .83, \eta^2 = .015$, DBP, $F(6, 168) = .87, p = .52, \eta^2 = .030$, or HR, $F(6, 168) = .47, p = .77, \eta^2 = .016$: see Figure 4.1. Further analyses examining cardiovascular differences between the comorbid and control groups specifically produced analogous results such that there were no significant group x time interactions for SBP, $F(2, 82) = .25, p = .78, \eta^2 = .006$, DBP, $F(2, 82) = 1.41, p = .25, \eta^2 = .033$, or HR, $F(2, 82) = .51, p = .54, \eta^2 = .012$.

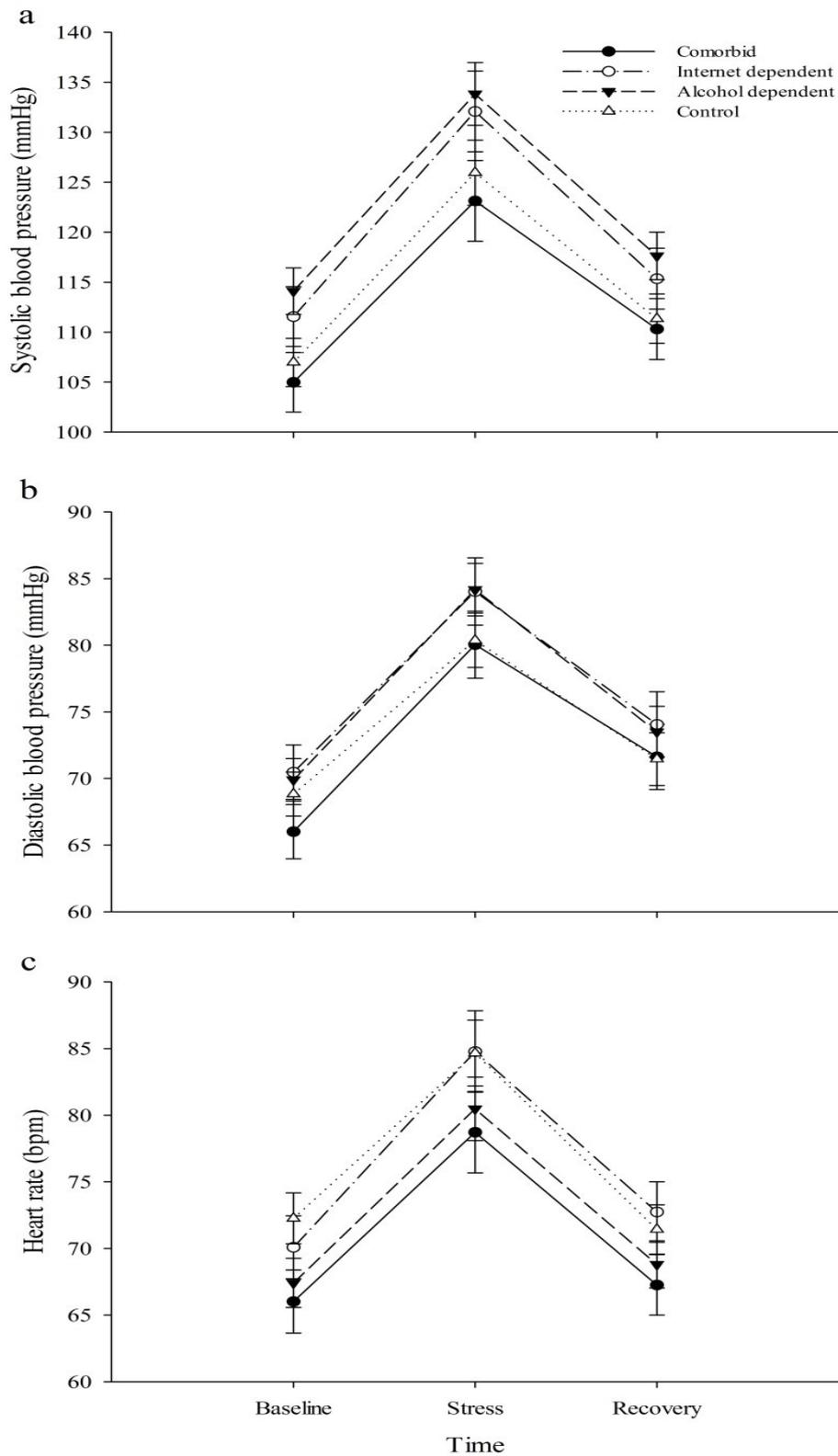


Figure 4.1. Mean (SE) (a) systolic blood pressure, (b) diastolic blood pressure, and (c) heart rate levels at baseline, during the acute stress task, and recovery by group

The stress tasks significantly increased cortisol as shown in Figure 4.2. Repeated measures ANOVA revealed a significant effect of time, $F(1, 82) = 4.35, p = .04, \eta^2 = .050$, but no main effects of group overall, $F(3, 82) = 1.52, p = .22, \eta^2 = .053$, or at baseline only. There was also no significant group x time interaction, $F(3, 82) = .38, p = .77, \eta^2 = .014$. Further analysis examining the cortisol differences between the comorbid and control groups only also revealed no group x time interaction, $F(1, 40) = .33, p = .57, \eta^2 = .008$.

As body mass index was significantly lower in the control group, and has been shown to relate to reactivity (Carroll et al., 2008; Phillips et al., 2012) the above analyses were repeated with body mass index as a covariate; this did not alter the outcomes.

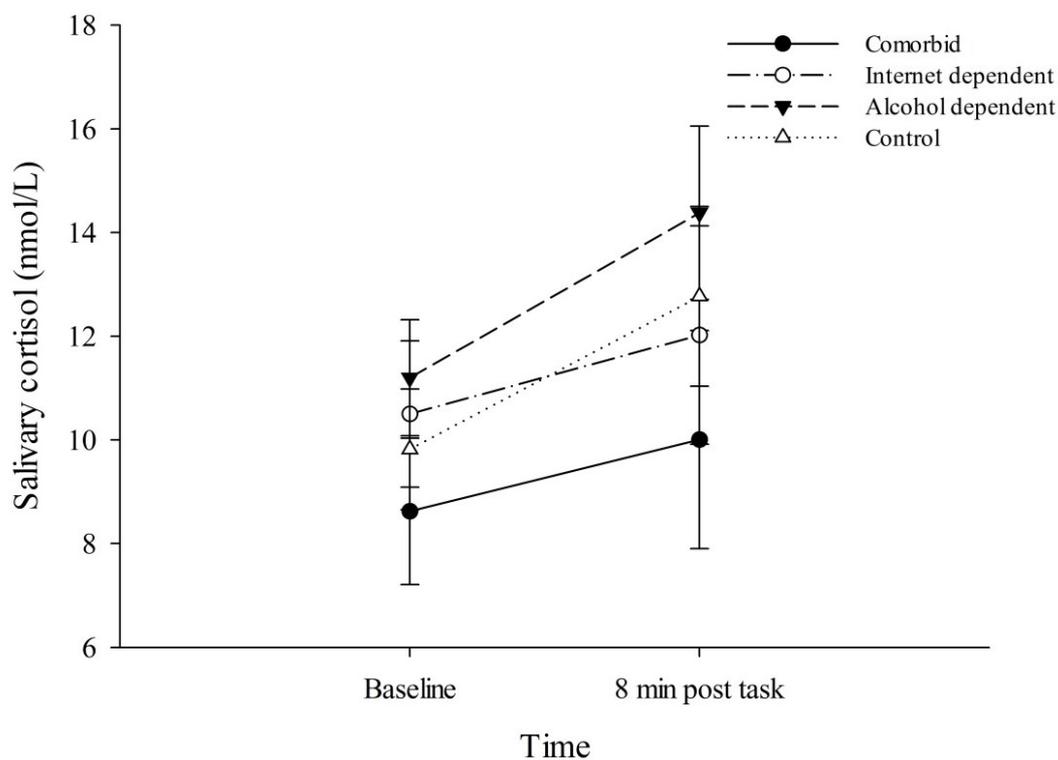


Figure 4.2. Mean (SE) salivary cortisol at baseline and 8 min post stress task by group (raw values shown)

DISCUSSION

The present study was concerned with whether problematic Internet behaviour and possible alcohol dependence on their own, and in combination, were associated with blunted biological reactions to acute psychological stress exposures. Neither of these behaviours individually nor when co-existing were associated with either cardiovascular or cortisol stress reactivity, such that there were no differences in reactivity between any of the groups compared to controls. These null results would appear to indicate that blunted stress reactivity is only present in those with life-long confirmed addictions rather than extremes of student behaviour. For example, blunted stress reactivity has been shown in individuals with tobacco (al'Absi et al., 2003; Evans et al., 2012; Girdler et al., 1997; Kirschbaum et al., 1994; Kirschbaum et al., 1993b; Rohleder & Kirschbaum, 2006; Roy et al., 1994; Sheffield et al., 1997; Straneva et al., 2000), alcohol (Bernardy et al., 1996; Dai et al., 2007; Errico et al., 1993; Lovallo et al., 2000; Panknin et al., 2002; Sinha et al., 2011), and other substance dependencies (Lovallo et al., 2000; Panknin et al., 2002; van Leeuwen et al., 2011), as well as those with behavioural dependence such as disordered eating (Ginty et al., 2012; Koo-Loeb et al., 1998; Monteleone et al., 2011; Pirke et al., 1992), exercise dependence (Heaney et al., 2011), and pathological gambling (Paris et al., 2010). It should be acknowledged that the potential null effects may be due to limited statistical power, thus a larger scale study may indeed reveal group differences in stress reactivity.

This is the first study we know of to examine problematic Internet behaviour in the context of stress reactivity. Internet addiction is a relatively new construct (Durkee et al., 2012), and remains a heterogeneous one (Griffiths, 2000; Widyanto & Griffiths, 2006; Young, 1999), with no agreed definition (Greydanus & Greydanus, 2012). For example, although excessive Internet use has been associated with many of the characteristics of substance addiction such

as disturbed psychological functioning, inability or unwillingness to reduce use, and increased tolerance (Griffiths, 1996; Young, 1996), there is little evidence of the physical symptoms of withdrawal, typical of substance dependence (Beard & Wolf, 2001). Accordingly, many have argued that Internet addiction is a misnomer (Griffiths, 2000; Weinstein & Lejoyeux, 2010; Widyanto & Griffiths, 2006), reflecting an impulse control disorder (Young, 1998). In support, Cao et al. (2007) reported that, in comparison to controls, adolescents with excessive Internet use exhibited greater levels of impulsivity as indicated by their responses to an impulsivity questionnaire and to the GoStop impulsivity paradigm. Further evidence has been provided by neurophysiological studies showing that those who use the Internet excessively are characterised by electroencephalographic signs of poor response inhibition and impulsiveness (Choi et al., 2013; Dong et al., 2012). In retrospect, then, if problematic Internet behaviour, in general and in the present study, does not constitute an actual long-term addiction, but more of a milder dependency, it is perhaps not surprising that we failed to find an association with blunted stress reactions.

Previous research linking high alcohol consumption with blunted biological stress reactivity has focused on adults with a confirmed diagnosis (Bernardy et al., 1996; Dai et al., 2007; Errico et al., 1993; Lovallo et al., 2000; Panknin et al., 2002). For example, Lovallo et al. (2000) reported alcoholics aged between 22 and 55 failed to elicit a significant increase in cortisol in response to a public speaking stressor, whereas non-dependent controls showed the expected rise, with Panknin et al. (2002) also demonstrating the same dependent patients elicited attenuated heart rate responses. Dai et al. (2007) reported, in response to arithmetic and monetary competition stressors, alcohol dependent individuals, aged 25 to 40, failed to stimulate a significant increase in cortisol while non-dependent individuals produced the expected increase. All of these studies classified their dependent patients according to the

Diagnostic and Statistical Manual-IV criteria for alcohol dependence (APA, 1994). The present study purposively attempted to extend this work by focusing upon alcohol consumption within a student population, especially given the paucity of research of students within this context and the association of excessive alcohol intake in this population with a range of maladaptive outcomes such as anti-social behaviour (Swahn et al., 2004), mental health disorders (Marmorstein, 2009), relationship problems (Odgers et al., 2008), and life-long impaired cognitive function (Brown et al., 2008). Our finding of no association between students with high levels of alcohol consumption and either cardiovascular or cortisol stress reactions suggest limits to the scope of the proposal that blunted stress reactions are characteristic of a range of problematic behaviours including alcohol dependence (Carroll et al., 2009; Carroll et al., 2011; Lovallo et al., 2000). However, it is possible that the present student sample were not truly addicted to alcohol. There is a range of evidence that as a body, students consume more alcohol than their non-student peers, and that alcohol consumption rises on transition from school to university (Dawson et al., 2004; Kypri et al., 2005). However, it is also clear from previous research that alcohol consumption decreases on graduation (Bewick et al., 2008; Klein, 1994; Lanza & Collins, 2006). Accordingly, excessive alcohol consumption by students may be a transient phenomenon rather than a reflection of a true addiction. Again, it should be acknowledged that that increased statistical power may indeed reveal group differences in stress reactivity.

It is worth noting that the two studies to examine biological stress reactivity in the context of high alcohol consumption in a younger sample, found no associations between alcohol intake and cardiovascular stress reactions (Evans et al., 2012; Wemm et al., 2013).

However, in line with the results from epidemiological studies of adult populations (Ginty et al., 2014; Phillips et al., 2009b; Sheffield et al., 1997), Evans et al. (2012) did find an

association between tobacco smoking and blunted heart rate reactivity. This would be consistent with the contention that early smoking is much more reflective of addictive behaviour than early excessive alcohol consumption (Breslau, 1995; Hu et al., 2006; Lanza & Collins, 2006) and that blunted stress reactivity is a marker for true addiction rather than behavioural excess. The studies demonstrating the associations between both disordered eating (Ginty et al., 2012) and exercise dependence (Heaney et al., 2011), with low physiological stress reactivity, in a similar student population as used in the present study, add further support to the notion that true addictions are required to reflect blunted biological responses especially given that these constructs are proposed to incorporate more pathological components (Bamber et al., 2000; Karim & Chaudhri, 2012; Robbins & Joseph, 1985) and are likely to persist into adulthood (Allen et al., 2013; Berczik et al., 2012).

Given the absence of previous research, a primary aim of the present study was to examine the association between comorbid behavioural and substance dependence, i.e. problematic Internet behaviour and excessive alcohol consumption, and cardiovascular and cortisol reactivity. Others have found that alcohol abuse is quite common among those that use the Internet excessively (Ko et al., 2008), with comorbid conditions often related to poorer health outcomes in comparison to single disorders (Vogeli et al., 2007). Our clear expectations, based on studies which showed individuals who had comorbid alcohol and substance dependency had the greatest blunting of heart rate (Panknin et al., 2002) and cortisol (Bernardy et al., 1996; Errico et al., 1993) responses relative to those who had single or no addictions, was that the group exhibiting both excessive Internet use and alcohol intake would show the most diminished stress reactivity. In reality, this group did not differ from single addiction groups or participants with low Internet use and alcohol consumption.

Given that it is unlikely that excessive Internet use in the present study constituted a genuine

addiction, and it is unlikely that our high alcohol consumers were actual alcoholics, it is perhaps not surprising that this particular comorbidity of problematic behaviours was not associated with blunted stress reactivity. Again, this would seem to reinforce the conclusion that blunted reactivity may signify genuine addiction rather than excessive behaviour.

It should be acknowledged that the present study had the following limitations. Firstly, although our cut off for Internet dependency was slightly lower than the very highest criteria band suggested by Young (1998), it still incorporated individuals with problematic Internet use, and was more stringent than criteria which has demonstrated sound psychometric properties (Widyanto & McMurrin, 2004) and criteria used in other studies (Hardie & Tee, 2007; Lam & Peng, 2010). Further, according to both the screening and follow-up validation dependency questionnaires, the groups significantly differed in the expected direction on the questionnaires used, thus suggesting correct group assignment. Secondly, the Internet Addiction Test (Young, 1998) and the Pathological Internet Use Scale (Morahan-Martin & Schumacher, 2000) were created before the invention and increased use of social media via the Internet, and could therefore be deemed out-dated or inadequate in for measuring Internet dependency. Nevertheless, both of these scales have been used in numerous studies of Internet addiction (Hardie & Tee, 2007; Lam & Peng, 2010; Laconi et al., 2014) and have reported good psychometrics (Laconi et al., 2014; Widyanto & McMurrin, 2004). Finally, the final size of each group could be considered modest and may have reduced the statistical power to find group differences. However, it was similar or greater in size to previous studies in this broad field (Ginty et al., 2012; Heaney et al., 2011; Lovullo et al., 2000) and was based on a selection from a substantial screening sample of over 2300 university students.

In conclusion, neither problematic Internet behaviour nor excessive alcohol consumption, either individually or in combination, were associated with blunted cardiovascular or cortisol reactions to acute psychological stress. This result was contrary to our expectations based on growing evidence that blunted stress reactivity is associated with a host of adverse health and behavioural outcomes, including addiction. In retrospect, though, given the controversial nature of the concept of Internet addiction and the relatively short term, and possibly temporary, excessive alcohol intake, it is unlikely that our sample displayed genuine and ingrained addictions. Our study, therefore, appears to indicate that the hypothesis that blunted stress reactivity is a peripheral marker of the central motivational dysregulation underpinning a wide range of behavioural problems is restricted to more serious disorders rather than the possibly time-limited excessive behaviours studied here.

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CHAPTER FIVE

**BLUNTED CARDIAC STRESS REACTORS EXHIBIT RELATIVELY HIGH
LEVELS OF IMPULSIVITY: A CASE CONTROLLED DOUBLE BLIND
BEHAVIOURAL STUDY**

ABSTRACT

Blunted physiological reactions to acute psychological stress are associated with a range of adverse health and behavioural outcomes. The aim of the present study was to determine whether extreme stress reactors differ in their everyday behaviours. Individuals showing blunted ($N=23$) and exaggerated ($N=23$) cardiovascular reactions to stress were selected from screening of a healthy student population ($N=276$). Impulsivity, risk taking, and persistence were then measured using standard laboratory tasks. Blunted reactors exhibited greater impulsivity than exaggerated reactors on both stop-signal, $F(1,41) = 4.99, p = .03, \eta^2 = .108$, and circle drawing, $F(1,43) = 4.00, p = .05, \eta^2 = .085$, tasks. The two reactor groups did not differ significantly in risk taking or persistence. Individuals showing blunted cardiovascular stress reactions are characterized by greater impulsivity which may contribute to their increased susceptibility to outcomes such as obesity, depression, and addiction.

Keywords: Behavioural traits; Stress reactivity; Psychological stress; Impulsivity; Risk taking; Persistence.

INTRODUCTION

Individuals differ markedly in their biological reactions to standard psychological stress exposures (Carroll, 1992). There is also compelling cross-sectional and prospective evidence that those who exhibit exaggerated cardiovascular reactions to acute psychological stress are at increased risk of developing various manifestations of cardiovascular disease, such as hypertension (Carroll et al., 2012b; Carroll et al., 2003; Newman et al., 1999), systemic atherosclerosis (Everson et al., 1997; Matthews et al., 1998), left ventricular hypertrophy (Kapuku et al., 1999; Murdison et al., 1998), and coronary artery calcification (Matthews et al., 2006). They are also at increased risk of dying from cardiovascular disease (Carroll et al., 2012a).

Blunted or diminished cardiovascular stress reactions were, by implication, presumed to be benign or protective. There is, however, accumulating evidence that blunted biological stress reactions are associated with a range of adverse health and behavioural outcomes (Carroll et al., 2009; Carroll et al., 2011; Lovallo, 2011; Phillips et al., 2013). Diminished cardiovascular reactivity has been associated with obesity (Carroll et al., 2008; Phillips et al., 2012; Singh & Shen, 2013) and depression (Brindle et al., 2013; Carroll et al., 2007; Salomon et al., 2009; Schwerdtfeger & Rosenkaimer, 2011), and also with increased likelihood of becoming obese (Carroll et al., 2008; Phillips et al., 2012) or depressed (Phillips et al., 2011). Further, blunted stress reactivity has been associated with substance abuse addictions and dependencies. For example, there is now a reasonable consensus that smokers are characterized by blunted cardiovascular stress reactivity (Evans et al., 2012; Ginty et al., 2014; Girdler et al., 1997; Roy et al., 1994; Sheffield et al., 1997; Straneva et al., 2000). Similarly, those dependent on alcohol (Bernardy et al., 1996; Dai et al., 2007; Errico et al., 1993; Lovallo et al., 2000; Panknin et al., 2002; Sinha et al., 2011) and/or other non-

prescription drugs (Lovallo et al., 2000; Panknin et al., 2002; van Leeuwen et al., 2011) have been found to show blunted biological responses to a range of stress tasks. In addition, the non-dependent offspring of parents who are alcohol dependent also display attenuated stress reactivity (Moss et al., 1999; Sorocco et al., 2006), indicating blunted reactivity may predate, and thus be a risk marker for, dependence. Finally, diminished biological reactions to stress would appear to be a feature of those who meet the criteria for exercise dependence (Heaney et al., 2011), gambling addiction (Paris et al., 2010), and those with symptoms of bulimia (Ginty et al., 2012; Koo-Loeb et al., 1998). Overall, this suggests that blunted stress reactivity may be a general feature of dependencies, including behavioural dependency.

It has been argued that the link between these diverse outcomes and blunted stress reactivity stems from a dysfunction in motivational processes within the brain. Indeed, low cardiovascular reactions to acute stress have been considered a peripheral marker for central motivational dysregulation (Carroll et al., 2009; Carroll et al., 2011; Lovallo, 2011), i.e. dysregulation of the neural systems in the brain that support motivation, emotional regulation, and goal-directed behaviour. A conceptual model has been proposed whereby modified frontolimbic function of the brain leads to reduced stress reactivity, altered cognition, and unstable affect regulation which then leads to impulsive behaviours and risk taking, with consequences for adverse health behaviours and addiction risk (Lovallo, 2013). This would suggest that those with blunted stress reactions may exhibit particular behavioural characteristics such as high impulsivity and risk taking and lack of persistence. Addictive behaviour is often described as impulsive (Brewer & Potenza, 2008). Therefore, impulsivity would appear to be implicated in the addictions and negative behavioural outcomes associated with blunted reactivity. Few studies have examined the association

between impulsivity and cardiovascular/cortisol stress reactivity in non-clinical populations and the findings are somewhat mixed. Whereas two studies found that high impulsivity was related to low cardiac reactivity (Allen et al., 2009; Munoz & Anastassiou-Hadjicharalambous, 2011), one study reported an association in the opposite direction (Diller et al., 2011), with no clear association also observed (Mathias & Stanford, 2003). In one of the few studies to have included behavioural as well as self-report measures of impulsivity, pre-adolescent children high in impulsivity had diminished cardiac responses to a mental arithmetic task (Bennett et al., 2014).

As might be expected, research has shown that risk taking is also elevated in individuals with addictions such as alcohol (Lawrence et al., 2009), tobacco (Cavalca et al., 2013), other non-prescription substances (Schutter et al., 2011), as well as in problematic gamblers (Lawrence et al., 2009). Likewise, individuals with eating disorders (Fischer & le Grange, 2007) and exercise dependency (Freimuth et al., 2011) have also been shown to exhibit increased risk taking. In the context of biological stress reactivity, risk taking has attracted limited attention. To our knowledge, one published study to date has examined the relationship between cardiovascular stress reactivity and risk taking in adolescent boys (Liang et al., 1995). The authors found no association; however, the study used self-reported risk taking and not behavioural measures.

Persistence and perseverance are key components in achieving many goals in daily life, and are related to greater academic and career progress (Andersson & Bergman, 2011), and the ability to refrain from unhealthy behaviours (Quinn et al., 1996). Given that blunted stress reactivity may signal central motivational dysregulation, it is possible that attenuated reactivity may be associated with lower levels of persistence. Indeed, individuals with

attenuated physiological stress responses have been shown to be more likely to relapse during smoking (al'Absi et al., 2005) and alcohol (Adinoff et al., 2005; Lovallo, 2006) cessation, perhaps reflecting reduced persistence. To the best of our knowledge, however, no studies have directly examined the association between biological stress reactivity and persistence.

Given that impulsivity, risk taking and persistence are associated with the unhealthy outcomes linked to attenuated biological stress reactivity, we re-examined their relationship with cardiovascular stress reactivity. However, instead of the correlational approach used in previous studies, we adopted a case-control design, stress testing a substantial sample and then selecting sub-samples of unambiguously exaggerated and blunted cardiac reactors. The selected individuals then completed a series of behavioural tasks designed to measure impulsivity, risk taking, and persistence. We considered that such an approach, using a case-control design and using behavioural rather than self-report measures, would afford a more powerful test of the hypothesis that blunted stress reactors would be characterized by greater impulsivity and risk taking, and reduced persistence.

METHODS

Participants

Two hundred and seventy six healthy University of Birmingham students (147 women) attended an initial laboratory stress-testing session during which cardiovascular stress reactivity was determined. Using cut-offs of the 15% highest and 15% lowest heart rate (HR) reactions, 23 exaggerated reactors and 23 blunted reactors were selected and returned to complete a battery of behavioural tasks. The mean (SD) age of the selected sample was

22.6 (8.09) years and their mean (SD) body mass index (BMI) was 22.9 (2.91) kg/m². The majority of the selected participants indicated they were “white” (89%). None had a history of cardiovascular disease, a current illness or infection, or were taking medication. All participants provided written informed consent, and the study was approved by the University of Birmingham ethics committee and was in accordance with the Helsinki Declaration of 1975, as revised in 2000.

Cardiovascular Reactivity Screening Procedure

Individuals were invited, via email and an online recruitment facility, to attend a laboratory stress testing session. Participants were requested to refrain from eating for 1 hour, drinking caffeine or smoking for 2 hours, and from physical exercise and drinking alcohol for 12 hours, prior to testing. On arrival, participants’ provided basic demographic information and had their height and weight measured, and BMI subsequently calculated as weight/height². A blood pressure cuff was then attached to the participants’ non-dominant arm above the elbow. They then sat quietly during a 10 minute adaptation period, followed by a formal 10-minute resting baseline period, and then undertook a 10-minute Paced Auditory Serial Addition Test (PASAT), which served as the stress exposure. Systolic (SBP) and diastolic (DBP) blood pressure and HR were measured discontinuously using a semi-automatic sphygmomanometer (Omron, IL) at minutes 2, 4, 6 and 8 during baseline and stress. A single measure was also taken during the adaptation period for familiarity, although this measure was disregarded.

The PASAT (Gronwall, 1977) was presented via a compact disk player. The PASAT has been observed to reliably perturb cardiovascular activity (Ginty et al., 2012; Ring et al., 2002) and to show good test-retest reliability (Willemsen et al., 1998). Participants were

presented with a series of single digit numbers and required to add the present number to the previously presented number, and report their answer aloud. To increase potential stress, participants' answers were conspicuously scored and they were instructed to attempt to beat scores on a prominently displayed peer leader board, and that five points would be deducted for each wrong answer from the starting score of 1000. Participants were filmed and their image displayed live on a television monitor that they were requested to focus on. They were also told that the recording would be assessed by "body language experts", although no such assessment was undertaken. Additionally, during the last five numbers of each block of 10 numbers, and mostly corresponding with an error or hesitation, a brief burst of loud aversive noise was presented using a buzzer which participants were told coincided with an error on their part or looking away from the television screen or hesitating. All participants received 21 noise bursts. Immediately following the PASAT, participants rated their perceived level of task engagement and task stressfulness by using a 7-point Likert-type scales ranging from 0, not at all to 6, extremely.

Participants' cardiovascular reactivity was calculated as the mean PASAT level – mean baseline level for each of the cardiovascular variables. Individuals scoring within the top and bottom 15% of HR reactions were invited back to the laboratory to complete a series of behavioural tasks. HR reactivity was chosen as the selection criteria as previous research has shown that blunted HR reactivity is more consistently associated with the adverse health and behavioural outcomes (Bibbey et al., 2013; Ginty et al., 2012; Phillips, 2011).

Behavioural Tasks

Impulsivity

Stop-Signal Task. The stop-signal task (Inquisit by Millisecond, Seattle) is a computerized program designed to measure the inhibitory control aspect of impulsivity (Logan et al., 1997). The paradigm involves two concurrent tasks, a *go task* and a *stop task* (for further information on the stop-signal paradigm see Verbruggen and Logan (2009)). At the beginning of each trial a fixation sign is presented, which after 250 msec is replaced by the primary task stimulus (i.e. an arrow). Participants are presented with an arrow in the centre of the computer screen which points in either the left or right direction and are then required to respond as fast and accurately as possible by respectively pressing either the 'D' or 'K' key on a keyboard with their index finger; the *go task*. On 25% of the go task trials, an auditory beep (stop signal) is presented and participants are required to inhibit their response to the go task on that trial; the *stop task*. Depending on whether the stop task or the go task is completed first on the stop trials, the participant will either correctly inhibit their response or still continue press the button as if it were a go trial. Inhibitory control therefore depends on the latency of the response to the go signal (go reaction time) and the latency of the response to the stop signal (stop-signal reaction time). Consequently, failing to inhibit when presented with a stop signal indicates poor impulse control and greater impulsivity (Logan et al., 1997). Following a tracking procedure (Logan et al., 1997), the delay between the stop signal and the go signal (*stop-signal delay*), originally set to 250ms, is adjusted: increased by 50ms if the participant successfully inhibits their response and decreased by 50ms if the participant fails to inhibit. Consequently, this adjustment ensures the participant inhibits their response approximately 50% of the time, i.e. the stop process and the go process finish at the same time. Therefore, the main outcome in the present study is the mean stop signal

reaction time (SSRT); longer times indicate greater impulsivity and less inhibitory control (for further details on the calculation of the stop signal reaction time see Logan et al. (1997)). The current study consisted of 32 practice trials, then three experimental blocks of 64 trials which were used for data analysis (for further information on the program see Verbruggen and Logan (2009)). The stop-signal task has a respectable pedigree as a measure of impulse control (Kwon & Kwon, 2013; Logan et al., 1997; Verbruggen & Logan, 2009).

Circle Drawing Task. The Circle Drawing Task (Bachorowski & Newman, 1990) is a measure of motor impulsivity and has been associated with impulsive behaviours and impulsivity-related disorders such as attention deficit hyperactivity disorder (Avila et al., 2004). A large printed circle (50.80 cm \varnothing), with a small intersecting line at the top to indicate the start/stop position, was drawn onto a wooden square. In the first condition, using their index finger, participants were asked to simply trace around the circle (neutral condition), whereas in the second condition they were instructed to trace the circle as slowly as possible without stopping (inhibition condition). Circle Time Difference was then calculated by subtracting the inhibition condition time from the neutral condition time; smaller circle differences indicate greater impulsivity and a lower ability to inhibit an ongoing motor response (Avila et al., 2004).

Risk Taking

The Balloon Analogue Risk Task (BART) (Lejuez et al., 2002) (Inquisit by Millisecond, Seattle) is a computerized assessment of risk taking behaviour and has been associated with both self-reported real-world risk behaviours and self-report measures of risk-related constructs (Lejuez et al., 2002). In this task, participants are required to inflate a simulated balloon, on a computer screen, to a desired level by clicking on a computer mouse. Each

click inflates the balloon by one degree and adds \$0.05 to a temporary reserve which is displayed on the screen as “Potential Earnings”. Participants were informed that each pump earned \$0.05 and that the balloons would pop, and if this occurred they would lose the money they had earned on that balloon. Additionally, they were informed they could keep the money from a balloon, which was added to an accumulative total, by stopping to pump before it popped and click on the button labelled “Collect \$\$\$”. In the present study participants were informed their total amount would be displayed on a leader board, with their aim to be top of this: individuals did not earn the actual money amount. The balloon number (1-30), number of pumps for that balloon, and their accumulative total were also displayed. The average breakpoint was 64 pumps as the balloons were set to explode on a variable ratio schedule (64 responses), and risk taking (i.e. further clicking of the pump) resulted in a negative outcome (i.e. loss of accrued money upon balloon explosion).

Participants were not given any information regarding when the balloons may explode. The task is set to model real world situations whereby excessive risk produces increased threat to one’s health and safety and produces diminished returns, i.e. each successive pump on a single balloon trial (a) increased the amount to be lost due to an explosion and (b) decreased the relative gain of any additional pump. The current task included 30 balloon trials and has been described in detail elsewhere (Hunt et al., 2005). In line with previous research (Hunt et al., 2005), the primary dependent variables indicative of risk taking were the total number of balloon explosions and the adjusted number of balloon pumps. The adjusted value is calculated by taking the mean number of balloon pumps on those balloons which did not explode because including all trials would have included balloons which exploded and therefore participants were forced to stop.

Persistence

Participants were presented with three solvable and then one unsolvable Euler puzzle to measure persistence (see Figure 5.1). The following instructions were given: “Here are a set of puzzles to complete. You have to trace along all of the lines, without lifting the pen from the paper and you can only trace along each line once. Once you have started to trace on each separate piece of paper, discard it if you make a mistake and use a new piece of paper. You must attempt all puzzles in numerical order. The puzzles involve more lines as you progress. You can have as many attempts on each puzzle as you like- there is no time or paper limit. So at any point you can decide you have given your best effort, but you need to let me know you would like to move on from the puzzle task completely”. All puzzles were initially presented face down. The number of attempts on each puzzle and the time spent on each puzzle were recorded; there was no limit to the number of pieces of paper participants could use. The primary measure of persistence was the time spent on the unsolvable final puzzle; if the participant was still persisting after 20 minutes then they were told to stop. Previous studies have applied similar paradigms to measure persistence (Eisenberg et al., 2001; Zhou et al., 2007).

Behavioral Task Laboratory Procedure

These behavioural tasks were undertaken in a laboratory specifically designed to minimize external or environmental distractions likely to affect performance: there were no windows with only a single desk and a laptop (Samsung Notebook, RC520) for the computerized tasks. Individuals were seated throughout and standard instructions were either presented orally or via the computer. A single experimenter, who led all the sessions, delivered the protocol with minimal subjective input and in a non-judgmental manner. The study

employed a double-blind testing procedure, such that neither participant nor experimenter was aware of the stress reactivity status of the participant. A Latin square procedure was used to create counterbalanced task order. The Euler puzzle persistence task was always completed last to protect from the Zeigarnik effect: the experience of intrusive thoughts as a consequence of failure to complete a task (Zeigarnik, 1935).

Data analysis

Group differences in socio-demographics, stress task performance and engagement, and cardiovascular baseline and reactivity variables were tested using univariate ANOVAs for continuous variables, and chi-square for categorical variables. Repeated measures ANOVAs were used to confirm that overall the stress task perturbed cardiovascular activity in the screening sample. Univariate ANOVAs were used to compare group differences in the behavioural task outcomes: SSRT, Circle Time Difference, BART adjusted number of pumps, BART total number of explosions, and Euler puzzle 4 persistence time. Due to previous research indicating sex differences in impulsivity (Weafer & de Wit, 2013), risk taking (Byrnes et al., 1999), and task persistence (Kiefer & Shih, 2006), ANCOVAs were subsequently applied to determine whether any group differences withstood adjustment for sex. Task order was also considered a possible confounder and, accordingly, also adjusted for. For the main ANOVAs and ANCOVAs, partial η^2 is reported as an index of effect size. Three participants for the stop-signal task and one participant for the circle drawing task were not included in the analysis due to technical failures.

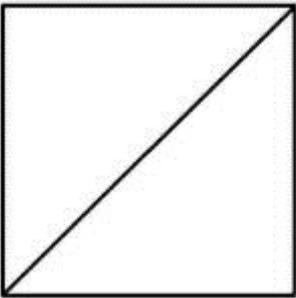
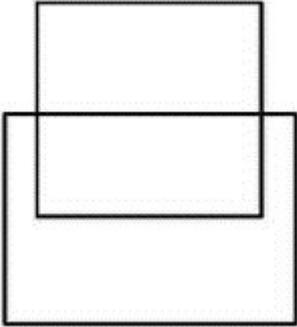
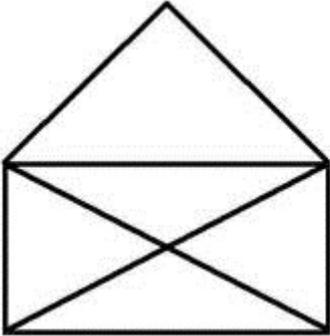
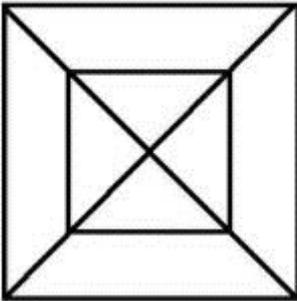
<p>Puzzle 1 (solvable)</p> 	<p>Puzzle 2 (solvable)</p> 
<p>Puzzle 3 (solvable)</p> 	<p>Puzzle 4 (unsolvable)</p> 

Figure 5.1. The Euler puzzles used to measure persistence: time duration on unsolvable puzzle 4 was the measure

RESULTS

Socio-demographics and cardiovascular stress reactivity

The summary data for socio-demographics, PASAT ratings, and cardiovascular stress reactivity measures are presented in Table 5.1. There were no significant group differences in any of the socio-demographic variables, or the PASAT total score and the self-reported task engagement or stress ratings (all p 's > .05).

From the overall screening procedure of 276 participants, the respective ranges for HR ($M=17.08$, $SD=11.56$), SBP ($M=18.25$, $SD=8.59$), and DBP ($M=12.16$, $SD=5.84$) reactivity were as follows: -7.75 to 75.00 bpm; -2.25 to 46.50 mmHg; -1.25 to 28.50mmHg. Repeated measures ANOVA indicated that the stress task significantly perturbed HR, $F(1,275) = 602.35$, $p < .001$, $\eta^2 = .678$, SBP, $F(1,275) = 1245.47$, $p < .001$, $\eta^2 = .819$, and DBP, $F(1,275) = 1197.78$, $p < .001$, $\eta^2 = .813$.

There were no significant group differences at baseline: for HR= 71.51 (12.75) vs. 71.14 (14.37) bpm , $F(1,44) = 0.01$, $p = .93$, $\eta^2 < .001$; for SBP= 120.10 (16.53) vs. 118.48 (10.43) mmHg, $F(1,44) = 0.16$, $p = .69$, $\eta^2 = .004$; or for DBP= 74.60 (10.11) vs. 72.32 (6.64) mmHg, $F(1,44) = 0.82$, $p = .37$, $\eta^2 = .018$. In contrast, and as would be expected, the selected groups differed substantially in cardiovascular reactivity. Mean (SD) values are presented in Table 5.1 for HR, $F(1,44) = 199.92$, $p < .001$, $\eta^2 = .820$; SBP $F(1,44) = 53.29$, $p < .001$, $\eta^2 = .548$; and DBP, $F(1,44) = 12.24$, $p = .001$, $\eta^2 = .218$.

Table 5.1. Socio-demographics, stress task performance and ratings, and cardiovascular reactivity measures for the blunted and exaggerated stress reactivity groups

	Blunted	Exaggerated
	Mean (SD)/N (%)	
Gender (females)	11 (48)	12 (52)
Age (years)	24.63 (10.75)	20.50 (2.66)
BMI (kg/m ²)	23.41 (3.43)	22.34 (2.14)
PASAT total score	695.43 (130.80)	715.65 (151.58)
Engagement	4.04 (1.15)	4.52 (1.12)
Stressfulness	3.91 (1.47)	4.48 (1.08)
HR reactivity (bpm)*	2.00 (3.17)	39.28 (12.24)
SBP reactivity (mmHg)*	12.05 (6.97)	28.11 (7.92)
DBP reactivity (mmHg)*	9.67 (7.18)	16.02 (4.92)

Note: * $p < .05$

Behavioral task outcomes

For the stop-signal task, ANOVA revealed a significant group difference in the SSRT, $F(1,41) = 4.99$, $p = .03$, $\eta^2 = .108$, with the blunted group registering a larger SSRT, indicating lower inhibitory control and greater impulsivity. This effect is illustrated in Figure 5.2a. For the circle drawing task, there was also a significant group difference in Circle Time Difference, $F(1,43) = 4.00$, $p = .05$, $\eta^2 = .085$, with the blunted group producing a shorter Circle Time Difference reflecting greater impulsivity. This effect is illustrated in

Figure 5.2b. Regarding risk taking as measured by the BART, there were no differences between the blunted ($M=30.44$, $SD=13.13$) and exaggerated ($M=30.25$, $SD=13.60$) reactor groups for either the adjusted number of pumps, $F(1,44) = 0.01$, $p = .96$, $\eta^2 < .001$, or the total number of explosions ($M=7.26$, $SD=3.86$ and $M=7.87$, $SD=2.85$, respectively), $F(1,44) = 0.37$, $p = .55$, $\eta^2 = .008$. There was also no significant difference between the blunted ($M=537.30s$, $SD=345.85$) and exaggerated ($M=643.48s$, $SD=423.78$) reactor groups, $F(1,44) = 0.87$, $p = .36$, $\eta^2 = .019$, in the time they persisted on the unsolvable puzzle.

Sensitivity Analysis. Sex and task order had no significant effect on task performance for any of the tasks (all p 's $>.05$), with the exception of an effect of task order on puzzle 4 persistence time; $F(9,36) = 2.25$, $p=.04$, $\eta^2 = .360$; those completing the BART as the third task tended to have the shortest persistence time on Euler puzzle 4. Nevertheless, as sex has previously been associated with differences in the behavioural measures (Byrnes et al., 1999; Kiefer & Shih, 2006; Weafer & de Wit, 2013) it was still controlled for. Similar outcomes emerged following statistical adjustment for sex and task order: for SSRT, $F(1,39) = 4.92$, $p = .03$, $\eta^2 = .112$; for Circle Time Difference, $F(1,41) = 4.17$, $p = .05$, $\eta^2 = .092$; for adjusted number of pumps on the BART, $F(1,42) = 0.10$, $p = .75$, $\eta^2 = .002$, the total number of explosions, $F(1,42) = 0.11$, $p = .74$, $\eta^2 = .003$; and for persistence time, $F(1,42) = 0.61$, $p = .44$, $\eta^2 = .014$.

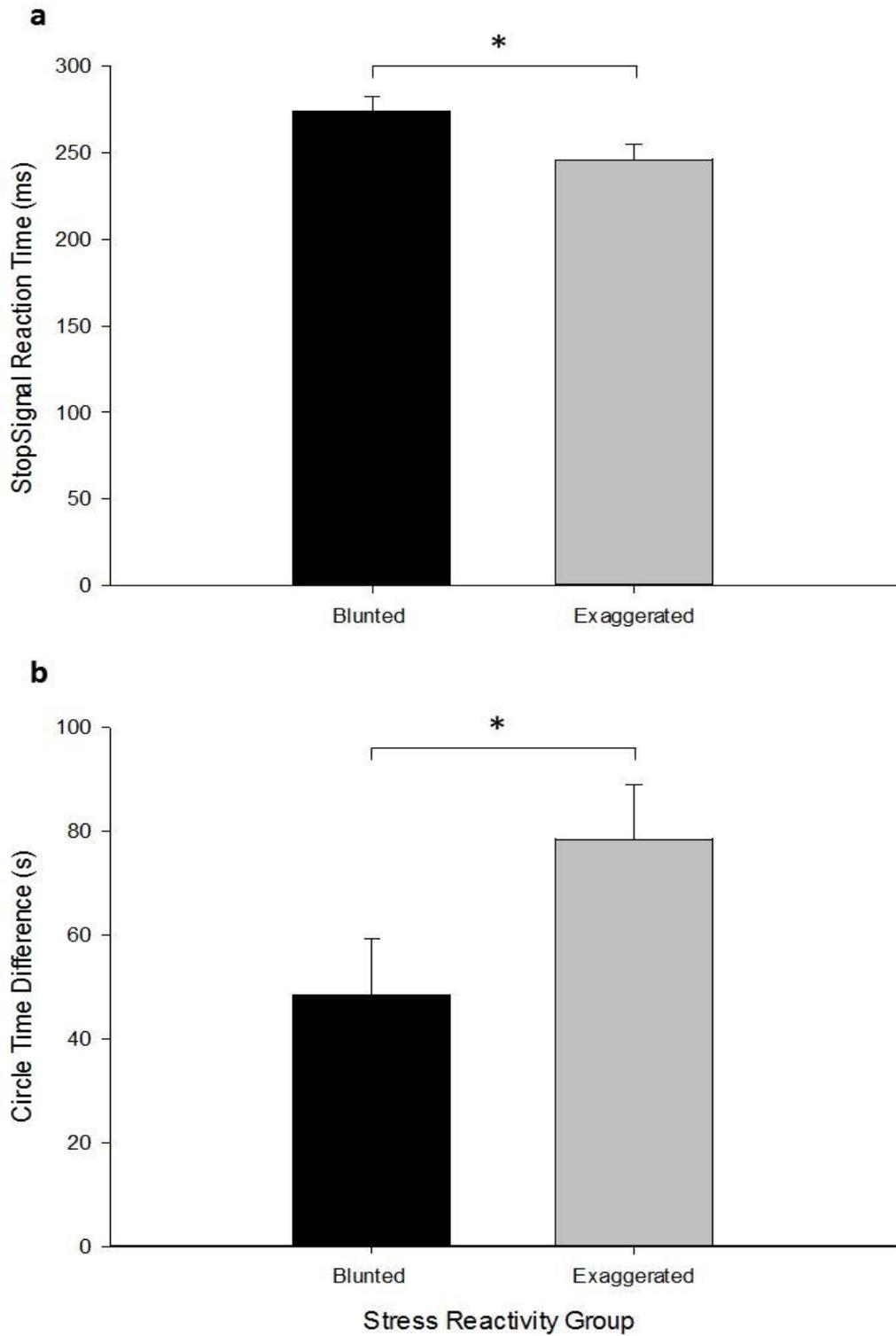


Figure 5.2. Mean (SE) (a) Stop signal reaction time, and (b) Circle time difference for the blunted and exaggerated stress reactivity groups. * $p \leq .05$

DISCUSSION

The present study is the first we are aware of to both adopt a case-control design and use behavioural measures to determine whether individuals with blunted or exaggerated cardiovascular stress reactions differ in terms of impulsivity, risk taking, and persistence. As hypothesized, individuals with blunted reactivity were more impulsive. This emerged for both measures of impulsivity and was still evident following adjustment for both sex and task order. In contrast, persistence and risk taking did not vary significantly with stress reactivity status.

The current finding of greater impulsivity in individuals with blunted cardiovascular stress reactions is in line with a recent study by members of our group on pre-adolescent children; they also observed an association between blunted cardiac stress reactions and poorer impulse control as determined by Circle Time Difference and failure to inhibit responses on a Go-NoGo task, as well as by maternal ratings of impulsivity (Bennett et al., 2014). What is true regarding the behaviour of children would also appear to hold for young adults.

Although counter evidence, based on two self-report studies, does exist (Diller et al., 2011; Mathias & Stanford, 2003), our results contribute to what might reasonably be regarded as an emerging consensus linking blunted stress reactivity and impulsivity (Allen et al., 2009; Bennett et al., 2014; Munoz & Anastassiou-Hadjicharalambous, 2011). To the best of our knowledge no previous studies have examined the association between biological stress reactivity and persistence. Although the aggregate data suggest that blunted cardiac reactors may be less persistent, the group difference was not close to statistical significance. The present lack of significant association between risk taking behaviour and cardiac reactivity is in accord with the one published study which also reported no association between cardiovascular reactivity and self-reported risk taking in adolescent boys (Liang et al., 1995).

Central motivational dysregulation in the brain has been proposed to be the link between all of the seemingly diverse clinical outcomes of obesity, depression, a range of substance and behavioural dependencies and bulimia; all of which have been associated with blunted stress reactions (Carroll et al., 2009; Carroll et al., 2011; Lovallo, 2011). Thus, blunted stress reactivity is considered a peripheral marker of dysregulation of the neural systems that support motivation, emotional regulation, and goal-directed behaviour. Evidence in support of this comes from functional Magnetic Resonance Imaging studies. Individuals characterized by blunted cardiovascular stress reactivity showed diminished activation/de-activation in the anterior and posterior cingulate cortex, and the amygdala during stress exposure (Gianaros et al., 2005; Ginty et al., 2013). These brain areas are involved in motivational processes and goal-directed behaviour as well as autonomic nervous system regulation (Bush et al., 2000; Hagemann et al., 2003; Lovallo, 2005). They have also been implicated in impulsivity; individuals with greater impulsivity show similar deficiencies in the frontolimbic areas of the brain (Horn et al., 2003; Inuggi et al., 2014). Further, impulsivity has been shown to be higher in individuals with addiction to alcohol (Aragues et al., 2011), tobacco (Balevich et al., 2013), other non-prescription substances (Perry & Carroll, 2008), gambling (Leeman & Potenza, 2012), and exercise dependence (Freimuth et al., 2011), as well as in those who are obese or have eating disorders (Schag et al., 2013). Recent evidence also indicates that individuals with blunted stress reactions have higher levels of criminality and re-offending (De Vries-Bouw et al., 2011), are more likely to drive under the influence of alcohol (Couture et al., 2008), and engage in general disruptive behaviour (De Vries-Bouw et al., 2011). Again, impulsivity has been shown to be associated with each of these behavioural outcomes (Patton et al., 1995; Treloar et al., 2012).

The idea of central motivational dysregulation has recently been incorporated into a conceptual model which proposes dysfunction in the frontolimbic areas of the brain leads to both diminished stress reactivity and compromised regulation of motivation and emotion (Lovallo, 2013). The latter is manifest as poorer impulse control and, consequently, increased risk for adverse health behaviours, anti-social behaviour, and addiction (Lovallo, 2013). By further demonstrating an association between impulsivity, as reflected in poorer inhibitory control, greater motor impulsivity, and blunted stress reactivity, our present findings lend further support to this model.

The present study is not without limitations. First, although the blunted stress reactors showed lower persistence on aggregate, the group difference was far from significant. It may be argued that the sample size of 23 individuals in each group was modest and therefore may have reduced the power to detect differences between reactor groups. However, the current study did screen a large number of participants and selected extreme reactors from a strict 15th percentile cut-off to ensure substantial reactivity differentiation between groups. The screening procedure and the selection criteria were even more stringent than previous studies that have selected extreme blunted and exaggerated stress reactors for secondary investigation (Gianaros et al., 2005; Ginty et al., 2013). Second, it is always possible that individuals, as a result of demand characteristics (Damaser et al., 2010), behaved differently in a laboratory than they would in a real world environment, and this might militate against group differences in behaviour. However, the BART is considered one of the best laboratory based measures of risk taking (Aklin et al., 2005), with high test-retest stability (White et al., 2008). Importantly, its ecological validity is also supported by results showing that riskiness measured by the BART was highly correlated with self-reported real world addictive, health, and safety behaviours (Lejuez et al., 2003; Lejuez et al., 2002). Future research may like to

utilise psychometric measures of impulsiveness, risk taking, and persistence, to provide further validation of the behavioural measures. In terms of strengths, the present study employed a double-blind testing procedure whereby neither experimenter nor participant were aware of participant's reactivity status. In addition, task order was randomized, save for the persistence task, to protect from the Zeigarnik effect, and also adjusted for statistically. Further strengths were the adoption of a case-control design and the use of behavioural rather than self-report measures.

In summary, blunted stress reactors were characterized by high levels of behavioural impulsivity. Blunted and exaggerated reactors did not differ in risk taking or persistence. Over a period of years this increased impulsivity could potentially contribute to the development of addictions and the adverse health outcomes associated with blunted stress reactivity. Future research should focus on the association between reactivity and additional proximal behaviours, such as delayed reward gratification and anti-social tendencies. This may help further define the phenotypes that arise from central motivational dysregulation and are marked by blunted stress reactions.

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CHAPTER SIX

GENERAL DISCUSSION

The overarching aim of the present thesis was to further understand and expand knowledge of the corollaries of individual differences in cardiovascular and/or cortisol reactions to acute psychological stress. In particular, the studies focussed upon whether and which personality and behavioural characteristics were related to physiological stress reactivity. Given that an individual's personality can be regarded as a constellation of consistent behavioural tendencies and emotional demeanour (McCrae & Costa, 1987), it should be recognised at the outset that personality (Studies 1 and 2: Chapters 2 and 3), dependencies (Study 3: Chapter 4), and proximal behavioural characteristics (Study 4: Chapter 5) are not distinct. Rather, they provide a linked platform to further examine the theory that blunted cardiovascular and cortisol reactivity are markers of central motivational dysregulation which underlies these characteristics and behaviours (Carroll et al., 2009; Lovallo, 2011; Phillips et al., 2013). Motivational dysregulation refers to a dysfunction of those systems in the brain that support motivated behaviour under conditions of challenge such as that afforded by stress (Ginty, 2013).

Summary of results

Personality and stress reactivity

Chapter 2 provided an examination, in a large middle aged cohort, of whether cardiovascular and cortisol responses to stress were associated with the Big Five personality traits.

Individuals scoring higher on neuroticism and lower on agreeableness and openness had smaller cortisol stress reactions. Neuroticism was also negatively associated with SBP, DBP, and HR stress reactivity, whereas agreeableness and openness were positively associated with HR reactivity. Given that previous research, had not controlled for a number of potential confounding variables (Williams et al., 2009b; Wirtz et al., 2007), it is important

to emphasise that these associations were still extant following statistical adjustment for a range of potential confounders: sex, age, SES, alcohol consumption, smoking, BMI, use of anti-hypertensive medication, use of anti-depressant or anxiolytic medication, perceived commitment to the stress task, and baseline activity. Extraversion and conscientiousness were not related to either cortisol or cardiovascular stress reactivity. Overall, it would appear that a negative constellation of personality traits, i.e., higher neuroticism, but lower agreeableness and openness, was associated with diminished stress reactions both of the cardiovascular system and the HPA axis. In addition, it is important to highlight the dissociation between self-reported stress task impact and physiological reactivity. Despite higher neuroticism being associated with greater perceptions of task stressfulness and difficulty, and lower feelings of control, those high in neuroticism exhibited blunted biological stress reactions. Similarly, those scoring higher in openness reported the stress tasks as less stressful and difficult, and reported greater feelings of control, despite displaying higher cortisol and HR reactions. This adds to the growing evidence that the magnitude of stress perceptions do not always parallel the magnitude of physiological stress reactivity (Campbell & Ehlert, 2012; Ginty et al., 2012a; Heaney et al., 2011).

Type D personality and stress responses

Chapter 3 compared the cardiovascular and cortisol stress reactions of Type D and non-Type D individuals assigned to either an asocial or socially evaluative stress exposure condition. Type D individuals in the socially evaluative condition exhibited the greatest SBP, DBP, and HR responses, and were the only group to mount a positive cortisol response. In the asocial condition, Type Ds tended to show blunted SBP and DBP reactivity compared to their non-Type D counterparts. As there were sub-group differences in age, cardio-respiratory fitness,

depression and perceived stressfulness, the main analyses were repeated adjusting for these variables in addition to the appropriate baseline cardiovascular and cortisol levels. All of the associations remained following statistical adjustment, and these associations survived further adjustment for anti-depressant medication use, sex, BMI, smoking status, and testing time. Again, there appeared to be some dissociation between self-reported task stressfulness and physiological reactivity. Overall, the Type D participants perceived the tasks to be more stressful than non-Type Ds, and although this was associated with greater physiological reactivity in the social condition, Type D individuals in the asocial conditions actually had lower SBP and DBP reactions than their non-Type D counterparts.

Stress reactivity and substance and non-substance abuse

Chapter 4 tested the limits of the hypothesis that blunted stress reactivity is a peripheral marker of the central motivational dysregulation, and as such a signifier of risk for addiction, by comparing the cardiovascular and cortisol stress reactivity of individuals with comorbid Internet and alcohol dependence, Internet dependence *per se*, alcohol dependence *per se*, and individuals showing no signs of dependence. There were no group differences in any of the physiological stress measures indicating that neither problematic Internet behaviour nor excessive alcohol consumption, either individually or in combination, were associated with blunted cardiovascular or cortisol stress reactions. As BMI was significantly lower in the control group, and has been shown to relate to reactivity (Carroll et al., 2008; Phillips et al., 2012) the above analyses were repeated with BMI as a covariate; this did not alter the outcomes. There were no group differences in age, sex, BMI, ethnicity, health behaviours, stress task performance or self-reported task ratings including engagement.

The proximal behavioural characteristics of those showing blunted stress reactivity

For the study reported in Chapter 5, 276 individuals were stress tested with blood pressure and HR measured. Those in the top and bottom 15th percentiles of HR reactivity were then selected to determine whether exaggerated and blunted reactors were behaviourally distinct; in particular, in impulsivity, risk taking, and persistence. In using behavioural tasks to measure these characteristics, rather than self-report measures preferred in previous studies, it was observed that blunted stress reactors displayed greater levels of impulsivity, as indicated by lower inhibitory control and greater motor impulsivity, while risk taking and persistence were not different between the reactor groups. The group differences in impulsive behaviour remained following adjustment for sex and task order. The finding that blunted reactors are characterised by greater impulsivity adds to the contention that blunted stress reactivity may be a peripheral marker of central motivational dysregulation, which is behaviourally manifest as increased impulsivity, which, in turn, may contribute to the risk for addiction and poor health behaviours (Lovallo, 2013).

Implications

Since the emergence of the reactivity hypothesis in the early 1980s (Obrist, 1981), there has been substantial research demonstrating that individuals with exaggerated cardiovascular (Carroll et al., 2012b; Carroll et al., 2003; Everson et al., 1996; Everson et al., 1997; Georgiades et al., 1997; Kapuku et al., 1999; Lynch et al., 1998; Matthews et al., 1998; Matthews et al., 2006; Murdison et al., 1998; Newman et al., 1999; Treiber et al., 1997) and cortisol (Girod & Brotman, 2004; Hamer et al., 2010; Hamer & Steptoe, 2012) reactions to acute psychological stress are at increased risk of developing cardiovascular disease and its various manifestations, with an overall increased risk of cardiovascular disease mortality

(Carroll et al., 2012a). Given such findings, it is perhaps hardly surprising that the main focus of stress reactivity research has been on conditions or characteristics that lead to exaggerated reactivity (Chida & Steptoe, 2010), with blunted physiological reactivity implicitly assumed to be benign or even protective (Carroll et al., 2009). However, more recent research has been emerging to suggest that blunted cardiovascular and cortisol reactivity may be associated with a range of adverse health and behavioural outcomes (Carroll et al., 2009; Lovallo, 2011; Phillips et al., 2013). Thus, this thesis is timely in that its findings suggest three things: further negative components, namely adverse personality traits, are associated with blunted reactivity; potential boundaries to the hypothesis that blunted reactivity is a marker of central motivational dysregulation; and that increased impulsivity may be a proximal behavioural characteristic of blunted stress reactors.

In Chapter 2, it was reported that higher neuroticism, but lower openness and agreeableness were associated with diminished physiological stress reactivity. Thus, it is a somewhat negative collection of traits that are related to blunted reactivity. Indeed, neuroticism, a tendency toward negative affectivity and an inclination toward impulsive behaviour (McCrae & Costa, 1987), is viewed as a negative trait and has been associated with a further range of negative health outcomes including tobacco and drug dependency (Malouff et al., 2006; Terracciano et al., 2008). Similarly, agreeableness, which signifies a willingness to be helpful and trusting, and to possess a prosocial orientation towards others (McCrae & John, 1992), is a positive trait with low levels seen as maladaptive and associated with negative outcomes such as depression and anxiety disorders (Bienvenu et al., 2001; Bienvenu et al., 2004; Samuels et al., 2002). Finally, openness, which is characterised by a tendency to be imaginative and attentive to inner feelings, is also seen as a positive trait, with low levels associated with increased disease progression (Ferguson & Bibby, 2012; Ironson et al.,

2008). Additionally, given that Type D personality is viewed as a negative trait (Denollet, 2005) and Chapter 3 demonstrated that during the asocial condition Type D individuals exhibited a tendency toward blunted cardiovascular reactions this would suggest a further adverse characteristic to be associated with attenuated stress reactivity. Thus, overall the findings extend our knowledge of the corollaries of physiological stress reactivity and further support the notion that blunted reactivity is maladaptive.

Thematic links

As previously noted, the four empirical chapters are not distinct in their focus, and some common themes would appear to emerge: 1. an individual's subjective stress rating and their physiological stress reactivity are not necessarily positively collinear and indeed in certain individuals and in certain contexts may be dissociated or indeed negatively associated; 2. the context of the stressful environment has an impact on the consequent biological reactions; 3. blunted reactivity appears to be related to stable adverse traits but not to more transient behaviours.

Firstly, many of the models of stress including Lazarus' Transactional Model (Lazarus, 1996), postulate that the magnitude of an individual's physiological response to stress varies in a consistent fashion according to their subjective interpretation of the stress exposure. Coherence between the cognitive-emotional and physiological systems has been assumed for many decades due to the following proposed pathway (for a review, see Mauss et al., 2005); the prefrontal cortex provides the cognitive evaluation of the importance of the stimulus and the available coping resources, this then initiates the formation of the emotional responses via limbic connections, with subsequent activation of the physiological systems (Feldman et al., 1999). The present thesis, however, provides evidence that this is not the case and that

there can be dissociation between psychological appraisal, i.e., perceived stressfulness, and physiological reactivity, also found in other studies (Campbell & Ehlert, 2012; Ginty et al., 2012; Heaney et al., 2011). In Chapter 2, despite higher neuroticism being associated with greater perceptions of task stressfulness and difficulty, and lower feelings of control, those high in neuroticism exhibited blunted biological stress reactions. Further, those scoring higher in openness reported the stress tasks as less stressful and difficult, and reported greater feelings of control, despite displaying higher cortisol and HR reactions. This dissociation also emerged in Chapter 3, whereby despite Type D individuals reporting the stress tasks as more stressful compared to the non-Type D individuals, they actually had lower blood pressure than non-Type Ds in the asocial condition. These data also help counter the argument that the adverse health and behavioural corollaries of blunted stress reactivity are due to blunted reactors not engaging in the stress tasks and thus not psychologically interpreting the situations as stressful. Given that in both Chapter 2 and 3 of the present thesis, task engagement, task performance, and perceived stressfulness and other subjective ratings were controlled for this would suggest that the blunted physiological reactivity is an underlying unconscious process which may indeed reflect central motivational dysregulation of the brain areas concerned with emotions and stress reactivity (Carroll et al., 2009; Carroll et al., 2011; Lovallo, 2011). In support, a 49-study review which reports little association between subjective and physiological stress responses, suggests that brain morphology, i.e. possible central motivational dysregulation, is likely to be a leading factor in this dissociation (Campbell & Ehlert, 2012).

Secondly, not only does personality affect an individual's stress reactivity but it would also appear that individuals respond differently according to the context and circumstance. As Chapter 3 states, much of the previous research on Type D personality and physiological

stress responses provided mixed results (Habra et al., 2003; Howard et al., 2011; Williams et al., 2009a). The current thesis suggests that this ambiguity was most likely due to the level of the social evaluation incorporated into the stressor, with the study reported in Chapter 3 demonstrating that social evaluation *per se* increased physiological reactivity, and that this was particularly the case for Type D individuals. On the other hand, in the absence of social evaluation, Type D individuals tended to exhibit lower cardiovascular stress reactions than their non-Type D counterparts. Thus, in the case of Type D personality, which has been linked to a range of adverse cardiovascular outcomes (Denollet, 2005), it is possible that normative deviation in the exaggerated direction under social stress and blunted direction under asocial stress may both lead to this increased risk of adverse health outcomes. This is supported by research implicating exaggerated physiological reactivity in the development of cardiovascular disease (Carroll et al., 2012b), and blunted reactivity in a range of negative outcomes, such as depression (Spindler et al., 2009), obesity (Carroll et al., 2008; Phillips, 2011; Phillips et al., 2012), and smoking (Ginty et al., 2014), all of which have also been linked to cardiovascular disease (Boden & Salehi, 2013; Chen & Boreham, 2002; Rugulies, 2002). Thus, irrespective of the nature of the stress exposure, Type D personality would appear to confer risk. When analysing stable traits such as personality and certain behavioural characteristics, future research might investigate and consider the possible contextual interactive effects of further underlying components such as competition and social support. Indeed, previous research has demonstrated that contextual social support and intimacy *per se* can have differential effects on physiological stress reactivity (Howard & Hughes, 2012; Hughes, 2007; Phillips et al., 2009c).

Thirdly, given that the present thesis reported that blunted reactivity was associated with a constellation of negative Big 5 personality traits, Type D personality under asocial stress,

and impulsivity, but not problematic Internet or alcohol use in a student population, this would suggest attenuated reactivity is only associated with stable adverse traits. In support, it has been shown that the Big 5 personality traits have genetic components (Jang et al., 1996) and have high stability for periods of over 45 years (Soldz & Vaillant, 1999; Terracciano et al., 2006). Similarly, Type D personality has shown high stability over a 9-year period with substantial heritability of genetic factors contributing to this (Kupper et al., 2011). Twin studies have also indicated that impulsivity is a stable construct throughout an individual's lifespan and genetic influences account for over half of its variance (Niv et al., 2012; Seroczynski et al., 1999). On the other hand, Chapter 4 tested the boundaries of the corollaries of blunted reactivity by assessing whether problematic Internet use and excessive alcohol intake in a student population would be related. Previous research had shown that adults with diagnosed alcoholism (Bernardy et al., 1996; Dai et al., 2007; Errico et al., 1993; Lovallo et al., 2000; Panknin et al., 2002) and tobacco (al'Absi et al., 2003; Evans et al., 2012; Ginty et al., 2014; Girdler et al., 1997; Kirschbaum et al., 1993b; Rohleder & Kirschbaum, 2006; Roy et al., 1994) dependence were characterised by blunted reactivity, but the present study found no association between excessive Internet use, alcohol intake and blunted reactivity. Despite alcoholism and tobacco smoking showing signs of stability over the life course, with neuroimaging studies showing dependent individuals have inherited neurological (Grant et al., 2006) and genetic (Goldman et al., 2005) differences, excessive alcohol and Internet use during university study could be considered more transient (Beard & Wolf, 2001; Bewick et al., 2008). Indeed, Internet addiction remains a controversial and heterogeneous construct with no agreed definition (Greydanus & Greydanus, 2012). It has not been included in the latest Diagnostic and Statistical Manual-V (APA, 2013), and there is little evidence of physical withdrawal symptoms typical of actual dependence (Beard and

Wolf, 2001). Similarly, research shows that alcohol consumption is higher in students and rises in transition from school to university, is commonly observed as bingeing (Gill, 2002), but then there is a clear decline upon graduation (Bewick et al., 2008; Klein, 1994; Lanza & Collins, 2006). Accordingly, within this thesis problematic Internet behaviour and excessive alcohol consumption by students may be a transient phenomenon rather than a reflection of a true addiction. Additionally, it should also be noted that impulsivity is often considered to be part of individual's stable personality constitution, more specifically a distinct component of neuroticism (McCrae & Costa, 1987), thus it is not surprising that both neuroticism and impulsivity were a particular feature of blunted stress responders in the present thesis. Upon review of all the present chapters it would suggest that blunted reactivity is associated with stable adverse components but not transient behaviours, and this adds further support to the notion that attenuated reactivity is a marker of underlying central motivational dysregulation.

Motivational dysregulation and blunted cardiovascular and cortisol reactions to stress

As this thesis has demonstrated blunted reactivity is associated with a negative constellation of personality traits and greater behavioural impulsivity. In terms of causation, there are three possibilities: 1. blunted reactivity predates and leads to these adverse outcomes; 2. blunted reactivity is a result of these corollaries; or, what is more likely, 3. blunted reactivity is a peripheral marker of the central motivational dysregulation which is reflected by a range of negative health and behavioural outcomes along with dysfunction of the body's physiological stress response. We know that individuals characterised by blunted biological stress responses have been found to show diminished activation in both the posterior and anterior cingulate cortex, and in the amygdala during stress exposure (Gianaros et al., 2005; Ginty et al., 2013). These areas are indeed implicated in motivational processes and goal-

directed behaviour, as well as autonomic nervous system regulation (Bush et al., 2000; Hagemann et al., 2003; Lovallo, 2005). The present finding that negative personality characteristics are associated with blunted stress reactivity is certainly in keeping with this hypothesis. After all, such characteristics have been linked to many of the adverse health and behavioural outcomes associated with blunted stress reactivity e.g., depression (Bienvenu et al., 2004), obesity (Sutin et al., 2011), disordered eating and exercise dependence (Bamber et al., 2000; Bulik et al., 2006; Cassin & von Ranson, 2005), and tobacco, alcohol, and substance dependence or addiction risk (Martin & Sher, 1994; Munafò et al., 2007; Terracciano & Costa, 2004; Terracciano et al., 2008). Further, an individual's personality has also been shown to be determined, at least in part, by the same neural systems and brain areas associated with motivational dysregulation (Cremers et al., 2010; DeYoung & Gray, 2009). Likewise, the same areas have also been implicated in impulsivity; individuals with greater impulsivity show similar deficiencies in the frontolimbic areas of the brain (Horn et al., 2003; Inuggi et al., 2014). As previously noted, it is not the case that blunted reactors are not engaging with the tasks. In the present studies and many previous studies implicating blunted reactivity in adverse health and behavioural outcomes, subjective task impact and/or task performance were controlled for statistically. This suggests that what is driving blunted stress reactivity is not only more nuanced than simple psychological disengagement but is largely hidden from consciousness. Rather what appears to be happening is a biological disengagement, resulting from the systems required to mount an adequate biological and behavioural reaction to challenge not activating optimally.

Future research might also explore the origins of blunted reactivity and the extent to which it is determined by genetic, environmental, or genetic x environmental influences. Regarding

genetics there are a number of candidate genes. The dopamine receptor gene, D2, has been strongly implicated in reward deficiency and many of the adverse outcomes associated with blunted stress reactivity (Blum et al., 2011; Ginty, 2013). Secondly, the serotonin transporter (5HTT) gene has been associated with behavioural dependencies such as bulimia (Akkermann et al., 2012), depression (Caspi et al., 2003; Lesch et al., 1996), and greater cardiovascular responses to stress (Williams et al., 2001; Williams et al., 2008). Thirdly, the polymorphism of the COMT gene has been associated with differences in emotional and physiological responsivity (Goldman et al., 2005; Zubieta et al., 2003), as well as increased risk for addiction (Enoch et al., 2006; Vandenberg et al., 1997). Finally, the variable number tandem repeat (VNTR) polymorphism of the MAOA gene has been implicated in the interaction between early life stress and impulsivity (Manuck et al., 2000; Manuck et al., 2002); particularly relevant given the findings of the present thesis. This gene also highlights the link to an important environmental factor which has been implicated in blunted stress reactivity and its negative corollaries: early life adversity. It has been shown that individuals who have experienced high levels of adversity during childhood exhibit a number of associated characteristics including reduced HR and cortisol stress reactivity, unstable regulation of emotions, and diminished cognitive capacity (Lovallo, 2013). In a theoretical model it has been proposed that this constellation of physiological, affective, and cognitive tendencies leads to changes in brain function, i.e., central motivational dysregulation, which then manifests as poorer impulse control and, consequently, increased risk for adverse health behaviours, anti-social behaviour, and addiction (Lovallo, 2013). The findings of Chapter 5 lend further support to this model by demonstrating an association between impulsivity, as reflected in poorer inhibitory control, greater motor impulsivity, and blunted stress reactivity.

Limitations

The present thesis is not without limitations. Specific limitations regarding each study have been addressed in the Discussion sections of the relevant empirical chapters thus this section focuses on the general limitations of the thesis as a whole. First, as previously alluded to, students classified as displaying excessive Internet use and high levels of alcohol consumption may not have constituted genuine and ingrained addictions. Nevertheless, the cut off criteria on the IAT and AUDIT, used to classify the Internet and alcohol dependencies respectively, generally conformed to the recommended criteria and were similar or more stringent than that used in other studies (Evans et al., 2012; Hardie & Tee, 2007; Lam & Peng, 2010; Saunders et al., 1993; Young, 1996a). The fact that these excessive behaviours may have been transient might highlight a potential boundary to the corollaries of blunted stress reactivity; they are largely stable characteristics and behaviours. Second, the sample sizes of the laboratory studies could be considered modest. It should be noted, however, that each of these studies screened a substantial sample via questionnaires or stress testing to isolate the most extreme groups. For the studies reported in Chapters 3 and 4, a large number of participants were screened (N = 2300 and 2313, respectively) and then the most extreme cases were selected according to similar or even stricter criteria suggested or previously used for Type D personality (Denollet, 2005; Howard & Hughes, 2013; Williams et al., 2009a), and Internet and alcohol dependency (Evans et al., 2012; Hardie & Tee, 2007; Lam & Peng, 2010; Saunders et al., 1993; Young, 1996a, 1996b). Chapter 5 also screened 236 potential participants through full cardiovascular stress laboratory protocols and selected extreme reactors from a strict 15th percentile cut-off to ensure substantial reactivity differentiation between groups. Additionally, the screening procedure was more extensive in terms of number of participants and the selection criteria was even more

stringent than previous studies that have selected extreme blunted and exaggerated stress reactors for secondary investigation (Gianaros et al., 2005; Ginty et al., 2013); i.e. using the extreme 15th percentiles rather than selecting extreme groups based on being above or below 2 standard deviations from the mean (Ginty et al., 2013). Finally, it remains a possibility that the current findings could, to an extent, arise from confounding by an unmeasured or poorly measured variable (Christenfeld et al., 2004); all of the studies however statistically adjusted for a range of potential confounders.

Strengths

The present thesis also has a number of strengths. All of the chapters were innovative and had novel aspects thus furthering the knowledge within the physiological stress reactivity field. Chapter 2 extends previous research on the Big 5 personality by utilising a large dataset, examining both cardiovascular and cortisol reactivity, controlling for a number of potential confounders, and by examining subjective stress task ratings. Chapter 3 advanced the research on Type D personality by directly comparing stress reactions to asocial and social stress situations, and was also only the second study to assess cortisol stress reactivity in this context (Habra et al., 2003). The examination of the association between Internet dependency and stress reactivity in Chapter 4 was also novel and excessive alcohol consumption in students has received little attention in the present context. Moreover, the concept of comparing comorbid, single, and no dependency was innovative. Finally, no research has selected extreme blunted and exaggerated physiological stress responders to assess their behavioural characteristics as the present thesis did in Chapter 5. It is also important to note that Lovallo (2005) proposes a three level stress response system: Level I incorporates the cognitive appraisals and the emotional and motivational responses of the

brain; Level II includes the hypothalamus, brainstem, and limbic systems as pathways to communicate with the body and affect endocrine response; the link between Levels II and III accounts for the autonomic and endocrine outputs to the periphery; and Level III includes the actual peripheral tissues of the body. Regarding the present thesis, not only did Chapters 2, 3, and 4 measure both cardiovascular and cortisol (endocrine) reactivity thus providing a comprehensive assessment of both branches of the stress response system: the autonomic nervous system and the HPA-axis, but this also provides information on Level II and the link between Level II and III in Lovallo's model. Additionally, the examination of the self-report stress task ratings regarding the cognitive and emotional appraisal addressed Level I of the model, and was also informative by providing an important theme that contrary to previous research (Feldman et al., 1999; Lazarus, 1996; Mauss et al., 2005) there can be dissociation between emotional and physiological stress responses. Overall, the present thesis provides an extensive examination of the levels and interconnections of Lovallo's model. As previously noted, in Chapters 3, 4, and 5 there were substantial screening procedures and strict selection criteria to ensure the most extreme cases were selected. The generalizability of the findings is aided by the fact that the thesis included a secondary analysis of a large dataset and laboratory work. Thus, the studies reported included both young adult (Chapters 3, 4, 5) and middle aged populations (Chapter 2), as well as including different nationalities (Chapters 3,4,5- UK, Chapter 2- The Netherlands) and a range of different stress tasks: i.e. Stroop, Mirror drawing, Speech, and PASAT. All of the studies also included both men and women.

Future directions

This thesis has extended the current knowledge of the research field and in doing so has unearthed a number of future directions. Firstly, following on from the present demonstration that there can be dissociation between an individual's emotional appraisal and physiological responses to stressful experiences, future research might further assess affective appraisals such as challenge and threat, cognitive and somatic anxiety, facilitative and debilitating interpretation, and other states such as self-efficacy and fear of failure. It was also evident from Chapter 3 that the context in which the stressful situation is experienced is important and may moderate associations between certain constructs and stress reactivity. Thus future research may focus upon the differential effects of aspects such as the available social support in an asocial and socially stressful situation, and competitive elements across varying situations; both these elements have been shown to affect stress reactivity *per se* (Harrison et al., 2001; Phillips et al., 2009). Despite the present null findings, the idea of studying co-morbidities has utility, given that it has been shown that comorbidity (Vogeli et al., 2007) such as obesity and depression (Singh & Shen, 2013), and major depressive disorder and generalized anxiety (Phillips et al., 2011), have been associated with poorer health outcomes than single disorders. If comorbid outcomes were related to greater blunting of an individual's stress response this would provide further support for the hypothesis of central motivational dysregulation. As the present thesis was the first to examine the behavioural traits of blunted and exaggerated stress reactors, investigation of additional proximal behaviours such as delayed reward gratification and altruism may help further define the behavioural phenotypes associated with blunted stress reactivity. As previously discussed, it would be informative to determine the contribution of genetic factors to blunted stress reactivity with specific focus upon the candidate D2

dopamine receptor, 5HTT serotonin transporter, VNTR polymorphism of the MAOA, and polymorphism COMT genes. Likewise, the contribution of environmental factors, in particular childhood adversity would help support the proposed model by Lovallo (2013). Finally, as it has been shown that blunted reactivity is related to both substance and behavioural addictions (Phillips et al., 2013) and as our society continues to change it would be informative to determine whether emerging dependencies are a characteristic of blunted stress reactors. Indeed, contemporary addictions such as computer gaming (Kuss, 2013), shopping (Murali et al., 2012), and sex (Rosenberg et al., 2014), have been shown to be increasing in prevalence and to have similar components and the adverse health and social implications as diagnosed substance addictions (Alavi et al., 2012).

Conclusions

In conclusion, this thesis used both laboratory testing and secondary analysis to further understand and expand the knowledge of the corollaries of individual differences in cardiovascular and/or cortisol responses to acute psychological stress. As a whole, three important themes emerged from the findings of the empirical chapters: there would appear to be dissociation between subjective affective and physiological responses to stress; the context in which the stressful situation is experienced is important; blunted reactivity appears to be related to adverse outcomes which are stable rather than transient. Additionally, the findings add support to the notion the blunted reactivity is a peripheral marker of central motivational dysregulation; dysfunction in the same neural systems in the brain which are involved in motivation, emotional responses, and physiological stress reactivity. This is evidenced by blunted reactivity being associated with an adverse constellation of personality traits and greater impulsivity. Overall, given the extensive research focusing upon

exaggerated reactivity to stress, and the findings of the current thesis alongside accumulating investigation of blunted stress reactivity, it would seem reasonable to conclude that both blunted and exaggerated reactivity may be maladaptive.

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